CJ FOUNDATION for SIDS partnership

The Baby Ball

The Alec Beattie Fund for SIDS Research

Jeremy Rill Centre

L'Hôpital de Montréal pour enfants
The Montreal Children's Hospital

Centre universitaire de santé McGill
McGill University Health Centre

This book belongs to: _____

Welcome from SIDS International

SIDS International would like to welcome you all to Canada and thank the Organizing Committee for their dedication and commitment in bringing together so many people to share their knowledge, expertise and experiences.

Our vision is to reduce the incidence of all sudden unexpected infant deaths throughout the world and to assist our members in providing a high standard of bereavement support for families who suffer the devastating death of their babies.

SIDS International has a commitment to continue risk reduction programs that have been so successful in saving thousands of young babies' lives over the last ten years.

We also have a commitment to work together with researchers to solve the mystery that still surrounds many of these deaths.

This Meeting brings together the collaborative team of researchers, health professionals and family members, that have achieved so much but still has a long way to go.

We have made discoveries about risks for infants, we have implemented programs that have reduced deaths and we have provided support for families, but we still do not know why many of these babies die.

This Meeting will challenge hypothesis, discover ideas, generate debate, see agreement and disagreement, but in the end, I hope that we will all leave Canada with a determination and renewed enthusiasm to meet the challenges ahead.

Maxine Weber
Chair, SIDS International

Committees

SIDS International Board Members

Maxine Weber, Australia, Chairman
Julia Kjaerstad, Norway, Secretary/Treasurer
Deborah Boyd, United States
Hazel Brooke, Scotland
Giampaolo Gabbi, Italy
Debra Keays-White, Canada

Conference Committee

Debra Keays-White, Nova Scotia, Conference Chair
Shelly Chubb, Alberta, Conference Co-chair

Social Committee

Shelly Chubb, Alberta, Co-chair
Michele Royal, Alberta, Co-chair
Shirley Bergen, Alberta
Ted Bergen, Alberta
Linda Reeves, Alberta

Parent Committee

Linda Campbell, Alberta, Co-chair
Jim Campbell, Alberta, Co-chair
Paul Coache, United States
Julia Kjaerstad, Norway
Carole Lapointe, Québec

Indigenous People Committee

Richard Jenkins, Alberta, Co-chair
Ruth Morin, Alberta, Co-chair
Catherine Carrty, Ontario
Sharon Clarke, Saskatchewan
Jessica Daniels, Alberta
Debbie Dedam-Montour, Québec
Jennifer Dickson, Ontario
Louise Garrow, Ontario
Fran Hyndman, Alberta
Diana Meabry, Ontario
Duane Morrisseau, Ontario
Leslie Randall, United States

Health and Allied Professional Committee

Local Committee

Teresa Gerez, Québec, Chair
André Corriveau, Northwest Territories, Vice-chair
Tina Langille, Ontario
Karen Martin, Alberta
Debbie Mpofu, Saskatchewan

International Advisory Committee

Hazel Brooke, Scotland
Deb Boyd, United States
Stephanie Cowan, New Zealand
Kathleen Gilbert, United States
Maxine Weber, Australia

Scientific Committee

Local committee

Aurore Côté, Québec, Chair
Jean-Paul Praud, Québec, Vice-chair
Aida Bairam, Québec
Carol Camfield, Nova Scotia
Ernest Cutz, Ontario
John Fisher, Ontario
Shabih Hasan, Alberta
Percy Kinney, Northwest Territories
Koravangattu Sankaran, Saskatchewan

International Advisory Committee

Roger Byard, Australia
Tracy Carbone, United States
Peter J. Fleming, United Kingdom
Carl E. Hunt, United States
Ed Mitchell, New Zealand
Hiroshi Nishida, Japan
Caroline Rambaud, France
Torleiv O. Rognum, United States

Special Symposia Committee

Roger Byard, Australia
Caroline Blackwell, Australia
Carol Camfield, Canada
John Fisher, Canada
Hannah Kinney, United States
Denis Leduc, Canada
Jean-Paul Praud, Canada
Toshiko Sawaguchi, Japan

First Candle/ SIDS Alliance

Deb Boyd, United States
Laura Reno, United States

General Conference Information

Conference Secretariat
International Conference Services Ltd.
604 - 850 West Hastings Street
Vancouver, BC Canada
V6C 1E1

Phone: (604) 681-2153
Fax: (604) 681-1049

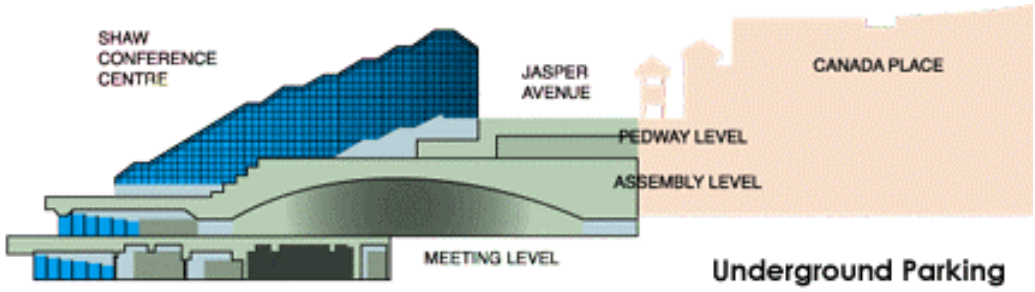
Conference Website: www.sidsi-canada2004.org
Conference e-mail: sids@meet-ics.com

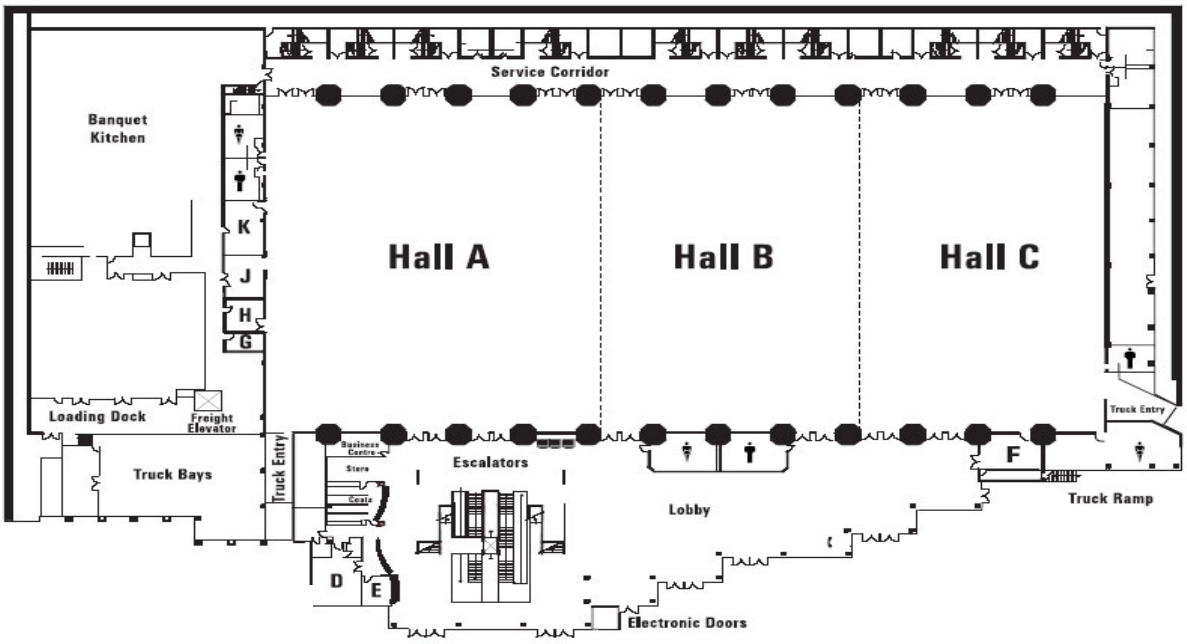
Conference Venue
Shaw Conference Centre
9797 Jasper Avenue
Edmonton, AB Canada
T5J 1N9

Website: www.shawconferencecentre.com

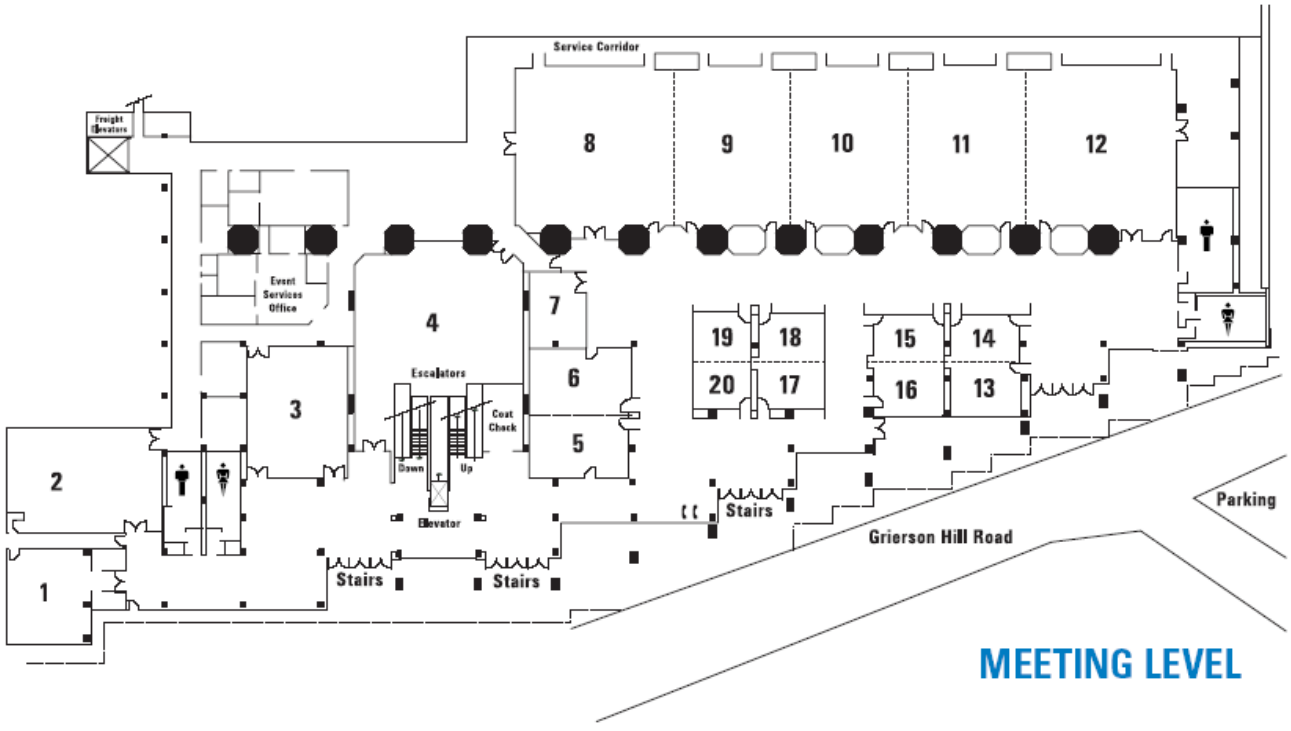
Offering a spectacular view of Edmonton's River Valley from the banks of the North Saskatchewan River, the award winning Shaw Conference Centre is situated in the heart of the City's vibrant downtown core. Gain easy access to Edmonton's major hotels, fine restaurants, theatre and downtown shopping district through direct ped-way connections.

Shaw Conference Centre Floor Plan





ASSEMBLY LEVEL



MEETING LEVEL

Conference Registration
Westin Hotel
Friday, July 2
07:30- 20:30

Shaw Convention Centre
Saturday, July 3
7:30-19:00
Sunday, July 4
7:30-14:30
Monday, July 5
7:30-16:30
Tuesday, July 6
7:30-12:00

Pre-Conference Workshops/Opening Ceremony/Award and Closing Ceremony
Pre-Conference Workshops
Friday July 2, 2004 from 8:30-17:30 at the Westin Hotel

Opening Ceremony
Friday July 2, 2004 at 19:30 at the Westin Hotel

Award for Best Abstract & Closing Ceremony
Tuesday July 6, 2004 at 12:00 at the Shaw Conference Centre

Speaker Ready Room
Salon 19, at the Shaw Conference Centre, has been allocated as the Speaker Ready Room. Laptops and LCD projectors will be available for preview of your presentation.

All Speakers must report to the Speaker Ready Room at least two hours prior to their presentation time, in order to ensure a consistent flow throughout the Conference.

Hours of Operation:
Saturday, July 3
7:30-17:30
Sunday, July 4
7:30-12:00
Monday, July 5
07:30-17:30
Tuesday, July 6
7:30-12:00

Name Badges
Attendees and guests are requested to wear their name badges at all times in order to participate in the Opening Ceremony, Pre-Conference Workshops, Scientific Program, Award & Closing Ceremony.

Colour Identification

Delegates	Blue
Students	Blue
Accompanying Person	Green
Single Day Registrations	Red
Exhibitor	Yellow
Volunteers	**Black**

Conference Language
The official language of the 8th SIDS International Conference is English

Poster Sessions
Poster Presenters are requested to adhere closely to the scheduled time period for displaying their Posters by 9:00 and for removing them no later than 17:30. Poster Presentations will be as follows:
Scientific Sessions
Saturday, July 3, 2004
13:30-15:30
Monday, July 5, 2004
13:30-15:30

Health & Allied Professional Session
Monday, July 5, 2004
10:00-11:00

All Poster Sessions are located in the Assembly Level Foyer at the Shaw Conference Centre. Pushpins may be used to attach Posters to the poster boards and the Conference Staff/Volunteers will have this available if required by Presenters.

Coffee-Breaks
During the Conference, complimentary coffee-breaks will be available in the Exhibitors Salon 8 and at the Assembly Level Foyer during Poster Sessions.

Lunches
Complimentary lunches will be available in Hall C, Assembly Level
Saturday, July 3
12:00-13:30
Monday, July 5
12:00-13:30

Exhibition
Exhibits will be held in Salon 8 of the Shaw Conference Centre
Saturday, July 3, 2004
10:00-15:30
Sunday, July 4, 2004
10:00-13:00
Monday, July 5, 2004
10:00-15:30

Social Program

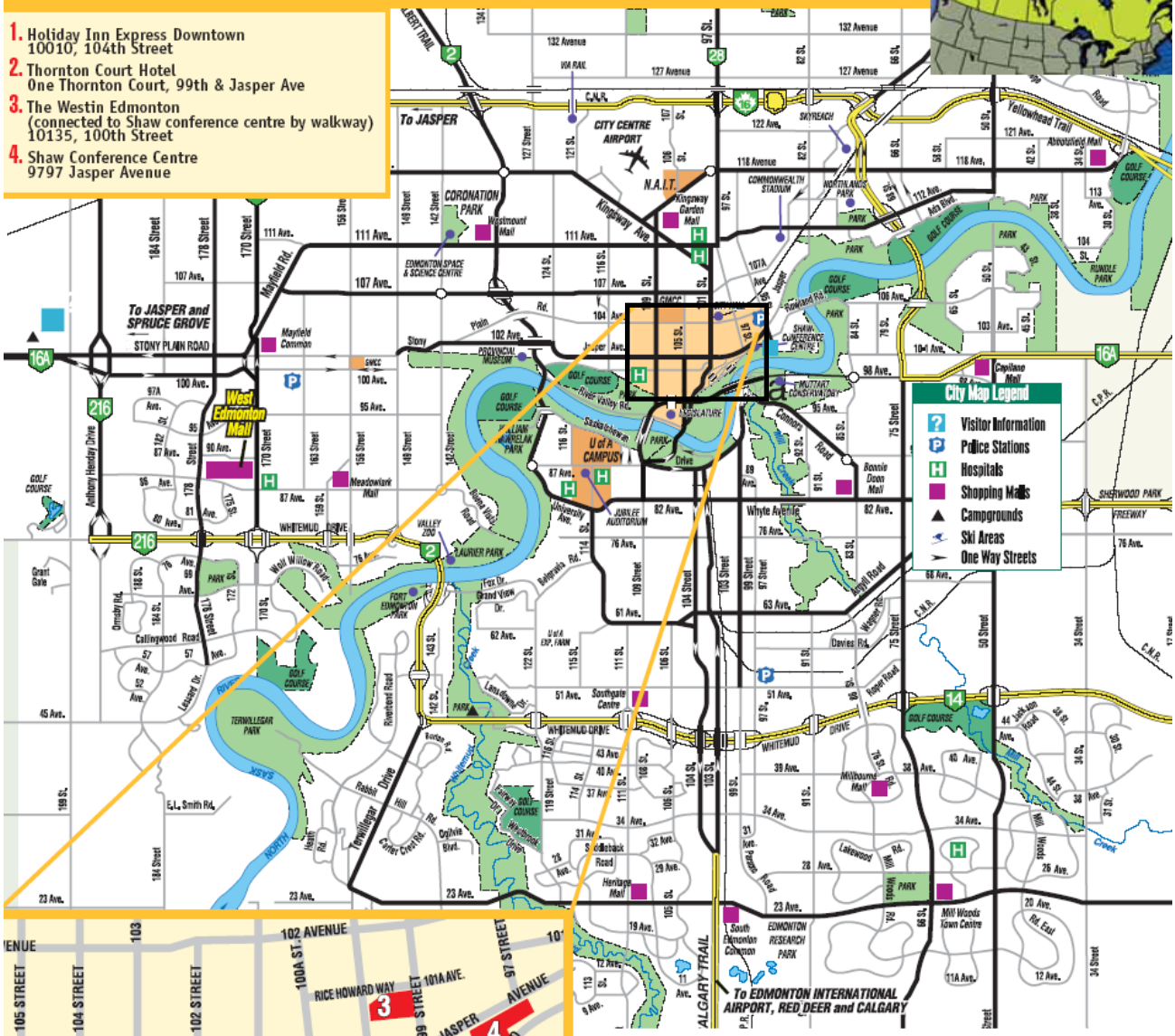
Fort Edmonton Park - Admission Fee and Transportation
Date: Sunday, July 4
Time: 13:00 - 17:00
Price: Adults $22, Children $15
One of Edmonton's premier attractions, Fort Edmonton Park represents four distinct time periods, exploring Edmonton's development from a fur trade post in the vast Northwest, to a booming metropolitan centre after the First World War. The Park features over 75 structures, many of which are the originals. Costumed interpreters operate the site and live the way of the past. Exploring each building, each room and talking to the 'inhabitants' makes for an extremely enjoyable recreational visit. This attraction can be viewed in a few hours or may take many return visits to appreciate the sense of the past. Free steam engine train and streetcar rides, as well as period rides such as wagon, stagecoach, pony and buggy are a big hit with everyone.
Let Fort Edmonton Park take you back in time!

Shuttle Bus to West Edmonton Mall (WEM)
Date: Sunday, July 4
Departure Shuttle from Shaw Conference Centre to WEM: 12:30 - 14:30
Return Shuttle from WEM to the Shaw Conference Centre: 16:30 - 19:30
Price for Adult Shuttle Bus Ticket (return trip): $20.00
Children: Complimentary
A shuttle bus will transport delegates from the Shaw Conference Centre to the famous West Edmonton Mall (WEM), the World's largest shopping mall and entertainment centre. A return shuttle service is available for delegates throughout the afternoon. Tickets for the shuttle are not included in the Registration Fee. Delegates require bus tickets to board the bus.

Evening at the Teepee Village
Monday, July 5th 18:00 - 22:00
Ticket Price: $30 Per Person
Children Under 12 Complimentary
Event Tickets Include: Dinner, Transportation and Entertainment
Enjoy an informal evening outdoors at a traditional Indian Teepee Village. Coaches will transport delegates from the Shaw Conference Centre to the Nechi Institute for an exciting evening of traditional Aboriginal food and Indigenous peoples' cultural performances. As is our way, we include and revere our Elders and children, so please bring your families. A Give-away Ceremony honours our guests with gifts. This event is not included in Conference registration and can be purchased at the time of registration. Coaches depart from the Shaw Centre at 17:30 arriving at the Teepee Village before 18:00. A return shuttle service is available for delegates throughout the evening.

Downtown Map

1. Holiday Inn Express Downtown
   10010, 104th Street
2. Thornton Court Hotel
   One Thornton Court, 99th & Jasper Ave
3. The Westin Edmonton
   (connected to Shaw conference centre by walkway)
   10135, 100th Street
4. Shaw Conference Centre
   9797 Jasper Avenue



PUBLIC TRANSPORTATION

Edmonton Transit System (ETS) has posted its route maps and schedule information on line at: www.takeETS.com. Telephone (780) 496-1611 (weekdays 6:30 a.m. - 9 p.m. and weekends/holidays 8 a.m. - 5:30 p.m.) to contact trained information operators who will help you plan your transit trip. ETS Lost and Found Office is open weekdays 8:30 a.m.-5:30 p.m. and can be reached at (780) 496-1622.

Sponsors, Scientific Program

Symposia Sponsorship

Federal Tobacco Control Strategy Programme, Health Canada

Full Sponsorship of the Tobacco and Smoking Sessions, July 5

Alec Beattie Memorial Fund, Montreal, Canada

Full Sponsorship of the Infection and Immunity Session

Canadian Institute of Health Research

Institute of Human Development, Child and Youth Health

Sleeping Arrangements, Context, Biology and Culture: Beyond the "Bedsharing Debate"

Institute of Circulatory and Respiratory Health

Translational SIDS Research: From Biome to Genome

Pampers

Sleeping Arrangements, Context, Biology and Culture: Beyond the "Bedsharing Debate"

Symposium on Upper Airways

Definition of SIDS and Diagnostic Criteria

Fonds Commémoratif Zachary Veillet, Québec, Canada

The Changing Profile of Sudden Unexpected Death in Infancy

Guest Speaker Sponsorship

Masimo Corporation, United States

Larry Becker Memorial Fund, Canadian Foundation for the Study of Infant Death

Jeremy Rill Centre for SIDS, Montreal, Canada

Hospital for Sick Children Research Institute, Toronto, Canada

Montreal Children's Hospital, Montreal, Canada

Respironics, United States

National Institute of Child Health and Human Development, United States

Alec Beattie Memorial Fund, Montreal, Canada

Norwegian SIDS Society, Norway

Wyeth Pharmaceutical, Montreal, Canada

Friday, July 2

Chairman Room

08:30-16:00 FIRST CANDLE/SIDS ALLIANCE WORKSHOP 1

Chancellor Room

08:30-16:00 FIRST CANDLE/SIDS ALLIANCE WORKSHOP 2

Leduc Room

08:30-16:00 FIRST CANDLE/SIDS ALLIANCE WORKSHOP 3

Yukon Room

08:30-16:00 FIRST CANDLE/SIDS ALLIANCE WORKSHOP 4

Centenial Room

08:30-17:30 WORKSHOP: FORENSIC AND PEDIATRIC PATHOLOGY

British Columbia Room

09:00-17:30 SLEEPING ARRANGEMENTS, CONTEXT, BIOLOGY AND CULTURE: BEYOND THE "BEDSHARING DEBATE"

Jasper Room – Thorton Court Hotel

08:30-17:00 CFSID BOARD OF DIRECTORS MEETING

Centenial Room

17:30-19:30 ISPID/ESPD MEETING

Saskatchewan & Manitoba Rooms – Westin Hotel

19:30-21:30 OPENING CEREMONY

Saturday, July 3

Salon 12

18:00 – 20:00 MEMORIAL SERVICE

The Memorial Service will begin with a Grande entry to the sound of traditional Aboriginal drums. Followed by prayers by Elders in several Aboriginal languages, words of encouragement and a ribbon held by all present to represent the line of life.

Participants will be invited to offer words, a poem, a thought, or a song in remembrance of the babies we have lost to SIDS.

An honour song, developed by the First people of Canada, will be presented as our gift.
It is the belief of Canada's Aboriginal people that children belong to the whole community - we all share in the grief of their loss and it is hoped, we will all share in the honour of this song.

At the end of the Memorial, each participant will be asked to tie a knot in the ribbon of life to represent our connection to each other and our love and remembrance of the babies we have lost.

It is the wish of the organizers of the Indigenous Stream and the Elders' Committee that this song be offered at the next International SIDS Conference in Japan and thereafter.

Saturday, July 3

	Salon 2	Salon 3	Salon 5	Salon 6	Salon 11	Salon 12	Salon 13	Salon 14	Salon 15	Salon 16
8:30 – 10:00					Plenary Contribution of SIDS Research to Global Infant Health and the Reduction of Infant Mortality					
10:00 – 10:30										
10:30 – 12:00	Aboriginal Leadership Forum on SIDS	Surviving the First Two Years	Hypoxia, Stressors and Vulnerability		The Changing Profile of Sudden Unexpected Infant Death	A Key to Improved Social Network Support	Abstract Presentation - Expanding Bereavement Services and Research Beyond SIDS		Abstract Presentation -Education & Prevention	
12:00 – 13:30										
13:30 – 15:00	That's Not the Right Way to Grieve! Is there Really a Right Way to Grieve?	Abstract Presentation - Classification of Sudden Infant Deaths	Pitapan's Traditional Healing Swing	Abstract Presentation: Back to Reality - An Indigenous Model for a SIDS Response	SIDS Update	Abstract Presentation - Epidemiology and Risk Factors	Abstract Presentation - Addressing Risk Factors		Abstract Presentation : Publish or Perish: How to Get a SIDS Publication Profile	Abstract Presentation: One Foot in the Door - A Cultural Icon - A Relevant Practice Today
15:00 – 15:30										
15:30 – 17:30	Forever Sleeping: The Design & Development of Culturally Appropriate Resource Material on SIDS	Translational Biology & SIDS: From Proteome to Biome		Abstract Presentation: Ending the Journey	Sibling Grief		Helping Bereaved Parents Cope with Guilt: What You Need to Know	Parent Advisory Area	Abstract Presentation Caring for our Volunteers, New Ideas, New Practices	

Salon E Registration: 07:30 – 19:00

Salon F Indigenous Therapy Swing: 08:00 – 12:00, 13:30-17:30

Salon 1 Elder's/Smudging Room: 08:00 – 17:30

Salon 7 Oasis Room: 08:00 – 17:30

Salon 8 Exhibits: 10:00–15:30
Coffee-Breaks: 10:00–10:30 and 15:00–15:30

Salon 19 Speaker Ready Room: 0730 – 17:30

Hall C Lunch: 12:00 – 13:30

Foyer, Assembly Level Scientific Posters: 13:30 – 15:30

Sunday, July 4

	Salon 2	Salon 3	Salon 5	Salon 6	Salon 11	Salon 12	Salon 13	Salon 14	Salon 15	Salon 16
8:30 – 10:00					**Plenary New Frontiers in Genetics and Neuroscience**					
10:00 – 10:30										
10:30 – 12:00	**Resilience**	**Abstract Presentation - Culturally Based Efforts to Reduce the Risk**	**Abstract Presentation: Innovative Strategies for Reducing SIDS Among American Indian/Alaskan Native Populations**	**Abstract Presentation: SIDS & Kids Queensland: Working Together Towards a Better Tomorrow**	**Symposium on Upper Airways**	**Larry Becker Memorial Lecture: Serotonin Symposium**	**Abstract Presentation - Fathers, The Forgotten, Bereaved Parent**		**Abstract Presentation: Creating Communities of Care with Aboriginal Families: A Relational and Shared Values Workshop**	**Talking Circle: Impact of SIDS on a Family**

Salon E Registration: 07:30 – 14:30

Salon F Indigenous Therapy Swing: 08:00 – 12:00, 13:30-17:30

Salon 1 Elder's/Smudging Room: 08:00 – 13:00
Teachings of the Moon: 13:00 – 17:30

Salon 7 Oasis Room: 08:00 – 17:30

Salon 8 Exhibits 10:00 – 13:00 and Coffee Break: 10:00 – 10:30

Salon 19 Speaker Ready Room: 7:30 – 12:00

Monday, July 5

	Salon 2	Salon 3	Salon 5	Salon 6	Salon 11	Salon 12	Salon 13	Salon 14	Salon 15	Salon 16
8:30 – 10:00					Plenary - Tobacco: The Traditional Path					
10:00 – 10:30										
10:30 – 12:00		How Men Grieve & How to Provide the Kind of Support They Want & Need	Developing Partnership with Tribes to Reduce SIDS Among Native Populations	Abstract Presentation: SIDS Risk Reduction - Tobacco as a Risk	Joint Session: Tobacco & Smoking		Walking with the Newly Bereaved Parent. What do I do? What do I Say?		Infection & Immunity	
12:00 – 13:30					Kaarene Fitzgerald Memorial Lecture					
13:30 – 15:00	Parent Abstract Presentation	Abstract Presentation - Sleeping Environment	Abstract Presentation - Cardiorespiratory Physiology						Abstract Presentation - Building a Caring Community for the Grieving Family	Abstract Presentation - SIDS Prevention & Education
15:00 – 15:30										
15:30 – 17:30	Walking the Path to Responsibility	"I have a headache" or "Just don't feel like It." Why Sex is Suspended Following Child Loss	Safe Sleep Practices to Reduce SIDS: It's Time for a Change	Abstract Presentation: What Do the Numbers Mean What We Did That Worked	SIDS Definition and Diagnostic Criteria		Abstract Presentation - Sensitive Issues, Sensitive Workers: The Death Scene & Parental Grief	Parent Advisory Area	Abstract Presentation - Tobacco Smoking Cessation in Pregnancy	

Salon E Registration: 07:30 – 16:30

Salon F Indigenous Therapy Swing: 08:00 – 12:00, 13:30-17:30

Salon 1 Elder's/Smudging Room: 08:00 – 13:00
The Journey of our Spirit: 13:00 – 17:30

Salon 7 Oasis Room: 08:00 – 17:30

Salon 8 Exhibits: 10:00–15:30
Coffee-Breaks: 10:00–10:30 and 15:00–15:30

Salon 19 Speaker Ready Room: 7:30 – 17:30

Hall C Lunch: 12:00 – 13:30

Foyer, Assembly Level Health Prof. Posters: 10:00–11:00
Scientific Posters: 13:30 – 15:00

Tuesday, July 6

	Salon 2	Salon 5	Salon 6	Salon 11	Salon 12	Salon 13	Salon 14	Salon 15	Salon 16
8:30 – 10:00				Plenary Living with Loss Over Time. Bottle it Up or Talk it Through?					
10:00 – 10:30									
10:30 – 12:00	Continuing Bonds with Your Deceased Child	Aboriginal Action Plan on SIDS		Future Research Directions	Nurturing Hope in the Face of Loss	Abstract Presentations - Tobacco Smoking Cessation in Pregnancy, Where Do We Go From Here?		Abstract Presentation: Soul Stories and Wise Ways	Abstract Presentation: Ensuring Good Business with Aboriginal Communities
12:00 – 13:30				"Best Abstract Award" Closing Ceremony					

Salon E Registration: 07:30 – 12:00

Salon F Indigenous Therapy Swing: 08:00 – 12:00

Salon 1 Elder's/Smudging Room: 08:00 – 12:00

Salon 19 Speaker Ready Room: 07:30 – 12:00

Tuesday, July 6

Salons 11 and 12

12:15 - 13:15 Closing Ceremony

The Closing Ceremony will open with the "Best Abstract Awards." There will be one Laureate from each Stream. The Award is presented by the Chair of each Stream.

In the tradition of SIDS International, the Closing Ceremony will consist of the symbolic passage of power to the Country organizing the next Meeting.

Saturday, July 3

Salons 11 and 12 — Plenary

Contribution of SIDS Research to Global Infant Health
Chairs: Raffaele Piumelli, Italy and Maxine Weber, Australia

08:30 – 8:35 **Introduction**
Raffaele Piumelli, Italy

08:35 – 9:00 **Preventing SIDS, Promoting Health**
Luigi Acerbi, Italy

09:00 - 9:25 **Fighting the Highest SIDS Rate**
Ed Mitchell, New Zealand

09:25 – 9:50 **Teen Health - STEPS to Reducing the Incidence of SIDS in the Teenage Demographic**
Mary McCormick, Canada

09:50 – 10:00 **Discussion**
Maxine Weber, Australia

Concurrent Sessions 10:30 – 12:00

Salon 12 **A Key to Improved Social Network Support** **H**
Kari Dyregrov, Norway
Chair: Karen Martin, Canada

Salons 13 and 14 **Expanding Bereavement Services Beyond SIDS** **H**
Oral Abstract Presentations
Chair: Hazel Brooke, Scotland; Paul Rusinko, United States

Salons 15 and 16 **Professional Education and Reduce the Risk** **H**
Oral Abstract Presentations

Salon 2 **Aboriginal Leadership Forum on SIDS** **I**
Dwight Dorey, National Chief, Congress of Aboriginal People
Kukdookaa Terri Brown, President, Native Women's Association of Canada
Chair: Ruth Morin, Canada

Salon 3 **Surviving the First Two Years** **P**
Joani Horchler, United States
Chair: Laura Reno, United States

H: Health and allied professionals **P: Parents**
I: Indigenous people **S: Scientists**

Salon 11 — **The Changing Profile of Sudden Unexpected Death in Infancy**
Chairs: Fern Hauck, United States and Åshild Vege, Norway

10:30 – 10:50 **International Statistics on Infant Mortality and SIDS**
Fern Hauck, United States

10:50 – 11:30 **The Changing Profile of SUDI – the Avon Experience**
Peter Fleming and Peter Blair, United Kingdom

11:30 – 11:50 **Sudden Unexpected Infant Death in Germany - Case Examination, Diagnostic Criteria, Morphology and Epidemiology**
Thomas Bajanowski , Germany

11:50 – 12:00 **Discussion**
Åshild Vege, Norway

S

Salons 5 and 6 — **Hypoxia, Stressors and Vulnerability**
Chairs: Toshiko Sawaguchi, Japan and Ernest Cutz, Canada

10:30 – 10:35 **Introduction**
Toshiko Sawaguchi, Japan

10:35 – 11:00 **Brain Mechanisms that Compensate for Cardiovascular Collapse**
Ronald Harper, United States

11:00 – 11:25 **Is Vulnerability a Cause or Consequence of Intermittent Hypoxia?**
Karen Waters, Australia

11:25 – 11:50 **The Cardiopulmonary Transition at Birth: Predictors of Future Failure?**
Shabih Hasan, Canada

11:50 – 12:00 **Discussion**
Ernest Cutz, Canada

S

Concurrent Sessions 13:30 – 15:00

Salon 11 — **SIDS: Sudden, Unexpected and Unexplained. . . But What do we Know and How Can We Continue to Learn and Prevent Future Deaths?**
Peter Fleming, United Kingdom

Salons 13 and 14 — **Addressing Risk Factors** Oral Abstract Presentations

H

Salon 5 — **Pitapan's Traditional Healing Swing**
Darlene Auger, Canada

Salon 6 — **Back to Reality – An Indigenous Model for a SIDS Response**
Oral Abstract Presentations

Salon 15 — **Publish or Perish: How to Get a SIDS Publication Profile**
David C. Tipene-Leach, New Zealand

Salon 16 — **One Foot in the Door - A Cultural Icon - A Relevant Practice Today**
Pauline Hopa, New Zealand

I

Salon 2	**That's Not the Right Way to Grieve! Is There Really a Right Way to Grieve?** Karen Martin, Canada Chair: Jordy Blackley, Canada	**P**
Foyer, Assembly Level	**Poster 1**	**Respiratory Physiology and Pathophysiology**
	Poster 2	**Risk Campaigns**
Salon 3	**Oral Abtracts**	**Classification of Sudden Infant Deaths**
Salon 12	**Oral Abstracts**	**Epidemiology and Risk Factors**

Concurrent sessions 15:30 – 17:30

Salons 15 and 16	**Caring for our Volunteers; New Ideas, New Practices** Chair: Betty McEntire, United States Oral Abstract Presentations	
Salons 13 and 14 **15:30 - 16:30**	**Helping Bereaved Parents Cope with Guilt: What You Need to Know** Bob Baugher, United States Chair: Karen Martin, Canada	**H**
16:30 - 17:30	Parent Advisory Area (Presence of Scientists and Health & Allied Professionals)	
Salon 2	**Indigenous Community Taking Action. Forever Sleeping: The Design and Development of Culturally Appropriate Resource Material on SIDS** Kay Half**,** Canada	**I**
Salon 6	**Ending the Journey** Randi Gage, Canada Abstract Oral Presentation	
Salon 12	**Sibling Grief** Janice Roper and Joani Horchler, United States Chair: Deb Boyd, United States	**P**

Salon 3 **Translational Biology and SIDS: From Proteome to Biome**
Chairs: Carl Hunt, United States and Carol Camfield, Canada

15:30 - 15:35 **Introduction** Carl Hunt, United States

Lessons from the Genome, Proteome and Cell Biology

15:35 – 16:00 **Mechanisms of Hypoxic Cell Death or Tolerance: Insights from Genetic Models**
Gabriel G. Haddad, United States

16:00 – 16:25 **Proteomic and its Application in Basic and Clinical Sciences**
Jenny Van Eyk, United States

16:25 --16:45 **Structural insight into Neuroepithelial Bodies: The Link to Hypoxia and SIDS**
Ernest Cutz, Canada

Lessons from the Biome and Integrative Biology

16:45 – 17:10 **Metabolism and Respiration: A Comparative Matrix - Effects of Temperature and Hypoxia**
Peter Frappell, Australia

17:10- 17:35 **Augmented Sleep Apnea in Orexin Knockout Mice**
Tomoyuki Kuwaki, Japan

17:35 – 17:50 **Integrative Cardiopulmonary Control in Mutant Murine Models**
John T. Fisher, Canada

17:50 – 18:00 **Discussion**

Memorial Service

Salon 12 **18:00 – 20:00**

Sunday, July 4

Salons 11 and 12 — **Plenary**

New Frontiers in Genetics and Neuroscience
Chairs: Hannah Kinney, United States and Shabih Hasan, Canada

08:30 – 8:35 **Introduction**
Hannah Kinney, United States

08:35 – 9:15 **Gene-environment Interactions : Implications for SIDS**
Carl E. Hunt, United States

09:15 – 10:00 **Developmental Neurotransmitters Pathology in SIDS**
Sachio Takashima, Japan

Concurrent sessions 10:30 – 12:00

Salon 3 **Culturally Based Efforts to Reduce the Risk I** Abstract Oral Presentations

Salons 13 and 14 **Fathers, the Forgotten Bereaved Parent** Abstract Oral Presentations
Chair: Barbara Heather, Canada

H

Salon 5 **Innovative Strategies for Reducing SIDS Among American Indians/Alaskan Native**
Mona R. Rosenman, United States Abstract Oral Presentations

Salon 6 **SIDS & KIDS Queensland: Working Together Towards a Better Tomorrow**
Sonia Hebert, Australia Abstract Oral Presentations

Salon 15 **Creating Communities of Care with Aboriginal Families: A Relational and Shared Values Workshop**
P Gaye Hansaon, Canada Abstract Oral Presentations

I

Salon 16 **Talking Circle: Impact of SIDS on a Family**
Brenda Jenkins, Canada
Theresa Whiskeyjack, Canada
Tess Jenkins, Canada
Albert Morin, Canaa
Vivian Jenkins, Canada
Shauna Lapatak, Canada

Salon 2 **Resilience**
Sally Miller, United States
Chair: Joani Horchler, United States

P

Salon 12

Larry Becker Memorial Lectures: Serotonin Symposium
Chairs: Roger Byard, Australia and John Fisher, Canada

10:30-10:35 **Introduction**
Roger Byard, Australia

10:35-11:00 **Dr Larry Becker: Contribution to SIDS Research**
Hannah Kinney, United States

11:00 – 11:25 **Serotonin Transporter Gene Polymorphism as a Risk Factor for SIDS**
Masaaki Narita, Japan

11:25 – 11:45 **Serotonin Neurons as Sensors of Blood CO_2: Role in Control of Breathing**
George Richerson, United States

11:45 – 12:00 **Relationship Between Serotonin Related Factors and Neuronal Plasticity in Sudden Infant Death Syndrome**
Toshiko Sawaguci, Japan

S

Salon 11

Symposium on Upper Airways
Chairs: Ronald Harper, United States and Jean-Paul Praud, Canada

10:30 – 10:40 **Introduction**
Ronald Harper, United States

10:40 – 11:00 **Evidence for Involvement of Obstructive Apnea in SIDS**
Bradley Thach, United States

11:00 – 11:20 **Anatomy of the Upper Airway in Infancy - And its Possible Relevance to SIDS: Is it the Baby Itself that Carries the Risk Factors?**
Shirley Tonkin, New Zealand

11:20 – 11:40 **Genetic Diseases and Syndromes Affecting Upper Airways**
Anat Shatz, Israel

11:40 – 12:00 **Infant and Environmental Factors Favoring the Development of Obstructive Sleep Apnea**
André Kahn, Belgium

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13:00 - 16:00

Salon 1

Teachings of the Moon - Women Elder's Circle on Parenting
Jenny Cardinal, Canada
Christine Daniels, Canada

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Monday, July 5

Salons 11 and 12 — Plenary

Tobacco… The Traditional Path

Jo-Ann Daniels, Aboriginal Community Support Coordinator

Roland Cotton – Elder, Blackfoot

Jerry Saddleback – Elder, Cree

Ernie Lennie - Dene

Salons 11 and 12 — Tobacco and Smoking Joint Session:

Chairs: Ed Mitchell, New Zealand and Debbie Mpofu, Canada

10:30 – 10:40 **Introduction**
Ed Mitchell, New Zealand

10:40 – 11:00 **Why Does Smoking Make Children More Susceptible to Infection?**
Caroline Blackwell, Australia

11:00 – 11:20 **Environmental Tobacco Smoke Exposure and SIDS in Germany**
Thomas Bajanowski, Germany

11:20 – 11:40 **Pathology and Prevention of Unexpected Perinatal Death and SIDS**
Luigi Matturri, Italy

11:40 – 12:00 **Tobacco and Smoking: Treating Tobacco Use among Pregnant and Parenting Smokers**
Cathy Melvin, United States

Concurrent Sessions 10:30 – 12:00

Salons 13 and 14 — **Walking With the Newly Bereaved Parent. What Do I Do? What Do I Say?**
Tina Langille, Canada
Chair: Karen Martin, Canada

Foyer — **Bereavement, Support and Counselling** Poster Presentation
Chair: Oe'livia Chasse, Canada
Smoking Cessation in Pregnancy Poster Presentation

Salon 5 — **Developing Partnerships with Tribes to Reduce SIDS Among Native Populations**
Heather Shotton, United States
John Sotton, United States

Salon 6 — **SIDS Risk Reduction - Tobacco as a Risk**
Cynthia Morris, Canada Abstract Oral Presentations

H

I

Salon 3	**How Men Grieve and How to Provide the Kind of Support they Want and Need** Oddbjorn Sandvik, Norway Chair: Jim Campbell, Canada	**P**

Salons 15 and 16 **Infection and Immunity**

Chairs: Caroline Rambaud, France and Koravangattu Sankaran, Canada

10:30 – 10:40	**Introduction** Caroline Rambaud, France	
10:40 – 11:00	**Evidence for Inflammation and Altered Immune Responses in SIDS Infants** Ashild Vege and Torleiv Rognum, Norway	
11:00 – 11:20	**Development of Infant Mucosal Immunity in Relation to Vulnerability to Infections** Magee Gleeson, Australia	**S**
11:20 – 11:40	**The Common Bacterial Toxin Hypothesis for SIDS** Jim A. Morris, United Kingdom	
11:40 – 12:00	**Risk Factors for Infection and Ethnic Differences in SIDS** Caroline Blackwell, Australia	

Concurrent Sessions 13:30 – 15:00

Salon 15	**Building a Caring Community for the Grieving Family** Chair: Margaret Robinson, Canada Oral Abstract Presentations	**H**
Salon 16	**SIDS Prevention & Education** Oral Abstract Presentations	
Salon 1	**The Journey of our Spirit (13:00 – 17:30)** Andy Blackwater, Canada	**I**
Salon 2	Abstract Oral Presentations Chair: Deb Boyd, United States	**P**

Foyer, Assembly Level	**Poster 1**	**Pathology**	
	Poster 2	**Neuropathology**	
	Poster 3	**Sudden Unexpected Death**	**S**
Salon 3	**Abstracts**	**Sleeping Environments**	
Salons 5 and 6	**Abstracts**	**Cardiorespiratory Physiology**	

Concurrent Sessions 15:30 – 17:30

Salon 5
Safe Sleep Practices to Reduce SIDS: It's Time for a Change
Tina Langille, Canada; Chrsitine O'Meara, United States

Salons 13 and 14
Sensitive Issues, Sensitive Workers: The Death Scene & Parental Grief
Chair: Dennis Caulfield, Canada Abstract Oral Presentations

Salons 15 and 16
Smoking Cessation in Pregnancy Abstract Oral Presentations
Chairs: Carol Sutherland , Canada; Cathy Melvin, United States

Salon 2
Walking the Path to Responsibility
Debbie Dedam-Montour, Canada

Salon 6
What Do the Numbers Mean? What We Did That Worked?
Abstract Oral Presentations - Maori-SIDS Joint Session
Coordination: Tamariki Maori, New Zealand

Salon 3
"I have a headache" or "I just don't feel like it"
Why Sex is Suspended Following Child Loss
Atle Dyregrov, Norway
Chair: Marian Sokol, United States

Salon 11
SIDS Definition and Diagnostic Criteria
Chairs: André Kahn and Torleiv O. Rognum

15:30 - 15:35 **Introduction**
Torleiv Rognum, Norway

15:35 – 16:00 **Sudden Infant Death Syndrome (SIDS) and Unclassified Sudden Infant Deaths (USID): A Definitional and Diagnostic Approach**
Henry Krous, United States

16:00 – 16:30 **International Consensus on Exclusion Criteria for the SIDS Diagnosis – Is it possible?**
Torleiv Rognum, Norway

16:30 – 17:00 **The Avon Multi-agency Approach to the Investigation of Sudden Unexpected Deaths in Infancy and the Care of Bereaved Families**
Peter Fleming, United Kingdom

17:00 – 17:20 **Australian Protocol and Definition Meeting**
Roger Byard, Australia

17:20 – 17:45 **Discussion**
André Kahn, Belgium

H I P S

Tuesday, July 6

Salons 11 and 12 — **Plenary**

08:30 - 10:00 **Living with Loss Over Time. Bottle it up or Talk it Through?**
Atle Dyregrov and Kari Dyregrov, Norway
Chair: Marian Sokol, United States

Concurrent Sessions 10:30 – 12:00

Salon 12 **Nurturing Hope in the Face of Loss**
Denise Larsen, Canada

Salons 13 and 14 **Tobacco Smoking Cessation in Pregnancy, Where Do We Go From Here?**
Chair: Carol Sutherland Brown, Canada
Panel: Cathy Melvin, United States; Debbie Mpofu, Canada

Salons 5 and 6 **Aboriginal Action Plan on SIDS**
Richard Jenkins, Canada

Salon 15 **Soul Stories and Wise Ways** Abstract Oral Presentations
Denise Miller, Canada

Salon 16 **Ensuring Good Business with Aboriginal Communities** Abstract Oral Presentations
Josie Dahlstrom, Australia

Salon 2 **Continuing Bonds with Your Deceased Child**
Dennis Klass, United States
Chair: Janice Roper

Salon 11 **Future Research Directions**
Chairs: Hiroshi Nishida, Japan and Aurore Côté, Canada

10:30 – 10:35 **Introduction**
Sylvia Limerick, United Kingdom

10:35 – 11:00 **Perspectives from the Pathologist**
Roger Byard, Australia

11:00 – 11:30 **Perspectives from the Physiologist**
Karen Waters, Australia
André Kahn, Belgium

11:30 – 12:00 **Perspectives from the Epidemiologist**
Peter Fleming, United Kingdom
Peter Blair, United Kingdom

Conclusion
Hiroshi Nishida, Japan

Contribution of SIDS Research to Global Infant Health and Reduction of Infant Mortality

Much has been accomplished in the fight to reduce SIDS rates throughout the world. The exciting news is that these efforts have, in turn, reduced overall infant mortality. We have invited scientists from the countries responsible for organizing the last few SIDS International conferences to give us the benefit of their experience. First, Dr. Luigi Acerbi, from Italy, will discuss the Preventing SIDS – Promoting Health program in his region: Lombardy. Then Ed Michell, from New Zealand, will talk to us about the experience in his country, which once had the highest SIDS rates. Canada is represented by Mary McCormick of the Canadian Foundation for the Study of Infant Death, who will highlight the pioneer work done with the STEPS program that specifically targets teenage mothers.

Preventing SIDS, Promoting Health in Lombardia Region – Italy

Luigi Acerbi and R. Lucchini, Italy

Lombardia is the most populated region of Italy; it lays in the northern part of the Country and has nearly nine million of inhabitants, eighty six thousand newborn per year.

In September 2001, the Regional Health Administration & the local section of the association "Seeds for SIDS" promoted a regional SIDS awareness campaign entitled "For your baby sleeping is quieter if…". It was aimed at promoting safer behaviour to reduce the risk of SIDS (back to sleep, quit smoking, avoid hyperthermia). The campaign was carried out in all fifteen Local Health Units (LHU or ASL).

The selected strategy included the following steps:

1- To establish a working group at LHU level.
2- To involve health professionals in small meetings on SIDS prevention (family paediatricians and general practitioners, district health managers, paediatric and obstetric hospital staff).
3- To distribute printed material (booklet and poster) to the nurseries, health care centres, child day care centres.

Objective: To measure the distribution coverage of printed material. To summarise the impact of the booklet "For Your Baby Sleeping is Quieter If…(SIQI)" on three behavioural items: Back to Sleep (BTS), avoidance of Environmental Cigarettes Smoke (ECS) and awareness of the risk of Baby Overheating (BOH).

To figure out the variability of BTS position in babies born to different hospitals. To find out the reasons behind the choice of parents who don't comply with the recommendations.

Method: At the beginning of the campaign and eighteen months later, all babies presenting to the health centres for first immunisation session (two months and half old) were recruited and their mothers interviewed. We finally enrolled 6520 subjects in 2002 (first round interviews) and 5322 in 2003 (second assessment). We analysed the association between safe behaviour (BTS, ECS, BOH) and being familiar with the SIQI booklet. Statistical analysis was carried out with the program EPI info 2000, CDC Atlanta.

Results: From our findings, SIQI booklet has been received by 74% (95% I.C.73.1-75.4) of parents. We found that the most relevant behavioral changes associated to reading the booklet were BTS and BOT. The results are summarised in table 1. Prevalence of BTS position showed to vary, from 76% (95% C.I. 67.8-83.7) to 55% (95% C.I. 47.6-62.3), according to the hospital of birth (data referred to the largest hospital of the region)

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Table 1

Behavioural Item	DID NOT read the SIQI Booklet	READ the SIQI booklet	O. R. (95% C. I.)
BTS position	**1067/1761 (60.2%)**	**2526/3510 (71.9%)**	1.69 **(1.50-1.91)**
Avoid risk of ECS	**1430/1753 (81.6%)**	**2967/3489 (85%)**	1.28 **(1.10-1.50)**
Know risk of BOH	**1565/1743 (89.8%)**	**3235/3473 (93.1%)**	1.55 **(1.25-1.90)**

Conclusion and recommendations: The booklet distribution reached a reasonable number of families. We feel that this proportion must be improved. Therefore a closer follow-up is needed to increase parents' coverage. If we consider the booklet as a marker, our data suggest that the campaign had a positive impact at regional level, mainly on two behaviors: BTS and BOH. Given the large number of the sample, we were able to identify some subgroups with poor compliance to the behavioral indications (lowest educational level, immigrates). The reasons expressed by parents not compliant with BTS position were: "sleep disturbances", "fear of choking with sicked up milk", "baby preference" and "colics".
The high variability of sleep position among babies born to different hospitals, underlines the importance to keep a good attitude among professionals entrusting the message. Moreover, the impact of SIQI booklet could be jeopardised by a poor adherence of nurseries to the recommendation of BTS. Our final recommendation is that "SIDS Awareness Campaign" should be carried out embracing the whole strategy. In particular we should pay attention to meeting hospital's nursery staff and paediatricians to obtain there alliance.

Fighting the highest SIDS rate

Ed Mitchell, New Zealand.

In the early 1980s there was concern about the postneonatal (1-11 months) mortality rate in New Zealand, as the rate was high compared with other developed countries and had not changed over two decades. Furthermore there were marked ethnic differences in postneonatal mortality rate with Maori being approximately twice the non-Maori, non-Pacific Islander (predominantly Europeans) rate. SIDS was the certified cause of 65% of all postneonatal deaths in New Zealand compared with approximately 30% in other countries. It followed that the postneonatal mortality problem in New Zealand was really due to the high SIDS mortality rate.

In 1984 I established and chaired the Auckland Regional Postneonatal Mortality Review Committee, which reviewed every postneonatal death in the Auckland Region. The main aim of this committee was to reduce mortality rate at the local level by the identification of preventable factors and local deficiencies. Only 10% were potentially preventable, such as infection and accidents. Many of the known risk factors for SIDS were apparent in the Auckland SIDS cases. There were also other factors which impressed the committee by their frequency eg prone sleep position, sharing parent's bed, previous pallor, cyanotic or breathing abnormalities, postnatal depression in the mother and changes in the infant's environment or daily routine. Their potential significance could not be assessed, as the prevalence of these factors in the population at risk was unknown.

While much can be learnt from overseas studies and retrospective review of SIDS cases locally there was a

need to obtain New Zealand data with appropriate controls in order to find causes for the high SIDS mortality rate in this country.

This led to the development of the New Zealand Cot Death Study. This was a three year prospective case-control study. This research project commenced on 1 November 1987 under my directorship and collected comprehensive information on infants dying from SIDS in the postneonatal age groups in most main centres in New Zealand (covering about 80% of all live births in New Zealand). This major study examined 485 SIDS cases and 1800 controls. Very satisfactory completion rates were achieved; obstetric records were examined and parental interviews were completed in 97.5% and 86.9% of subjects respectively. This major study would not have been possible without the skills and enthusiasm of Drs Scragg (epidemiologist), Becroft (pathologist), Taylor, Hassall, Barry, Allen, Roberts and Ford (paediatricians) and Mr Stewart, Mrs Williams and Dr Thompson (biostatisticians) and the co-operation of the Health Statistics Services and the Royal New Zealand Plunket Society.

Because of public and health professional concern about SIDS we analysed some of the data from the first year of the study. As expected many risk factors for SIDS were confirmed including:

- Socioecomic factors: (unmarried, manual occupation and younger age mother left school),
- Pregnancy (younger age of mother at first pregnancy, younger age of mother at infants birth, less than 4 months pregnant when first attended antenatal clinic, non-attendance at antenatal education classes and increasing number of previous pregnancies),
- Infant: (Maori ethnicity, male, low birthweight, and preterm birth), and
- Postnatal factors: (age of infant, time of day, season, region, infant admission to a neonatal unit).

In addition, however, we identified risk factors, which were potentially amenable to modification. These were:

- prone sleeping position of baby,
- maternal smoking, and
- not breastfeeding.

After controlling for all the above variables, the relative risks associated with prone sleeping position, maternal smoking and not breast feeding were still statistically significant. Population attributable risk calculations suggested that these three risk factors accounted for 79% of deaths from SIDS in New Zealand. The SIDS rate could theoretically be reduced from 4/1000 live births to 1/1000 live births if infants were not placed prone to sleep, mothers did not smoke and babies were breastfed.

The National Cot Death Prevention Programme was developed. A group consisting of the Plunket Society, Department of Health, New Zealand Cot Death Association, Area Health Boards, Maori representatives, the Commissioner for Children and the research group met to devise strategies to produce the desired change in infant care practices. A national co-ordinator for the programme was appointed. The Cot Death Prevention Programme was launched on 27 February 1991, although change in prone sleeping position occurred from the time the programme was being developed.

The SIDS prevention programme received a lot of interest both locally and overseas, partly due to the Thames television (UK) documentary "Every mother's nightmare" featuring our work, which has been shown in many countries. The prevention programme has been spectacularly successful. SIDS mortality rate in New Zealand has fallen from 4.4/1000 live births in 1988 to 2.3/1000 in 1992 and total postneonatal mortality from 6.1 to 3.6/1000 (1997). This equates to over 150 lives saved each year in New Zealand. Other countries followed our lead.

It is essential to monitor changes in SIDS mortality and changes in infant care practices to show a causal relationship between the two. We undertook a prospective study which collected data at birth and at 2 months of age on all infants born in New Zealand over a 2 year period. It showed that the prevalence of prone sleeping position decreased to less than 5% and confirmed the major risk factors identified in the New Zealand Cot Death Study.

Although the New Zealand Cot Death Study has identified the risk factors for SIDS in New Zealand it does not explain why the New Zealand rate is higher than that in other countries. We (Jones, Esmail and Mitchell) undertook a prevalence study of risk factors for SIDS in the South West Thames region of the United Kingdom, using the same methodology as the

New Zealand study, funded by the Foundation for the Study of Infant Death. The higher SIDS mortality rate in New Zealand would appear to be due in part to the higher prevalence of the known risk factors prone sleep position, maternal smoking, infant sharing a bed with another person. Breastfeeding was the only infant care practice, which was better in New Zealand than in South West Thames.

A fourth risk factor, namely infants sharing a bed with another person, was added to the prevention programme in 1992. Further analyses found that the risk was predominantly among infants of mothers who smoked. The recommendation that infants of mothers who smoke should not bed share with their infants caused considerable controversy. Lactation consultants had been promoting bed sharing to encourage and support breastfeeding, and it was perceived by some Maori as an attack on their culture.

In 1995 a Maori SIDS Prevention Team was established under the guidance of Dr Tipene-Leach. Initially this focussed on the established risk factors but the social and health issues facing Maori are bigger than "risk factors" and much of the community health workers work evolved around dealing with social issues, such as housing. However, the importance of bed sharing as a cause of SIDS (more than 50% of SIDS deaths in NZ occur while co-sleeping), became more apparent, and the Prevention Programme started addressing this issue. SIDS rates have continued to fall slowly, but more could be done.

What needs to be done? New Zealand Government policy is that health funding is now done at district level. This means that trying to establish a national programme is just about impossible. There needs to be national agreement about health risk (and benefits) of co-sleeping, and for that matter pacifier use, and this needs to be prominently promoted.

Teen Health
STEPS to reducing the incidence of SIDS in the Teenage Demographic

Mary McCormick, Canadian Foundation for the Study of Infant Death

Although many research studies have identified that teen mothers have a higher risk of SIDS, none of these studies actually address why. Steps for teen education to prevent SIDS --STEPS was developed in hope that we will be able to answer this question. The Canadian Foundation for the Study of Infant Deaths identified this need for educating this high-risk group and received a 2-year grant from the Trillium Foundation to develop and implement the STEPS program.

STEPS is a special communication and educational program that targets teen mothers and those that may be assisting in the care of an infant of a younger mother. This program responds to a unique target audience and at every step, involves this target group: teens and young adults. This program responds to an urgent need to educate young moms through a supportive educational training, teaching the steps to reducing the risk of SIDS and promoting good health before, during, and after pregnancy.

As stated previously, teen mothers have a higher risk of SIDS. A concern arose as to whether or not teens were receiving the back to sleep message. The belief was that they probably were, but perhaps not in a manner that they could relate to. Teenagers often have a carefree nature and sometimes seem disinterested in the medical environment. Some teens see nurses and doctors, parents and social workers as preaching and judgmental. Often teens avoid prenatal classes because of the fear that their limited knowledge may be grounds for having their child removed from their care. They can ask fewer questions and pretend to understand everything!

Mini focus groups were contacted in several areas to determine what type of materials should be developed to communicate the steps to reducing the risk of SIDS to teens.

Results of the survey led to the creation of a video accompanied with a guide and brochure, as well as a public service announcement. In addition, as teens are very technically inclined today, a special STEPS site was developed on the web with a chat area and a forum to post messages.

The public service announcement is a 30-second commercial for TV that makes the audience want

more information and directs the viewer to call the 1-800 number for more information. A similar auditory PSA was developed for radio. In order to make the material more appealing and more impressionable to teens, we added elements similar to the fast paced formats currently used by music video producers to the look of the public service announcement and video. Our poster and brochure were designed in a color scheme chosen by teens with large graphics and as few words as possible. The graphics speak for themselves and communicate the message effectively.

Furthermore, although literature about SIDS is readily available, STEPS information concentrates on reducing the risk, not the SIDS event, so this literature is seen as less scary and threatening.

This program begins before pregnancy educating teens in the school system. All STEPS materials have been accredited by Curriculum Canada Services to be used as teaching tools for parenting classes or Family Studies. By reaching students before they have a child, we encourage good health practices, promoting healthy pregnancies and healthier babies. Students learn the steps to reducing the risk of SIDS, which can be beneficial if they are babysitters too. Nearly everyone knows pregnant women shouldn't take illicit drugs, but it's the legal ones--alcohol and tobacco--that are more commonly the source of pregnancy problems. Smokers put their babies at a significantly higher risk of pre-term birth, low birth weight, and SIDS compared with nonsmokers. Students are taught good health is imperative to a successful pregnancy and parenthood.

STEPS also address the need of the expectant teen. Workshops are held educating the teens in the steps to reducing the risk of SIDS, stressing the importance of communication and a healthy lifestyle. Surveys are conducted to determine the needs and understanding of the participants.

The third aspect of the STEPS program addresses the teen mom and her baby. At this level, STEPS empowers the teen mom with the knowledge of reducing the risk of SIDS and increases her self esteem and confidence in dealing with those caregivers that assist her in childcare.

This training may have the added benefit of teens gaining presentation skills and in turn, higher levels of self esteem which can only positively affect all aspects of their lives.

In educating teens, it is important to address the need of educating all caregivers that may be involved in the care of the teen mom and baby. Grandparents, babysitters, teachers, social workers, prenatal instructors and more must be knowledgeable in these easy steps to reduce the risk of SIDS. This education must be a collaborative effort throughout the community to ensure that the message is the same from all involved.

In just one generation there have been significant changes in the way we parent our babies and children. It's not that grandma did the wrong things; she followed what were considered the best practices at the time. Grandparents may be confused about these new guidelines and insist that a teen mom is doing it wrong. STEPS empower teens with the knowledge necessary to reduce the risk of SIDS. Hopefully grandparents will have a healthy curiosity about the changes in baby care and will respect and support a teen's choices as a mother.

Currently, statistics are being gathered and in the future it is hoped that a reduction in the rate of SIDS in this target group will be seen. In order to measure the continued success rate of the STEPS program; trends will be monitored for several more years.

Together, teens and the Canadian Foundation for the Study of Infant Deaths explore the question of why teen mothers have a higher risk of SIDS, provide literature that is informative and non-threatening, and empower young mothers with knowledge that is science-based but friendly. This year, the program has provided support to nine teenage mothers whom have lost a child to SIDS. Of these nine, 3 babies were sleeping on their tummies and 2 on their sides. Age does not determine if someone will be a good parent or not, there are good and bad parents at all age levels, but education is key to reducing the risk of SIDS.

The changing profile of Sudden Unexpected Deaths in Infancy (SUDI)

Now that the campaigns to reduce the modifiable risk factors for SIDS have prove successful, researchers are starting to focus on unexpected deaths from other causes. Questions abound. Do the risk factors and characteristics of unexplained non-SIDS death --not to mention the explained sudden unexpected deaths-- differ from those of SIDS? Will our understanding of the characteristics of these other categories lead to a better understanding of the cause of SIDS?

International Statistics on Infant Mortality and SIDS

Fern Hauck, United States

Dramatic declines in SIDS rates have occurred in virtually all countries in which these statistics are available.[1] The reductions have exceeded 50% in most countries, and in some, approach 90% or more. While the final causal mechanism of SIDS is still uncertain, the decline of SIDS around the world has been attributed to reductions in prone infant sleeping position in countries in which this was the routine practice.[2] Risk reduction campaigns, focusing on sleep position placement of infants among other risk factors, have succeeded in bringing about this change, which, in turn, has been clearly tied to the continuing decline in the number of SIDS deaths.[3-12]

While these findings have been highly consistent, there is still considerable variability in SIDS rates across countries.[1] Differences in rates could reflect a number of factors, including differences in: 1) risk factors for SIDS and their prevalence; 2) definitions; 3) autopsy protocols and rates of use; 4) death scene investigation protocols and rates of use; and 5) classification of death based on autopsy, scene investigation and other information about the infant.[13] Additionally, concerns have been expressed that the reductions in SIDS are not as large as they appear, but may represent changes in classification within countries ("diagnostic shift") of some deaths that may previously been identified as SIDS more recently being identified as other causes of death, such as accidental asphyxia or "undetermined." A clue to this can be found in part by examining trends in postneonatal mortality rates in comparison to SIDS mortality rates within each country. Examination of cause-specific rates of sudden unexpected deaths in infancy (SUDI) can even better illuminate this question. In South Australia, for example, there has been a genuine decline in SIDS deaths, but there has also been a change in the diagnostic profile of SUDI, with an increase in deaths attributed to accidental asphyxia due to unsafe sleeping environments and cases designated as undetermined.[14] The availability of extensive background information about complex issues (social and environmental, among others) may result in the greater use of the undetermined designation.[14] So, while the overall postneonatal mortality rate fell, supporting a true fall in SIDS rates, it is important, also, to look at cause specific rates.

In this presentation, updated international data will be presented on SIDS rates and postneonatal mortality rates to examine and compare trends. Further, it will introduce the diagnostic and classification issues that play an important role in the interpretation of these statistical findings.

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The changing profile of SUDI – the Avon Experience

Peter S Blair and Peter J Fleming, United Kingdom

Since the acceptance of Sudden Infant death Syndrome (SIDS) as a registerable cause of death in 1971, the subsequent SIDS rate in England & Wales has followed a similar pattern to that observed in many other countries; a rise in incidence in the 1970's, peaking in the 1980's and a dramatic fall after the 1991 "Back to Sleep" Campaign. It has since been recognised that the initial rise in incidence was actually a diagnostic shift from death previously labelled as 'respiratory infant deaths' to the new classification of SIDS[1]. Thus, there is no evidence to suggest the high SIDS rates observed in the 1980's were substantially different in the previous decade, or for that matter, prior to the recognition of SIDS as a cause of death. The dramatic fall in SIDS rates over the last 15 years from 2 deaths per 1000 live births to less than 0.5 is directly related to the change in infant sleeping position over this time[2] . Given this change in infant care practice and decline in deaths we need to investigate how the epidemiological profile of SIDS infants has changed. National statistics show us that some factors such as gender remain unaffected, just over 60% of SIDS infants in England & Wales were males before and after the campaign, whilst the decline in deaths during the colder months and rise in younger SIDS infants suggest the profile has altered. However the limited detail of National statistics is restrictive and a more extensive collection of Regional data is required to provide the full picture.

We have been collecting SIDS data in Avon since 1984. Avon County is situated in the South-West of England with a population of over 800,000 people, predominantly white, with over 9,000 livebirths a year. The region is a mixture of rural and urban communities, the largest city being Bristol ranked 119th most deprived borough of 354 in the UK index of Local Deprivation[3]. The SIDS rate in Avon was higher than the national average during the 1980's (averaging 3 deaths per 1000 livebirths) and has fallen to around 0.5 deaths per 1000 livebirths subsequently. In the eight year period between 1984 and 1991 there were 256 SIDS deaths in Avon, whilst in the twelve year period since 1991 there have been just 69 SIDS deaths.

We will present changes in infant, maternal and social factors prior to and after the "Back to Sleep" campaign, a preliminary look at the data has revealed 3 important changes.

SIDS has traditionally been the largest component (80-90%) within the overall group of all sudden unexpected deaths in infancy (SUDI). The umbrella term SUDI also includes unexpected deaths that are subsequently explained such as death from unrecognised infection, unrecognised congenital anomaly, accidental death, non-accidental injury and death due to metabolic disorder. In Avon, prior to the "Back to Sleep" campaign 13% of SUDI deaths were explained; after the campaign this proportion has risen to 39%. Because it is usually not possible to differentiate between SIDS and explained SUDI until after the post-mortem the epidemiological investigation into SIDS in Avon has always been an investigation into SUDI, although information collected on the explained deaths has rarely been utilised. Given the proportional rise of explained SUDI future investigations can now use these deaths as a comparative epidemiological and pathological control group.

SIDS occurs in all social groups but is more prevalent in the socio-economically deprived groups. Recent studies suggest this economic divide between SIDS families and the UK population has widened.[4,5]. Since the "Back to Sleep" campaign the vast majority of SIDS families in Avon are from the socially deprived group with all that this entails in terms of low birth weight infants, short gestation, high parity, young maternal age, single parenthood and propensity to smoke and bottle-feed. Future SIDS studies can no longer just use a random control group from the population, a second control group of more deprived families is needed if we are to compare like with like.

The proportion of bed-sharing SIDS deaths has doubled from 14% between 1984 to 1991 to 28% in the subsequent 12 years in Avon. On top of that the proportion of co-sleeping deaths occurring on a sofa has also risen from 1% to 10%. However the actual number of bed-sharing deaths has declined from 4-5 a year from 1984 to 1991 to 2-3 deaths a year in subsequent years. This suggests that the number of SIDS deaths occurring in a cot has declined faster than those SIDS deaths occurring in the parental bed. Why this has happened is not clear but may be related to differences in how infants have been put down to sleep depending on whether the sleep is a solitary one or next to the parent. Again, however, this has implications on future studies in that we need not only to match for socio-economic status but also where the infant sleeps.

The epidemiological profile of SIDS infants has clearly changed and the future design of SIDS studies has got to change with it. The anticipated number of SIDS deaths has fortunately decreased whilst our knowledge of these infants and families has significantly increased. It is therefore important to utilise this information to construct groups of future control infants more closely aligned to the circumstances of SIDS infants.

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Sudden unexpected infant death in Germany
Case examination, diagnostic criteria, morphology and epidemiology

Thomas Bajanowski, M. Vennemann, Germany

In 1990, 1,283 cases of sudden and unexpected infant death (SUDI) were registered in Germany corresponding to an incidence of 1.42/1,000 live births, but showing large local differences. Only about 50% of these cases were investigated by autopsy.

In the same year the Westfalian crib death study was started. This morphological/epidemiological case-control study was performed in the north-west part of Germany, in an area where about 4.0 million people live. Between 1990 and 1994, 238 SUDI cases (SIDS n=205, explained SUDI n=33) were investigated using a standardised protocol (death scene investigation by the police; autopsy including histology, toxicology, microbiology, virology and neuropathology; questionnaire for parents interview; case-control-ratio 1:2). During the same time period 23 cases of unnatural death (8 infanticides) could be identified.

The prone sleeping position, face-down position, smoking during pregnancy, complete covering, pillow in bed, lack of breast feeding, bedsharing with an adult and low socio-economic conditions could be established as the main risk factors. Differences in the prevalence of these factors were present comparing the various groups of cases (typical SIDS, SIDS+, explained SUDI), but were not significant.

At the beginning of the year 1992 a local prevention campaign was initiated and the incidence of sudden unexpected infant death decreased constantly until now. Simultaneously we observed changes in the epidemiological profile of the cases between 1990-1991 and cases recruited between 1992-1994: the frequency of

- the prone sleeping position decreased from 84 to 54%,
- infants who were born with low weight (1500-2499g) increased from 7 to 17%,
- mothers who did not smoke during pregnancy increased from 34 to 48%
- mothers who smoked more than 10 cigarettes per day during pregnancy increased from 16 to 32%.
- fathers who did not have a completed school education or training increased (2 to 16% and 14 to 27%, respectively),
- non-German mothers increased from 1 to 8%,
- infants who had needed hospital care before the age of 28 days increased from 12 to 37%.

In the German SIDS study (GeSID) which had been carried out between 1998 and 2001 in 18 centres in Germany representing about 50% of the population, 455 cases of sudden and unexpected infant death (8% typical SIDS, 80% SIDS+, 12 % explained SUDI) were investigated using a standardised protocol similar to that of the Westfalian study as well as 28 unnatural deaths. The groups defined by morphological criteria differed for 3 main variables only: breastfeeding, position placed to sleep and coughing the day before death. The epidemiological profile showed that the prone sleeping position further decreased (42%), the frequency of smoking mothers as well as of heavy smoking mothers (>10 cigarettes per day) increased again (64% of the mothers smoked during pregnancy, 38% were heavy smokers). Important new factors are that 18% of the mothers were younger than 20 years old (1990-1994: 1%), and that a higher number of SIDS victims (50%) came from the lower socio-economic class.

Conclusions:

- Risk factors changed.
- A local and time-limited prevention campaign led only to limited success.
- It is neccessary to recognize new risks and to develop risk group-specific prevention methods.

Hypoxia, Stressors and Vulnerability

For more than thirty years, the topics of hypoxia, stressors and vulnerability have been discussed at length. Even in 1971, it was thought that SIDS victims died because, "while passing through a period of increased physiological vulnerability, some critical combination of extrinsic and intrinsic factors proves lethal (Peter Froggatt)." This 2004 session will update the progress of the past decade. In particular, we will discuss whether the alleged "vulnerability" is acquired pre- or post-natally. As well, we will look at whether hypoxia is a cause or a consequence of the vulnerability.

Brain mechanisms that compensate for cardiovascular collapse

Ronald Harper, United States

A number of characteristics preceding and coincident with the fatal event in the Sudden Infant Death Syndrome (SIDS) bear similarities with cardiovascular shock, a condition associated with blood loss, visceral pain, or severe infection. The shared characteristics include indications of enhanced sympathetic outflow, perhaps triggered in SIDS by a momentary loss in blood pressure, prior to the incident. The increased sympathetic outflow is followed by a sudden loss of sympathetic action and increased parasympathetic influences on the heart, a sequence resulting in profound bradycardia and hypotension which, if continued, leads to loss of perfusion and death. Several protective strategies have evolved to combat blood pressure loss, including recruitment of respiratory and somatic musculature to assist perfusion. Our objective was to determine the neural mechanisms recruited to sense hypotension and integrate somatomotor and autonomic processes to overcome blood pressure loss. Because SIDS occurs at a defined age period, it is essential to examine action of brain structures mediating cardiovascular and breathing control in developing preparations as well as in adult models; both human and animal studies are used, and pathological respiratory and cardiovascular conditions in humans (obstructive sleep apnea, heart failure, and congenital central hypoventilation syndrome) provide insights.

Blood pressure increases are associated with suppression of respiratory motor activity and apnea,[1] while profound hypotension is accompanied by tachycardia, extreme extensor muscle activation, and respiratory patterns that switch in strategy from very rapid breathing to marked, enhanced inspiratory and expiratory efforts.[2] Neural structures mediating transient blood pressure increases and decreases include areas ranging through the cerebellum, midbrain and forebrain, not just medullary baroreflex pathways, as indicated by functional magnetic resonance imaging studies in adults, adolescents, and animal preparations.[3,4] The responses are heavily lateralized, with the greatest proportion of signal changes found on the right side of the brain.

Studies of adults with sleep disordered breathing, congenital central hypoventilation syndrome subjects or heart failure patients show common structural damage or functional deficits to blood pressure challenges in the cerebellum and insular cortex, especially on the right side.[5,6,7] The right insular cortex modulates sympathetic action,[8] and can exert inhibitory or disfacilitatory influences on sympathetic output, an essential issue in compensation for shock.

Examination of the ventral medullary surface (VMS), a region classically associated with control of blood pressure, shows that activity declines in response to blood pressure elevation and increases to transient hypotension. Overall VMS activity declines in rapid eye movement sleep,[9] and a loss of dampening of responses to hypotensive challenges also occur, suggesting a removal of descending forebrain or other influences during that state.[10] If those other influences provide compensatory recovery action, any insult to the VMS or associated local reflex action would place the infant at special risk during that sleep state.

Optical imaging and functional magnetic resonance imaging (fMRI) data suggest that VMS responses to both transient hypertension and hypotension are inverse to those of adults in the very young feline preparation, adopting the adult pattern only by 25-30 days of age.[11,12] The fMRI evidence suggests a

substantial role for cerebellar and forebrain areas to serve depressor challenges in the young animal.

Both the cerebellar cortex and deep cerebellar nuclei apparently play large roles in mediating blood pressure challenges. That participation would be expected in vestibular or motor manipulations, but is also found in tasks not involving such challenges. The findings pose concerns with the aberrant cerebellar cortex development noted in SIDS victims and cerebellar Purkinje cell damage following hypoxic or ischemic events.[13,14] The findings also provide a basis for understanding the implications of impaired responses to tilt noted in infants at risk for SIDS,[15] as well as the enhanced apoptosis in vestibular areas in SIDS victims,[16] and suggest simple non-invasive means to evaluate risk for competency in blood pressure control in young infants.

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Is vulnerability a cause or consequence of Intermittent Hypoxia?

Karen Waters, Australia

SIDS may represent a common end point or response to a diversity of stressors. "Triple-risk" take into account epidemiologic, physiologic and autopsy findings (Filiano & Kinney, 1994). These models propose that an infant will die of SIDS when the following three overlapping factors occur simultaneously:

1- underlying vulnerability in the infant,
2- a critical developmental period in homeostatic control, and
3- an exogenous stressor(s) (Fig. 1.1).

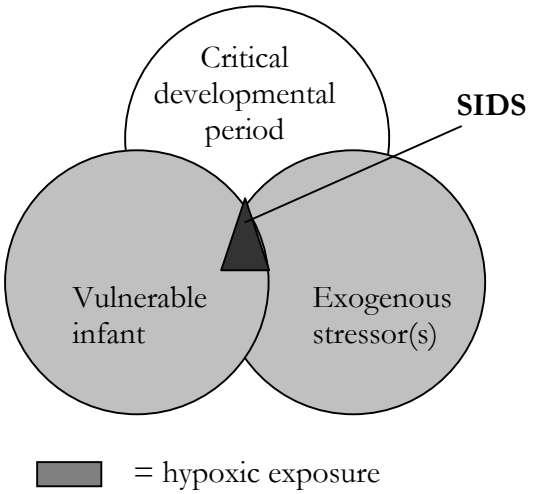

Fig. 1.1: SIDS triple-risk model.
The triple-risk model proposed by Filliano and Kinney, (1994), stating that an infant will die of SIDS only when the following three factors, represented by each circle, overlap simultaneously: 1) underlying vulnerability in the infant, 2) a critical developmental period in homeostatic control and 3) an exogenous stressor(s).

In triple-risk models, the risks include inherent vulnerability in the infant, as well as the age-specific risk for SIDS. Guntheroth supports the concept of a triple-risk model, but argues that there is insufficient evidence to state that the vulnerability of the infant has prenatal origins (Guntheroth & Spiers, 2002).

More specific hypotheses regarding the cause of SIDS have focussed on there being a brainstem abnormality that affects neuroregulation of cardiorespiratory control involving both neurotransmitter and receptor systems (Hunt & Brouillette, 1987; Kinney et al., 1995; Valdes-Dapena, 1986) In this case, the final result is increased neuronal cell death (apoptosis) in selected vulnerable brainstem nuclei (Sawaguchi et al., 2002; Waters et al., 1999).

Hypoxia has been implicated as a major contributor in producing this abnormality in SIDS (Hunt, 1992). The hypothesis that I will explore is that episodic, or intermittent hypoxia may induce vulnerability in otherwise normal infant, and that it may do so in the postnatal period. Hypoxia, whether it occurs in a single acute episode, or as an intermittent stimulus, causes considerable stress during early development. Specialised adaptive mechanisms exist in the young infant, which although life-saving during an acute response, can become maladaptive if they are sustained (Waters & Gozal, 2003). Clinical conditions that are known to increase the risk for SIDS, and which could induce such an exposure, include episodes of face-down position in the prone sleep position, or obstructive sleep apnoea.

When premature infants are placed in the prone position they have fewer desaturations, whereas older infants in the prone position have an increase in risk for rebreathing and episodic desaturation (Chiodini & Thach, 1993; Dimaguila et al., 1997; Patel et al., 2003; Waters et al., 1996) Obstructive sleep apnoea is clearly associated with moderately severe hypoxia and hypercapnia. Children with conditions which cause OSA are also known to have increased for sudden death, and OSA has also been linked with SIDS (Sawaguchi et al., 2002; Tishler et al., 1996). In this context, exposure to hypoxia may hold the concurrent role of being both an external stressor and a cause of the infant's vulnerability to death.

Data presented in this talk support:

1. the hypothesis that SIDS infants are likely to have neurochemical abnormalities.
2. the hypothesis that intermittent hypoxia can induce equivalent patterns of cell death
3. IHH can induce neurochemical changes in the brains of young piglets at an equivalent stage of

development as that usually associated with SIDS.

A variety of animal models have also been studied, but effects of intermittent hypoxia have only been of recent interest. The clinical conditions that are associated with hypoxia, also tend to also be associated with hypercapnia. For this reason, although many studies of relevance consider effects of intermittent hypoxia alone, the models in my own laboratory examined effects of hypercapnic hypoxia (HH), particularly when delivered as an intermittent stimulus (IHH).

Some of the findings that have now been associated with a diagnosis of SIDS include increased cell death markers in the brain, variable markers suggesting prior exposure to hypoxia, and environmental risk factors including prone sleeping and exposure to cigarette smoke. Similarities between the blood gas changes of those clinical conditions have been used to develop a piglet model in my laboratory, where pathological changes have been found which have many similarities to those observed in infants who have died of SIDS (Machaalani & Waters, 2003). Factors that have been linked between infants and animal models of hypoxia (whether sustained or intermittent) include changes in the presence and/or distribution of cell death markers, and changes in NMDA neuronal phenotypes.

NMDA is important because of its role in both respiratory control, and mechanisms of cell death. Studies in our laboratory have examined the distribution of these neurotransmitters in SIDS infants compared to controls, and then correlated those findings in piglets that were exposed to IHH compared to control animals that underwent the same protocol without IHH.

Limitations of these studies include the fact that the patterns of change are not completely equivalent, and more recently, that cigarette smoke exposure within the SIDS population is so high, that any influences associated with this particular risk factor may overwhelm other differences. Ongoing work will refine the nature of the insult required to induce the extensive changes observed in SIDS infants, and whether the presence of more than one stressor is likely to be required even for these neuropathological observations. Additional studies of human infants will further define the defects, and their clinical associations in SIDS infants.

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Scientific Sessions, July 3, Afternoon

Translational SIDS Research: From Biome to Genome

This session brings together scientific aspects ranging from the subcellular (proteomic and molecular biology) to the most complex (integrative and organ systems). The intent is not only to highlight the impact of these areas on SIDS research, but to look at the ensuing investigative opportunities. These areas of research are but examples of the broad disciplines that can contribute to our understanding of the pathophysiology of SIDS.

Mechanisms of hypoxic cell death or tolerance: insights from genetic models

Gabriel G. Haddad, United States

One of the major advances in the past 10 years has been the sequencing of hundreds of genomes belonging not only to invertebrates but also to mammals and humans. The result of this tremendous achievement has been the appreciation of the striking similarities in the genetic endowment between animals of vastly different phenotypes, such as the yeast, Drosophila melanogaster and man.

We have recently taken advantage of these discoveries to understand the molecular basis of how cells from animal phyla, species and various tissues respond to low O_2 environment. We capitalized on observations that we have made in the past on nerve cells from mammals and Drosophila. For example, we discovered several years ago that Drosophila melanogaster is exceedingly tolerant to anoxia. There is no apparent injury to the brain of Drosophila (evidenced by LM and EM) after several hours of 0% O_2 in the environment. Three approaches were started: i) a mutagenesis screen (forward genetics), ii) differential display and microarrays (reverse genetics) and iii) an approach relying on tools that facilitate the study of gene function in Drosophila. The presentation will highlight these approaches and some of our exciting newest results, a synopsis of which is detailed below.

Recent studies have shown that trehalose plays a protective role in yeast in a variety of stresses, including heat, freezing and thawing, dehydration, hyperosmotic shock and oxidant injury. Since a) heat shock and anoxia share mechanisms that allow organisms to survive, b) *Drosophila melanogaster* is tolerant to anoxia and c) trehalose is present in flies and is metabolically active, we asked whether trehalose can protect against anoxic stress. Here we report on a new role of trehalose in anoxia resistance in *Drosophila*. We first cloned the gene trehalose-6-phosphate synthase (*tps1*) which synthesizes trehalose and examined the effect of *tps1* over-expression as well as mutation on the resistance of *Drosophila* to anoxia. Upon induction of *tps1*, trehalose increased and this was associated with increased tolerance to

anoxia. Furthermore, in vitro experiments showed that trehalose reduced protein aggregation caused by anoxia. Homozygous *tps1* mutant (P-element insertion into the third intron of the gene) leads to lethality at an early larval stage, and excision of the P-element rescues totally the phenotype. We conclude that trehalose contributes in a major way to anoxia tolerance in flies; this protection is likely to be due to a reduction of protein aggregation.

We have also started on testing the role and importance of this tps1 gene and trehalose in potentially rescuing mammalian cells (human embryonic kidney cells) from hypoxic injury. These studies have yielded very interesting and exciting results; we have recently found that a Drosophila gene, when expressed in mammalian cells protects mammalian cells from hypoxic injury.

Proteomic and its application in basic and clinical sciences

Jennifer Van Eyk, United States

Proteomic analysis refers to the ability to isolate and identify specific proteins within different tissue, cells or subcellular organelles. The recognition that disease processes result not only in changes in specific genes, but also in specific proteins that subserve specific cellular functions has long been recognized. However, proteins can also be altered or changed independently from genetic changes and it is now clear that specific diseases result in a pattern of change in proteins within the affected organ or nervous tissue. Dr. van Eyk will review the current state-of-the-art techniques in proteomics to isolate and identify proteins. She will also review examples of the successful use of proteomics to identify changes in proteins that serve as markers of cardiopulmonary disease, as well as proteins that may serve to or predict the onset of disease. The development of sophisticated proteomic analysis, accompanied by the practice of assembling tissues banks of material from SIDS victims, should provide a unique opportunity to identify target proteins that play critical roles in SIDS.

Structural insights into neuroepithelial bodies : the link to hypoxia and SIDS

Ernest Cutz, Canada

Our studies on respiratory control in SIDS are focused on the possible role of peripheral chemoreceptors, particularly the newly characterized airway O2 sensors, pulmonary Neuroepithelial Bodies (NEB) (1-3). The aim of this presentation is to review recent findings in human and animal experimental models on structural and functional characterization of NEB as they may relate to the pathobiology of SIDS.

Morphologic features, innervation and development

In mammalian lungs, typical NEB appear as organoid clusters of 5-10 cells widely distributed within the epithelium of intrapulmonary airways. Since NEB cells produce amine (serotonin, 5HT) and variety of peptides (ie. bombesin, CGRP) as well as express a number of neural and neuroendocrine markers they can be visualized by means of immunohistochemical methods using specific antibodies (1-3). At the ultrastructural level, NEB exhibit many features consistent with a chemoreceptor function ie. the presence of (a) dense core vesicles (DCV), required for the synthesis and storage of amine/peptide neurotransmitters/neuromodulators, (b) preferential location of NEB at airway bifurcations with apical surfaces directly exposed to the airway lumen to monitor concentration of O2, and (c) afferent sensory innervation of vagal origin with nerve cell bodies residing in the nodose ganglion (4,5). Recent studies using multilabel immunohistochemistry and confocal microscopy have identified complex innervation of NEB in rat lungs including nitriergic and purinergic components (6). Quantitative studies on the distribution and frequency of NEB in both human and animal lungs revealed striking developmental changes with prominence of NEB during the fetal and perinatal period and decline postnataly indicating that their function may be important during lung

development and neonatal adaptation (1, 3). It has been proposed that in neonates, NEB's may complement the function of arterial chemoreceptors - the carotid bodies (CB) when CB function is still immature.

Cellular and molecular mechanism of O_2 sensing

The investigation of O2 sensing mechanism in NEB cells became possible with the development of suitable in vitro models and after devising means to identify NEB in a living state to allow electrophysiological recordings (7). There are now several models which can be used for functional studies on NEB at cellular and molecular level, including culture of NEB isolated from fetal/neonatal lung, fresh lung slice preparation and small cell lung carcinoma cell lines (SCLC, tumor counterpart of NEB in normal lung) (8). These studies using whole-cell patch- clamp demonstrated voltage- activated Na+,Ca2+and K+ currents in NEB cells. Hypoxia (pO2~25) reversibly inhibited K+ current while other conductances were unaffected (7). Subsequent studies have further defined NEB- O2 sensor as a membrane delimited O2 sensing molecular complex composed of multicomponent NADPH oxidase linked to an O2 sensitive K+ channel[K+(O2)](9). According to the membrane model, hypoxia leads to decreased oxidase activity with reduced ROS generation including H2O2. This in turn alters the redox status of K+(O2) causing its inactivation or closure, leading to membrane depolarization, activation of voltage -gated Ca2+ channels, Ca2+ influx triggering exocytosis of DCV with release of 5HT and other neurotransmitters onto afferent nerve endings transmitting the hypoxia stimulus to the CNS (1,3,8). The critical role of the oxidase as an O_2 sensor protein in NEB cells has been confirmed in a oxidase deficient (OD) mouse model showing no response to acute hypoxia stimulus (10). Furthermore, neonatal OD mice show abnormal breathing characterized by rapid and shallow respiration compared to wild-type mice when tested by whole body plethysmography (11).

Hypoxia chemotransduction in NEB

The candidate neurotransmitters that mediate fast chemosensory transmission of hypoxia stimulus from NEB cells via vagal sensory afferents include 5-HT, acetylcholine (ACh) and ATP. The evidence for the role of 5-HT has been strengthen recently by the demonstration of a dose response to various concentrations of O2 using carbon fiber amperometry (12). At present there is only indirect evidence for hypoxia evoked release of ACh and ATP from NEB cells. On the other hand expression of pre and post synaptic ionotrophic receptors i.e. 5 HT-3 receptor, nACh receptors (alpha3/beta2, alpha4/beta2 and alpha 7) and ATP (P2X2/3) have been demonstrated in NEB of rabbit and hamster lung (13-15). At least two of these receptors (5HT-3, P2X2/3) appear to be involved in the modulation of hypoxia chemotransduction acting as autoreceptors, amplifying hypoxia evoked 5-HT release (13, 15).

Pulmonary NEB in SIDS (Morphology and Pathobiology)

Hyperplasia of both solitary neuroendocrine cells and NEB has been reported in lungs of SIDS victims when compared to age matched controls (16). Chronic hypoxia and/or developmental delay have been postulated to account for NEB cell hyperplasia. We have found that maternal smoking during pregnancy potentiates NEB hyperplasia, possibly mediated via nACh-R (17). In addition to maternal smoking, airway inflammation (found in ~30% of SIDS victims) could act as a potential trigger for SIDS mediated via ROS and /or cytokines generated during inflammation. According to this hypothesis, an interaction between mild respiratory infections, airway inflammation and chemoreceptor dysfunction, potentiated by exposure to nicotine (since maternal smoking is a recognized risk factor for SIDS) could act as "external" stressors leading to SIDS (18).

Future prospects for SIDS research

Great advances have been made in uncovering the complexity of cellular and molecular mechanisms of O_2 sensing in airway chemoreceptors using animal models. The relevance of this information to SIDS can now be tested directly using genomics/proteomic approaches to NEB isolated or micro dissected from lungs of SIDS victims. For example expression of various O_2 sensitive K+ channels, classical and alternate O_2 sensing protein(s), hypoxia -inducible factors (HIF's) as well as neurotransmitter and neurotransmitter receptor genes can now be assessed using molecular techniques. The significance of various molecular defects can be further validated using transgenic mouse models with abnormal respiratory control mimicking SIDS.

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Metabolism and respiration: a comparative matrix - effects of temperature and hypoxia.

Peter Frappell, Australia

In mammals the rate of oxygen consumption ($\dot{V}o_2$) is roughly equivalent to the production of ATP. If the level of inspired O_2 declines, regulated adjustments in either conduction (structural parameters) and/or functional changes in the partial pressure gradient for all or any step in the oxygen cascade ensures that $\dot{V}o_2$ more or less is maintained constant. When the supply of oxygen can no longer sustain aerobic ATP production the animal must either down-regulate its energy requirements or find alternative sources of energy. In either case, $\dot{V}o_2$ will decline. Alternatively, the decline in $\dot{V}o_2$ could occur before oxygen becomes limiting if another process that utilized oxygen was attenuated. In small mammals and newborns, in general, the hypoxic decrease in $\dot{V}o_2$ is mostly related to a decrease in thermogenesis and

hence is associated with a decline in body temperature; the result of a decrease in thermoregulatory operational points rather than hypothermia. It should be noted, however, that a thermogenic component of the hypoxic induced hypometabolism is not present in all newborns. Indeed, in the ectothermic marsupial newborn exposure to hypoxia results in a decrease in $\dot{V}o_2$ and this is clearly not attributable to the attenuation of thermogenesis.

Most interestingly is that in situations where supply is not limiting, ventilation is appropriately matched to changes in $\dot{V}o_2$ such that convection requirement per unit of O_2 uptake is maintained appropriate for the level of inspired O_2, regardless of the change in $\dot{V}o_2$ that may or may not have occurred. This suggests that supply is correctly matched to demand and this step in the O_2 cascade is therefore appropriately regulated. Further, continued exposure to hypoxia is often, though not always, associated with the maintenance of hyperventilation and a return to pre-hypoxic levels of $\dot{V}o_2$. The return to normoxic values of $\dot{V}o_2$ in the presence of sustained hypoxia is achieved through a number of acclimation mechanisms that improve O_2 delivery and in the case of the newborn is essential to prevent compromises in growth.

Despite appropriate ventilatory responses to hypoxia, the presence or absence of thermoregulatory responses in mammals can complicate the interactions between hypoxia and temperature in terms of respiratory control. For example, in the newborn rat the effects of cooling on the strength of the Hering Breüer (HB) reflex are not attributable to changes in body temperature *per se,* but to the corresponding changes in metabolic rate that were associated with a limited thermogenic effort. Whereas, in the ectothermic marsupial newborn it is suggest that the decline in the HB reflex observed during moderate hypothermia is the result of a direct effect of body temperature on vagal mechanisms rather than a temperature driven decline in metabolic rate that should have acted to strengthen the HB reflex. Further, in both species, whether a thermogenic component of metabolic rate is attenuated or not, in the presence of hypoxia the strength of the HB reflex is confounded by an induced hypoxic drive.

This talk adopts a comparative approach in understanding the interplay between metabolic rate and ventilation and in exploring the effects of hypoxia and temperature on this relationship and the possible implications for respiratory control.

Augmented sleep apnea in orexin knockout mice

Tomoyuki Kuwaki, Japan

Basic research on sleep apnea using experimental animals may help understanding and prevention of sudden infant death syndrome (SIDS) because the syndrome is thought as inability to wake up from respiratory arrest (apnea) during sleep. Although several animal models of sleep apnea have been described previously, mice would be useful experimental animals in that these animals are frequently used in genetic engineering and there is a possible link between genetics and etiology of SIDS. These considerations prompted us to establish a method for measuring ventilation of mice concomitantly with electro-encephalography (EEG) and electromyography (EMG) for assessing sleep-wake states (ref. 1).

Normal wild-type mice developed two types of central sleep apneas, that is, post-sigh and spontaneous apneas, as normal humans do. Moreover, post-sigh apneas in mice were observed exclusively during slow-wave sleep (SWS) while spontaneous apneas were seen in both SWS and rapid-eye-movement (REM) sleep. These characteristics are very similar to those of sleep apneas in healthy human infants and children. Therefore, mice seem to be a promising experimental animal model for studying the genetic and molecular basis of respiratory regulation and dysregulation during sleep in humans, especially infants and children (ref. 2).

Applying this newly developed method to orexin knockout mice, we examined whether orexin

participates in the breathing control during sleep. Orexin, a recently discovered hypothalamic neuropeptide, regulates not only orexis (appetite) but also sleep/awake states and cardiovascular homeostasis (ref. 3). We hypothesized that orexin may contribute to the vigilance-state-dependent respiratory adjustment. Ventilation, together with EEG and EMG, was recorded for six hours in the daytime, resting period for nocturnal mice. Recording chamber was continuously flushed with either (1) room air, (2) hypoxic (15% O_2), or (3) hypercapnic (5% CO_2, 21% O_2) gas mixtures. Respiratory frequency, tidal volume, minute volume, and frequencies of apneas and augmented breaths were separately determined during quiet wakefulness, SWS, or REM sleep. Hypercapnic ventilatory responses during quiet wakefulness were attenuated in orexin knockout mice, although hypoxic responses were comparable to those in the wild-type littermates. Moreover, spontaneous apneas during SWS were more frequent in orexin knockout mice than in wild-type littermates.

Our findings suggest that orexin-containing neurons play a crucial role both for CO_2-sensitivity in wakefulness and for preserving ventilation stability during sleep. This approach may open a new avenue for SIDS research although we should keep in mind such limitations that mice are nocturnal rodents and they sleep in the prone position.

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Integrative cardiopulmonary control in mutant murine models

John Fisher, Canada

Scientists have long used animal models to test hypotheses about the cardiopulmonary control systems that may be involved in SIDS. However, the advent of the human genome project, as well as the sequencing of the genome of other animals such as the mouse, has provided an explosion of information and opportunity for research. Dr. Fisher's presentation will review the development of the mouse as a model for cardiopulmonary control and the strengths and weaknesses of the model to predict human physiology. The development of mutant and transgenic animals, which have specific genes "knocked out" or up-regulated, provide a highly novel development in the use of murine models and such approaches now impact on virtually every area of biological understanding. Several promising knockout murine models have been studied, as outlined by Dr. Kuwaki. This presentation will focus on other examples of genetically altered mice including some that display altered control of breathing relevant to SIDS. These include mice lacking the dopamine transporter (DAT), muscarinic receptor proteins, and the transient receptor potential vannilloid 1 (TRPV1) ion channel.

New Frontiers in Genetics and Neuroscience

Exciting new developments take place almost every day in genetics, as new genes are identified and their roles determined in health and disease. And, for more than a decade now, the role of neurotransmitters in SIDS has been under investigation. This 2004 session reviews, for a broad audience, the possible genetic and environmental interactions involved in SIDS; as well it examines the nature of neurotransmitters and their role in the SIDS enigma.

Gene-Environment Interactions: Implications for Sudden Infant Death Syndrome

Carl E. Hunt, United States

Sudden Infant Death Syndrome (SIDS) has historically been considered to be caused by adverse environmental exposures. Most human conditions, however, are determined by genetic and environmental components interacting in ways such that the whole is not only greater but may be different than the sum of its parts. Rather than choosing between "nature or nurture," therefore, both genetic and environmental contexts are essential. This presentation will review what is known today about genetic and gene-environment (G-E) interactions for representative human diseases and about the relevance of G-E interactions to a broadened perspective on pathophysiology of SIDS.

There are several implied but incorrect assumptions that serve as barriers to accepting genetic risk factors and G-E interactions as causal in SIDS. They have persisted as barriers despite our rapidly expanding genomic knowledge base:

- SIDS is a unique and distinct disorder that does not follow the same "rules" as other human disorders
- Genetic disorders are caused by an abnormal gene; there is a 1:1 relationship between gene and disease, and having that gene predicts destiny
- If SIDS is a genetic disorder, then there must be a SIDS gene
- If there is a SIDS gene, then the parental source of this gene is "at fault"
- Diseases are either genetic or environmental in their origin, but not both
- The existence of major environmental risk factors, therefore, precludes the concurrent existence of causal genetic factors

Several genes have now been identified for which the distribution of polymorphisms differs in SIDS victims compared to control infants. Corresponding phenotypes are known for SCN5A and can be inferred to a very limited extent for the ANS genes, but are totally unknown for 5-HTT polymorphisms:

- Sodium channel gene (SCN5A)
- Promoter region of serotonin transporter
- protein (5-HTT) gene
- Genes pertinent to development of autonomic nervous system (ANS):
    - Paired-like homeobox 2a (PHOX2a)
    - Rearranged during transfection factor (RET)
    - Endothelin converting enzyme-1 (ECE 1)
    - T-cell leukemia homeobox (TLX 3)
    - Engrailed-1 (EN 1)

The clinical physiological studies implicating impaired neuroregulation of cardiorespiratory control or other autonomic functions are consistent with the brain stem abnormalities and the molecular genetic findings in SIDS victims. The list of genotypical differences in SIDS victims may expand considerably as additional molecular genetic studies are performed. There are some genotype and phenotype data to suggest that impaired immune responses to infectious stressors may also be a mechanism leading to SIDS. Investigation of other serotonin genes and other genes known to be involved with circadian rhythmicity and sleep regulation may be important.

Several important environmental factors are associated with increased risk for SIDS and may indeed often be the trigger that perturbs homeostasis sufficiently to result in sudden death. Susceptibility to SIDS in individual infants, however, is likely determined not by the trigger alone, but by genes and the proteins they encode interacting with challenges from the environment. Like other diseases, therefore, SIDS likely occurs in genotypes in which the environmental challenge leads to a lethal change in the presence or expression of one or more protein products affected by that polymorphism.

There are multiple ways in which known environmental risk factors may be interacting with specific genotypes. There appears, for example, to be an interaction between prone/side sleep position and impaired ventilatory and arousal responsiveness. There may also be links with modifiable risk factors such as soft bedding, prone sleep position and thermal stress, and links between genetic risk factors such as ventilatory and arousal abnormalities and temperature or metabolic regulation deficits. The increased risk for SIDS associated with fetal and postnatal exposure to cigarette smoke also appears at least in part to depend on genetic risk factors.

In summary, genes and proteins interact to produce complex networks which in turn interact with the environment to influence every aspect of our biologic lives. Failure to consider *both* genetic *and* environmental context will impede research progress whereas the study of dynamic G-E interactions constitutes a powerful strategy for enhanced understanding of SIDS and sudden unexpected death in infancy.

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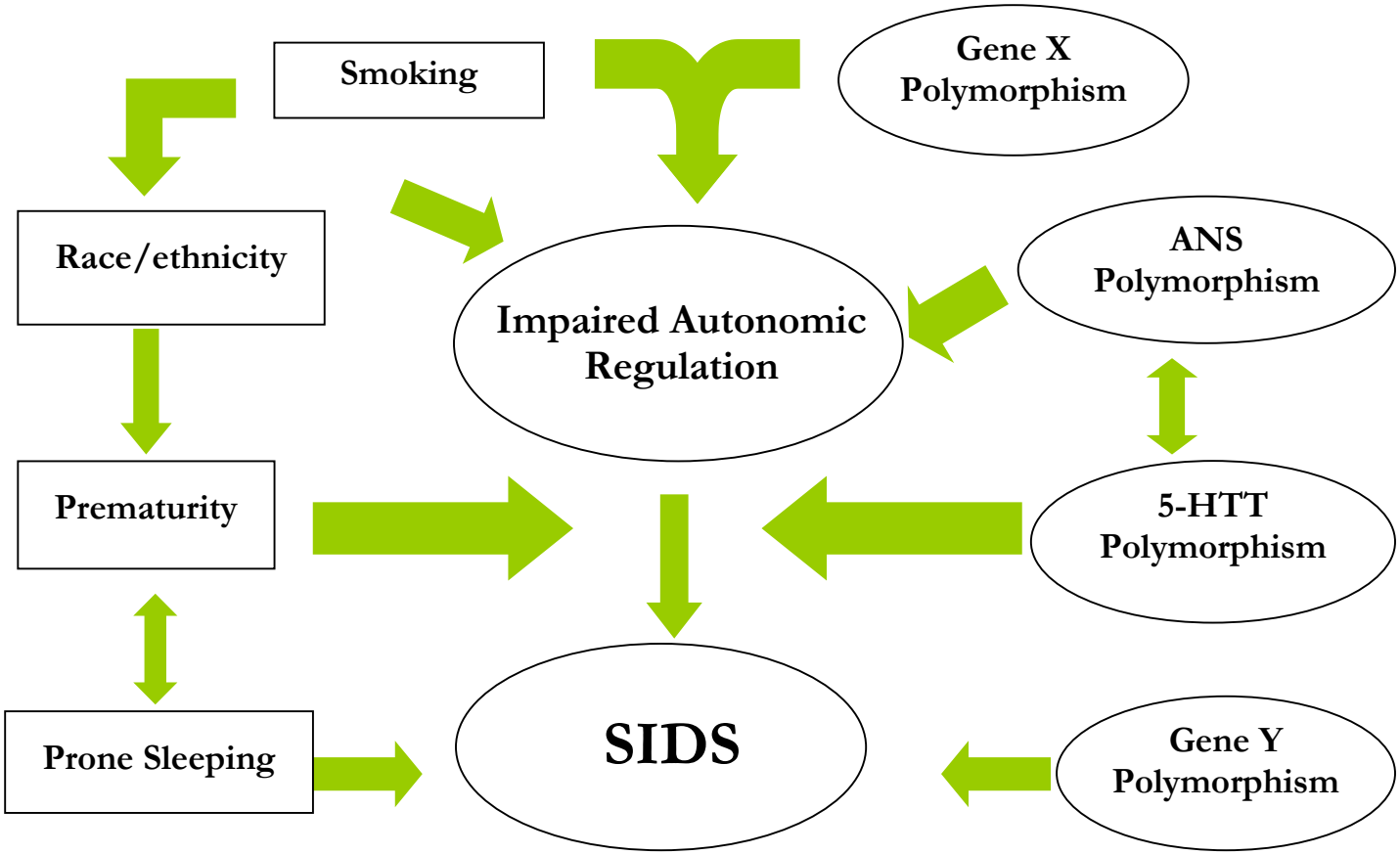

Figure: Schematic illustration of some interactions between adverse environmental and genetic risk factors potentially associated with SIDS. ANS: Autonomic nervous system. 5-HTT: 5-Hydroxytryptamine (serotonin) transporter

Developmental neurotransmitters pathology in SIDS

Sachio Takashima, Yuri Ozawa, Japan

The campaign of sleep position in supine state has reduced the incidence of sudden infant death syndrome (SIDS), but SIDS is still the main cause of postnatal infant death. The causes and mechanisms in SIDS have not been elucidated. SIDS has the characteristics of occurrence time in early infantile period and in sleeping state. Previous studies on the brain of SIDS have revealed ischemic lesions of subcortical leukomalacia and diffuse astrogliosis in the white matter, suggesting chronic or repeated hypoxia. Also, the brainstem pathology of SIDS infants has shown gliosis in respiratory centers, developmental delay of dendritic spines and synapses, and abnormalitites of neurotransmitters, suggesting the possibility that abnormal development of the neuronal circuitry in the brainstem, the neurons of which regulate rhythmic breathing and arousal, underlies cardiorespiratory instabilities.

In the SIDS infants with brainstem gliosis, substance P was increased in the pons and medulla oblongata, suggesting the close relationship between chronic hypoxia and neurotransmitter development in SIDS.

Development of catecholaminergic neurons and serotonergic neurons in the brainstem of controls and SIDS was performed by the immunochemistry of tyrosine hydroxylase (TH), tryptophan hydroxylase (TyH) and their receptors. By comparative immunohistochemistry of TH and TyH between SIDS and control infants, catecholaminergic neurons were found to be decreased in vagal dorsal and solitary nuclei of the medulla oblongata and serotonergic neurons were also reduced in the periaqueductal gray matter (PAG) of SIDS infants. α2-adrenergic receptor(α2-AR) showed devemopmental changes and was decreased in the medulla oblongata compared with controls. These may be related to some developmental impairments of the cardiorespiratory neuronal system.

The immunoreactivities of 5-hydroxytryptamine (5-HT)1A and 5-HT2A recepters in the brainstem also showed developmental changes of an increase from the early fetal period and gradually decrease in the ventrolateral medulla and PAG. In SIDS infants, both receptors were significantly decreased in the vagal dorsal and solitary nuclei and ventrolateral medulla of the medulla oblongata, and increased in the PAG of the midbrain compared with in age-machted controls. The increase in the PAG may exhibit delayed neuronal maturation or a reaction to the function of lower respiratory center.

These nuclei comprise a common circuitry for the CNS control of breathing. 5-HT acts specifically on structures in the rostral VLM, which has been assumed to be a respiratory rhythm generator, but not diffusely on the neurons of the ventral respiratory group. The respiration-related nuclei located in upper parts of the CNS may develop later and control the cardiorespiratory circuitry in lower parts of the brainstem. Although the origin of the serotonergic input to the central gray matter has not been studied in primates, a study on the rat has indicated that the projections arise from pontine and medullary reticular and raphe nuclei. So the increases of 5-HT1A and 5-HT2A receptors in the PAG may be the reaction associated with the serotonin decrease in the medulla oblongata and pons.

In the pathogenesis of SIDS, there are predisposing and environmental factors. Recently genetic studies disclosded some responsible genes such as SCN5A channel and serotonin transporter as predisposing factors, while neuropathological studies revealed subtle developmental or dysplastic abnormalites in the medulla oblongata, cerebellum and others.

The changes of neurotransmitters may be secondary ones induced by chronic hypoxia or repeated ischemia in SIDS, but may be causally related to some impairment of the developing neuronal system, which may exhibit a development abnormality of the antinociceptive reaction of cardiorespiratory control and arousal response mechanism in SIDS.

Serotonin Symposium
Larry Becker Memorial Lectures

The Larry Becker Memorial Lectures are in honour of the great Canadian scientist whose devotion to SIDS has contributed so enormously to the work of the Canadian SIDS Foundation. This session will focus on the theme of serotonin. Dr Toshiko Sawaguchi (Japan) and Dr Hannah Kinney (USA) have teamed up with the scientific committee to organize the symposium.

Contributions to SIDS Brain Research

Hannah Kinney, United States

Dr. Larry E Becker was an extraordinary pediatric neuropathologist and colleague. He made many outstanding contributions to our understanding of brain tumors, mental retardation, inborn metabolic disorders, and muscle disease in children. He is most remembered by this community for his important contributions to SIDS brain research. In this memorial lecture, Dr. Becker's work concerning the neuropathology of SIDS will be reviewed in light of his many insights into deciphering SIDS.

Serotonin transporter gene polymorphism as a risk factor of SIDS

Masaaki Narita, Naoko Narita, Japan

Sudden infant death syndrome (SIDS) is defined as "the sudden death of infant which is unexpected by history, and in which a full postmortem examination fails to demonstrate an adequate cause of death." Dysfunction of neurotransmitter serotonin (5-hydroxytryptamine) has long been proposed for the pathogenesis of SIDS, although precise role(s) of serotonin, including involvement of serotonin-related genes in SIDS, has not been known.

Recently, we first identified genetic risk factor for SIDS (Pediatrics, vol. 107, No.4, 690-692. 2001) by analyzing the association between serotonin transporter gene (5-HTT) polymorphism and SIDS. The polymorphisms in the 5' regulatory region of 5-HTT (5-HTTLPR) was first reported by Lesch et. al., (Science, 274, 1527, 1996), and are composed of 14 ("S allele" for short), 16 ("L allele" for long), or rarer frequent 20 ("XL allele" for extra long) repetitive elements. This polymorphism is also known to regulate 5-HTT transcriptional activity, depending on the number of the repetitive elements. In our study, we studied the distribution of 5-HTTLPR in 27 Japanese SIDS victims and 115 Japanese age-matched health control participants. Genomic DNA was obtained from the whole blood and 5-HTTLPR was analyzed by the number of repetitive elements using PCR amplification of the DNA Significant differences in genotype distribution and allele frequency of the 5-HTT promoter gene were observed, namely, the L and XL were statistically more frequently found (22.2 % and 5.6 %, respectively) in SIDS victims than in age-matched control participants (13.5 % and 0.4 %, respectively). These findings were further confirmed by the study of US group (Weese-Mayer, et al., Am J Med Gen, 2003).

Since the activity of serotonin (i.e. extracellular serotonin) is regulated by 5-HTT, of which transcriptional activity is influenced by 5-HTTLPR, these results indicate that (1) genetic factors are, at least in part, involved in SIDS (2) serotonergic abnormalities might exist in SIDS.

We thus propose that the longer alleles (L and XL alleles) are genetic risk factor for SIDS. These results might lead to the neonatal genetic screening for the prevention of SIDS.

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Serotonin neurons as sensors of blood CO_2: Role in control of breathing

George B Richerson, United States

SIDS has recently been linked to abnormalities of serotonin receptors and the serotonin transporter (Panigrahy et al. 2000; Narita et al. 2001). However, it is not currently known how a defect in the brainstem serotonin system could cause SIDS. SIDS has also been linked to defects in CO_2 chemoreception, breathing and arousal (Hunt and Brouillette 1987), suggesting that the brainstem serotonin system may be involved in these processes.

We are studying the basic neurobiology of serotonin neurons in rats and mice. Since the function of these neurons in animals is likely to be similar to their function in humans, defining their normal role in rodents may help to determine how a defect in these neurons could lead to SIDS. We have found that serotonin neurons have the properties expected for sensors of arterial CO_2 levels, and have proposed that their primary role is to initiate a variety of reflexes aimed at returning CO_2 to normal. This CO_2 control is important, because CO_2 is locked in equilibrium with pH through the chemical reaction $CO_2 + H_20 \leftrightarrow H^+ + HCO_3$, and even a small increase or decrease in pH can be fatal. The primary short-term mechanism for control of CO_2 is through lung ventilation. Equally important for CO_2 control in a sleeping individual is the arousal that allows correction of airway obstruction, if it exists. Our evidence supports a role of serotonin neurons in both processes: breathing and arousal (Richerson 2004).

Using patch clamp recordings from neurons in rat brain slices, we have found that serotonin neurons increase their firing rate in response to an increase in CO_2 (Richerson 1995). This response is preserved when these neurons are isolated in tissue culture (Wang et al. 2001). The response to CO_2 is mediated by the resulting decrease in pH, and it is physiologically-relevant because neurons increase their firing rate an average of 3-fold in response to a decrease in pH from 7.4 to 7.2. Further supporting a role as central CO_2 chemoreceptors, the dendrites and bodies of serotonin neurons wrap around large arteries in the brainstem, allowing close monitoring of blood after it leaves the lungs. This relationship with arteries occurs in both the medulla (Bradley et al. 2002) and midbrain (Severson et al. 2003). Since serotonin neurons in the medulla stimulate breathing and those in the midbrain induce arousal, it follows that a rise in blood CO_2 would stimulate breathing and induce arousal in part via effects on serotonin neurons.

The basic neurobiology of serotonin neurons suggests a model for a subset of SIDS that is consistent with the model of Hunt & Brouillette (Hunt and Brouillette 1987) as well as the triple risk theory of Filiano and Kinney (Filiano and Kinney 1994). A defect in serotonin neurons due to environmental or genetic factors, when combined with the normal immaturity of the brainstem and the normal instability of breathing during sleep, may lead to a decreased ability to recognize the rise in CO_2 that occurs when the face is covered by bedding. An infant would not respond by waking up and breathing more, which might result in a severe increase in CO_2, decrease in pH, and hypoxia. Such an infant may appear healthy while awake, since the brainstem serotonin system is more active during wakefulness (Jacobs and Azmitia 1992), permitting relatively normal CO_2 control while awake. These findings suggest that animal models with defects in the serotonin system (Hendricks et al. 2003) may be appropriate for studying the mechanisms of a subset of SIDS cases.

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Relationship between Serotonin related Factors and Neuronal Plasticity in Sudden Infant Death Syndrome

Toshiko Sawaguchi, Patricia Franco, Hazim Kadhim, Jose Groswasser, Hiroshi Nishida, Andre Kahn

Aim

Recently a Serotonin-related pathophysiological hypothesis on SIDS was proposed. On the other hand, a recent report suggests that abnormality neuronal plasticity detected by growth-associated phosphoprotein 43(GAP43) was possibly associated with the arousal pathway in sudden infant death syndrome(SIDS). This study tested additional pathological stainings that could show changes of serotonin-related factors and neuronal plasticity and the presence of sleep apnea was correlated with the neuropathological findings for each infant. In addition, the latent relationship between serotonin related factors & neuronal plasticiry in SIDS has been investigated in this study.

Materials and Methods

Among 27000 infants studied prospectively to characterize their sleep-wake behavior, 38 infants died under 6 months of age including 26 infant victims of SIDS. The frequency and duration of sleep apnea (obstructive and central) events recorded some 3-12 weeks before the infants' death were analyzed.

Brainstem materials from these 38 infants was studied in an attempt to elucidate the relationship between sleep apnea and neuro-pathological changes in the arousal pathway. The quantitive histochemical analysis included immunohistochemistry of tryptophan hydroxylase (TrypH) and 5HT1A receptor as markers of serotonin-related factors were carried out and the density of reaction positive neurons were calculated. The quantitative histochemical analyses included Bielschowsky staining positive neurofibrae and the immunohistochemical analyses included the evaluation of the density of synaptophysin positive neurofibrae and the density of microtubule-associated protein 2 (MAP2) positive dendritic spines as markers for synaptic plasticity were carried out. Pathological and physiological data were linked for each infant and non-parametric (Kruskal-Wallis) and correlation analyses were carried out.

Finally, correlation analysis between GAP43-positive findings and the TrypH-positive neurons was carried out.

Results

One significant positive correlation between the density of Tryp-H positive neurons in the dorsal raphe nucleus of the midbrain and the duration of central apnea($p=0.027$) was found in SIDS victims.

As the result of the non-parametric test, the density of MAP2 positive dendritic spines in the pedunculopontine tegmental nucleus(PPTN) were statistically significant higher in the SIDS group than in the non-SIDS group($p=0.0314$).

As the result of the correlation analyses between physiological data and pathological data, some SIDS-specific significant correlation were found as follows:

1. The negative correlation between the frequency of central apnea and the density of Bielschowsky-positive neurofibrae in the dorsal raphe nucleus in the midbrain(MBDR)($p=0.010$) and in the periaqueductal gray matter in the midbrain(MBPG)($p=0.010$).
2. The negative correlation between the frequency of obstructive apnea and the density of Bielshowsky-positive neurofibrae in the MBDR($p=0.026$).
3. The negative correlation between the frequency of central apnea and the density of synaptophysin-positive neurofibrae in the PPTN($p=0.006$).
4. The negative correlation between the duration of obstructive apnea and the density of MAP2-positive dendrites in the PPTN($p=0.017$).

No significant correlation between GAP43-associated findings and TrypH-positive neurons.

Conclusion

These findings suggest that serotonin related factors and neuronal plasticity in the brainstem arousal pathway might be related with SIDS independently. Further investigation should be carried out for the correlation between serotonin related factors and neuronal plasticity.

Symposium on Upper Airways

From the earliest days of research on SIDS, upper airway obstruction has been a hypothesis to explain the unexpected deaths. Upper airway development and physiology have generated much interest. Repeated episodes of airway obstruction in infants appear to lead to decreased arousal responses and altered cardiovascular autonomic control. In turn, decreased arousal responses and altered cardiovascular autonomic control have been implicated in the pathophysiology of SIDS. In this session, scientists from various fields will present an overview of the role of upper airways in the context of SIDS.

Evidence for the Involvement of Obstructive Sleep Apnea in SIDS

Bradley Thach, United States

Is acute upper airway obstruction a significant stressor causing SIDS? If so, is obstructive sleep apnea (OSA) a primary cause of upper airway obstruction in SIDS? Upper airway obstruction has long been a leading theory for cause of SIDS (1). Evidence for this comes from multiple studies. This evidence is summarized here: 1) The vast majority of SIDS infants are presumed to die during sleep and OSA is entirely a sleep dependant disorder (1). 2) Severity of OSA is increased by increased nasal resistance such as occurs with viral upper airway infection which is itself a risk factor for SIDS (2). 3) Certain craniofacial morphological features are strongly associated with increased risk for OSA (2). Significantly, such abnormalities are reportedly increased in SIDS infants (3,4,5). Furthermore, sudden death from OSA is well documented in infants with these abnormalities (eg. micrognathia) (6). 4) A large epidemiological study found that a family history of OSA is a risk factor for SIDS (7,8). 5) A prospective polygraphic study of sleeping infants found that episodes of obstructive apnea were more common in infants who ultimately died of SIDS compared to survivors (9). 6) The distribution of thymic petechiae in SIDS victims suggests that airway obstruction is a precipitating event in 70 % of SIDS cases (10). Finally, new physiologic evidence links OSA with the prone sleep position (see below). All in all, we conclude that there is strong, albeit indirect, evidence indicating that OSA either predisposes and infant to SIDS or actually acts as the final critical stressor causing death. Preliminary data from our lab provides direct evidence suggesting that complete airway obstruction immediately precedes death in some SIDS cases (see Pylipow M, Harris K, Thach BT, Abstract presented at this meeting). Whether or not this represents OSA or some other entity is unclear. In any case, this seems worth pursuing with additional studies of fatal event recordings in SIDS infants.

Is OSA more likely to occur in prone sleeping infants as compared to other sleep positions? If so, what is the mechanism? Clinical experience suggests that infants with micrograthia are more likely to experience life threatening airway obstruction when sleeping prone. We have assumed from this that OSA could not be linked to the prone position. However, this view is brought into question by recent evidence from Isono et. al. indicating that passive compliance (collapsibility) of the infant's pharyngeal airway is increased in the prone position (11). Clearly this should predispose infants to OSA when infants sleep prone (12). Accordingly, Isono proposed that increased risk of OSA could be a mechanism for the increased SIDS risk in prone sleeping infants. However, passive compliance is only one property of the pharyngeal airway that predisposes to OSA (2,12). The action of pharyngeal dilating muscles and associated reflexes are also important. Isono's measurements were performed in paralyzed infants prior to surgery. Also important is studies of sleeping infants. In the past we found that, in addition to neck flexion, very slight pressure applied to a sleeping infant's neck, much less than that required to interrupt sleep is sufficient to completely occlude the pharyngeal airway (13). It is possible that this can occur naturally. It is well established that increased body mass index (BMI) and increased neck circumference are both risk factors for obstructive sleep apnea (2). Noteworthy is that BMI falls at birth then rapidly rises (30% increase) to a peak at 3 to 4 months of age, the peak risk period for SIDS (14). Previously, studies from our lab showed that one

mechanism for increased OSA risk with increased BMI is increased pressure on the pharyngeal airway imposed by thick neck adipose tissue (15). We also have observed that many infants accumulate fat around their necks during the first 6 postnatal months, which is consistent with their increasing BMI. It is possible that certain head positions cause increased pressure in soft tissues surrounding the infant's neck when sleeping prone and that this can cause pharyngeal closure analogous to that occurring with neck flexion. Of course since arousal is the primary mechanism producing recovery from obstructive apnea episodes failure of arousal would be a factor in deaths during obstructive apnea episodes.

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Anatomy of the Upper Airway in Infancy - and its Possible Relevance to SIDS

Shirley Tonkin, New Zealand.

Firstly, I would like to pay tribute to SIDS parents whose sad words have given me the ideas that may explain the mechanism of some of these tragedies - and thus suggest ways to lessen the chance of future deaths.

One parent told me that when she picked up her prone sleeping baby the bottom part of his face ' was all squashed in '.

Investigations showed that this jaw movement was indeed possible in early infancy.

How can the lower part of a baby's face become deformed?

The infant head at birth is about 1/4 of the whole body length – adults heads are 1/8 of the height. Infant spines are almost straight, the head sits on top like a toffee apple on a stick. To lie flat on the back the infant head must be flexed forwards.

Adult spines have two curves forwards - at the neck , and at the pelvis. Thus the back of the head is in line with the back of the shoulders in early adulthood and a person can lie supine without neck flection.

The infant's cranium is almost adult size, but the lower face is very small and the mandibular articulation with the cranium is unstable as there is no mandibular head, and no glenoid fossa th limit backward movement.

The infant jaw has strong muscles for suckling with a biting motion. Within the infant jaw is situated the almost adult sized tongue which provides the infant with the ability to swallow liquids. It is in close proximity to the back of the pharynx, and thus forms a portion of the anterior wall of the upper airway.

The size of the airway will thus depend upon the size of the jaw - and the relative size of the tongue. Both are variable. Babies born with very small jaws (micrognathia) have been shown to have more breathing problems than normal babies.

So the size and thus the function of the upper airway is vital for the delivery of oxygen to the lungs. Upper airway size can be compromised by birth abnormalities of the passage - or of the mandible.

It can be influenced by the muscle relaxation of sleep.

It's size can be reduced by suction within the infant's airway - or by pressure from without. External pressure can come from outside agencies, or from the infant's own body.

We have shown with imaging techniques that these circumstances do occur - and also the compromise of the upper airway can cause oxygen desaturation and severe bradycardia.

Could this be the mechanism of many SIDS deaths?

Genetic Diseases and Syndromes Affecting Upper Airways

Anat Shatz, Israel

Upper airways disorders may be responsible for sleep-disordered breathing (SDB), obstructive apneas, apparent-life-threatening events (ALTE) or sudden infant death, as well as for partial airway obstruction episodes or upper airway resistance syndrome. Upper airways disorders may include bone malformations, soft tissue infiltration, and neurological lesions.

A large number of genetic disorders manifest SDB. Understanding how these genetic diseases and syndromes affect sleep and breathing may shed light on pathogenetic and regulatory mechanisms of SDB.

We can classify genetic disorders that manifest SDB into 3 major subgroups:

1. Craniofacial malformations;
2. Autonomic nervous system (ANS) dysfunction;
3. Disorders of the neuromuscular system

Research into craniofacial malformations can shed light on the anatomical mechanisms for upper airway obstruction Children with craniofacial anomalies may experience obstructive apneic episodes during sleep due to a variety of causes, including maxillary hypoplasia with a resultant narrow nasopharyngeal airway (Crouzon and Apert syndromes), retrognathia (Treacher Collins syndrome), and palatopharyngoplasty performed in a child with velopharyngeal incompetence (VPI) and glossoptosis (Pierre Robin, Treacher Collins).

ANS dysfunction is found in many pediatric genetic disorders. Studying its pathophysiology could improve our understanding of the maturation of the ANS and the abnormalities that occur in SDB. Many children with neuromuscular system disorders exhibit dysfunction of the respiratory and upper airway musculature that contributes to the development of SDB.

In certain instances, genetic disorders manifest SDB due to the combined effect of two or all of the three subgroups, namely anatomic malformation, neuromuscular weakness, and/or ANS dysfunction.

Down syndrome (trisomy 21), a common genetic syndrome, is an example of such combination. Here, an array of congenital structural airway anomalies is combined with generalized hypotonia and a high rate of atlantoaxial instability.

Another example is congenital central hypoventilation syndrome. This rare respiratory control disorder may be presented by apneas, mostly long central apneas. However, this syndrome may also be presented by obstructive apneas related to abnormal upper airway muscle control (1). SDB and symptoms of ANS regulation disorders have also been found in Rett syndrome and Familial Dysautonomia (2). Other examples of genetic and familial craniofacial syndromes will be reviewed.

In a recent study of infants presenting with apnea and findings of pharyngeal wall collapse we found combinations of anatomical anomalies and neuromuscular disorders. Of 50 infants studied, 4 (8%) had rare genetic congenital anomalies (e.g., Aniridia) and 16 (32%) exhibited isolated other anatomical anomalies (e.g., micrognathia, high and narrow palate, macroglosia) contributing to "small airways" (3). In this study, 80% of the infants had mild generalized hypotonia. Of the 25 infants (50%) who underwent polysomnographic studies, 96% had obstructive sleep apnea (OSA). Other studies reported clinical symptoms of obstructive apneas among family members of infants who succumbed to Sudden Infant Death Syndrome (SIDS) or had near-miss incidents, apparently related to the presence of a small posterior airway (4). Tishler *et al* showed SIDS/ALTE families exhibited higher frequency of brachycephalia and reduced dimensions of the oral-pharyngeal airways (5). Guilleminault et al (6) found that familial sleep-disordered breathing was common in ALTE infants presenting mild facial dysmorphia. This study points out that ALTE may be an indication of a sleep disordered breathing syndrome. Infants with ALTE may have an unrecognized increase in upper airway resistance long before having the ALTE episode (7).

Studies of the role of upper airways in SDB provide clinically significant conclusions: infants with narrow upper airways (as in craniofacial malformation) are at higher risk for obstructive apneas; ALTE may be related to upper airway anomalies; and infants with congenital central hypoventilation syndrome may present with unexplained obstructive apneas.

These studies also lead to some practical recommendations:

a) Infants presenting with ALTE or SDB should have clinical evaluation of craniofacial features and upper airway patency (8);
b) Family history should be carefully examined and noted;
c) Polysomnographic examinations should be performed; and
d) Families with sleep-disordered-breathing should undergo genetic tests.

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Infant and environmental factors favoring the development of obstructive sleep apnea

André Kahn, Belgium

Prenatal cigarette smoking has been shown to induce several changes in the respiratory, cardiac and arousal control mechanisms in newborn infants. Infants of mothers who smoked during pregnancy have more frequent and long obstructions of the upper airways during sleep (called obstructive sleep apneas). Similar apneas are relatively rare in infants of non-smoking mothers (Kahn 1994). A dose-response pattern exists between the number of cigarettes smoked during pregnancy and the frequency of obstructive sleep apnea. Paternal smoking contributes to the risk of apneas only if the mother is also a smoker. Maternal smoking after birth does not add significantly to the risk of apnea. Infants born to mothers who are smokers also have a reduced drive to breathe and a blunted ventilatory response to hypoxia (Lewis 1995). These depressed respiratory mechanisms add to the breathing difficulties associated with repeated airway obstructions.

Newborns and infants of mothers who are smokers have higher arousal thresholds to environmental noises and tactile stimulation than those born to non-smoking mothers (Franco 1999, Horne 2002). The infants of smoking mothers are not as easily aroused from sleep as those of non-smokers. These airway obstructions and arousal characteristics can be observed at an early age. The impact of exposure to cigarette smoke thus occurs before birth.

Prenatal smoking is also associated with changes in cardiac function (Franco 2000). Infants of mothers who are smokers have higher basal heart rate and a smaller heart rate variability than infants of non-smokers. These characteristics result from a significant increase in sympathetic vagus nerve control of cardiac autonomic function. Similar changes are seen in acute or chronic stress conditions. Similar breathing patterns, heart rate and arousal characteristics were found in infants who eventually became victims of Sudden Infant Death (Kahn 1992).

In post-mortem studies, infants of mothers who are smokers show specific increases in the density of brain stem nicotine receptors. These changes are correlated with maternal smoking and occur in areas that regulate vital autonomic functions, such as breathing, cardiac or arousal controls.

Suggested reading

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July 5, Morning sessions

Tobacco and Smoking

Maternal smoking during pregnancy and passive exposure to smoking after birth have long been recognized as risk factors for SIDS. Yet despite recent advances in the identification of modifiable risk factors, as well as the success of the "Reduce the risk" and "Back to sleep" campaigns throughout the world, the task of actually decreasing the fœtus and newborn exposure to smoking has prove to be difficult. Now that the goal of modifying an infant's sleeping position has largely been achieved, we must concentrate on the next key risk factor.

The scientific sessions will highlight new research findings in three major areas relevant to tobacco exposure in infants (pre- and postnatal): infection, heart lesions and tissues nicotine levels (in relation to risk factors). The health professional stream contribution will focus on "Tobacco use among pregnant and parenting smokers."

Introduction

Ed Mitchell, New Zealand.

There are now over 50 cohort and case-control studies that have investigated the relationship between maternal smoking in pregnancy and sudden infant death syndrome (SIDS). In a recent meta-analysis the pooled relative risk associated with maternal smoking before the intervention programmes was 2.91 (95% CI=2.82, 3.01), and after was 4.67 (95% CI=4.04, 5.35). Adjustment for potential confounders lowers the risk estimate; however, many studies over-adjust, e.g. by controlling for birthweight, which is also affected by smoking, resulting in an inappropriately low estimate of the risk. The increased odds ratio for maternal smoking since the reduction in the prevalence of prone sleeping was unexpected. The increase in the risk of SIDS associated with smoking is not as great for infants who sleep prone as it is in infants sleeping non-prone. Thus with the reduced prevalence of prone sleeping, the overall odds ratio for smoking will increase. The biological explanation for this observation is unknown.

Epidemiologically it is difficult to distinguish the effect of active maternal smoking during pregnancy from postnatal passive smoking by the infant; clearly these two possibilities would have quite different implications.

Most attention has been directed at the mother's smoking behaviour, however 13 studies have examined smoking by fathers. The pooled relative risk associated with paternal smoking was 2.31 (95% CI=2.09, 2.59). However, as maternal smoking behaviour is associated with smoking by the father, it is important to control for maternal smoking when examining this. An alternative method is to examine the effect of father smoking where the mother is a non-smoker. There have been six such studies. The pooled relative risk for the father alone smoking was 1.39 (95% CI=1.11, 1.74).

A number of tentative mechanisms by which maternal smoking may increase the risk of SIDS have been proposed, but so far none of them has been conclusively proven. A reduction in birthweight has long been recognised as a consequence of maternal tobacco use and low birthweight is an established risk factor for SIDS. Exposure to tobacco products during fetal life restricts visceral organ growth and alters the neural control of autonomic, behavioural and homeostatic functions and impairs cardio-respiratory defence mechanisms, such as arousal mechanisms.

Population attributable risk (PAR) is the estimated proportion of cases that can be attributed to a risk factor. For maternal smoking in pregnancy, using the lower pooled relative risk (before intervention programmes: OR=2.91) and a conservative estimate that 25% of mothers smoke in pregnancy, then the PAR is 0.32. The same calculation for the higher pooled relative risk (after the intervention programmes: OR=4.67) gives a PAR of 0.48. This suggests that SIDS mortality might be reduced by between a third and a half if no fetus was exposed to maternal tobacco smoke. In contrast the PAR for father's smoking where

the mother is a non-smoker is just 0.06 (OR=1.39 and proportion exposed=0.15).

In conclusion, smoking is causally associated with SIDS, although which of the many mechanisms is the most important is uncertain. Other speakers in this symposium will address possible mechanisms and smoking cessation.

Smoking, Infection and SIDS

Caroline Blackwell, Australia

Smoking is a major risk factor for SIDS and also for serious bacterial and viral diseases in infants and young children. The term passive exposure to cigarette smoke somehow implies that this route of exposure is less harmful than active smoking. Cotinine is a breakdown product of nicotine produced by the liver. Studies in Australia found that some infants exposed to cigarette smoke had levels of cotinine similar to those of active smokers [1]. This indicates that interactions between cigarette smoke and factors contributing to susceptibility to infection in active smokers might also apply to children who are "passive" smokers. Cigarette smoke affects two important steps leading to infectious diseases: 1) colonisation of mucosal surfaces by pathogenic bacteria; 2) induction or control of the inflammatory responses to micro-organisms or their toxins.

Colonisation: Infants obtain their bacterial flora from their mothers. Smokers more often carry potentially harmful bacteria in their nose and throat than non-smokers. Smokers are also more likely to harbour *Staphylococcus aureus,* bacteria that produce powerful toxins which we identified in tissues over half of SIDS infants from 5 different countries [2]. Close prolonged contact such as bed sharing could increase opportunities to pass bacteria from mother to baby and help explain why the combination of these two factors increase the risk of SIDS.

Cells from the mouths of smokers bound significantly higher numbers of many species of bacteria that cause infections in young children [3]. Tar in cigarette smoke might be one of the "sticky" components responsible. Cigarette smoke also makes adults and children more vulnerable to respiratory virus infections. Infection of HEp-2 cells with either respiratory syncytial virus (type A or B) or influenza virus (type A or B) significantly enhanced binding of a range of bacterial pathogens and significantly up-regulated expression of host cell antigens that act as receptors for bacteria [4,5].

Inflammatory responses: There is increasing evidence that pro-inflammatory cytokines play a role in triggering the physiological disturbances leading to SIDS. Both environmental and genetic factors affect the levels of pro- and anti-inflammatory cytokine responses to bacteria and their toxins. Leukocytes of smokers produced significantly lower levels of the anti-inflammatory cytokine interleukin-10 (IL-10) to bacterial toxins [6]. Parents of SIDS infants had higher interleukin-1β (IL-1β) responses to toxic shock syndrome toxin (TSST) compared with unrelated control adults. Smokers in both groups had significantly lower responses to both TSST and endotoxin [6]. We examined the interactions between smoking and cytokine gene polymorphisms in the IL-1β (C-511T) and IL-10 (G-1082A) genes. Smoking resulted in lower IL-1β responses from donors with the wild type genotype CC ($P = 0.00$) and the heterozygote CT ($p = 0.03$); however, it increased the response of the TT genotype [7]. Smoking consistently reduced the production of IL-10, but the most significant effect ($p < 0.01$) was observed for the AA genotype. Compared with European populations, the AA genotype is significantly increased (>80%) among both Aboriginal Australians and Bangladeshis ($p = 0.00$) [6]. If genetic makeup were a major factor for susceptibility to SIDS, the incidence of these deaths should be similar; however, they are significantly different. The incidence of SIDS and deaths due to respiratory infections was significantly lower in British Asian infants compared with infants of European origin [8, but both conditions are significantly higher among Aboriginal Australians [9]. The proportion of Asian women who

smoke is about 3% compared to Aboriginal Australian women (75%) [10,11].

Further studies on interactions between cigarette smoke and gene polymorphisms are needed to elucidate these interactions in relation to: the epidemiological findings for differences in the incidences of SIDS and serious infections in different ethnic groups; the cellular and molecular interactions that make cigarette smoke a risk factor for infection and SIDS; the role of cytokines in the pathophysiology of SIDS.

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Environmental tobacco smoke exposure and SIDS in Germany.

Thomas Bajanowski, Mechtild Vennemann, Germany

The *German Study on Sudden Infant Death* (GeSID) is a multi-centre case-control study aiming at the assessment of etiological and risk factors of SIDS including smoking. The information about cigarette consumption was obtained by parents interview.

To assess the relative risk for SIDS, the results obtained in 333 SIDS families were compared to 998 controls. Furthermore in 154 cases of sudden infant death (135 SIDS, 19 explained causes of death) nicotine concentrations (NC) in hair as well as cotinine levels (CC) in pericardial (PF) and cerebrospinal fluids (CSF) were determined using a specially adapted GC-MS method in the SIM mode. The results of chemical analyses were correlated to the data obtained during interview.

Results: Of the "SIDS mothers" 62.2% smoked during pregnancy. Therefore smoking during pregnancy in the "SIDS mothers" is about 3 times more frequent compared to a large random survey of pregnant women in Germany. Mothers who smoke during pregnancy did not change their smoking behavior after delivery. Maternal smoking during pregnancy was associated with an increased risk for SIDS (OR 3.0, CI 95% 2.0, 4.6).

Infants of mothers who smoked during pregnancy had higher NCs in hair than infants of non-smoking mothers ($p=0.008$). Furthermore there was a weak but statistically significant relationship between NC in hair and the daily cigarette consumption of the mother during pregnancy ($n=64$, $r=0.24$, $p=0.05$). Infants of mothers who smoked postnatal showed higher CCs in PF ($p=0.0009$) and CSF ($p=0.0002$) than infants of mothers who were non-smokers. CCs in PF were strongly correlated with CC in CSF ($r=0.62$, $p=0.0027$), and NC in hair was correlated with pericardial ($r=0.34$, $p=0.03$) or CSF cotinine ($r=0.36$, $p=0.02$). CCs 3 times higher than the average of the concentration determined in "heavy smokers" were detected in five infants indicating an intensive exposure of these infants directly before death or shortly before they were put to bed.

In SIDS cases showing respiratory tract infections, higher NCs than in so-called typical SIDS cases were not determined.

Conclusion: Maternal smoking during pregnancy is a dose-dependent risk factor for SIDS in Germany. Maternal cigarette consumption during pregnancy can be evaluated by determination of NC in infant's hair (a marker for exposure over weeks). The NC shows a

weak correlation to the cigarette consumption. Detectable CC in CSF or PF indicates an exposure over the previous few hours. There was no evidence that NCs and CCs differed between different categories of sudden unexpected infant death.

Pathology and prevention of unexpected perinatal death and SIDS

Luigi Matturri, Italy

Among the many factors predisposing toward, albeit not determining, sudden infant death syndrome (SIDS), parental cigarette smoking has a predominant role. Indeed, the data in literature indicate that parental smoking is the major risk factor not only for SIDS but also for unexpected fetal death (1,2).

Our anatomo-pathologic investigations of a vast series of unexplained perinatal deaths and SIDS have revealed frequent congenital anomalies, of a primary nature, both of the brain stem nuclei which modulate respiratory, cardiovascular and initial digestive tract activities and of the cardiac conduction system, in both SIDS and unexpected perinatal death victims. These findings seem to indicate a link between these congenital anomalies and a presumably genetic basis (3-10).

Study of the risk factors has demonstrated that the combustion products of cigarette smoke have a role in both atherogenesis and neural damage. We recorded a high incidence of initial atherosclerotic lesions in the coronaries of fetuses with smoker mothers (11). If the maternal smoking habit persists, true plaques can be observed in the infant, which are accentuated if the effect of nicotine is associated with that of artificial milk, due to a synergic action of the two atherogenic factors (12,13).

Regarding the harmful effect exerted by the combustion products of tobacco, a significant association was found in our series between hypoplasia of the arcuate nucleus and maternal smoking habit during pregnancy (6).

All the above lesions can be attributed to the direct action of the combustion products of nicotine on the smooth muscle cells of the tunica media of the arterial walls and/or on the neurons, interfering with homeostasis and cell differentiation, as well as to an indirect action of hypoxemia induced by arteriosclerosis.

These results suggest that the significance of the other risk factors considered in the pathogenesis of these diseases should be revised, especially the question of the newborn's position in the cot, which has been assigned a fundamental importance in recent years that is not supported by anatomo-pathologic data.

Analysis of our series shows that it is essential for a complete autopsy to be performed, only by an experienced, reliable pathologist, in SIDS victims, including in-depth histological analysis of the cardiorespiratory innervation and specialized myocardium, as described in our web site: http://users.unimi.it/~pathol/sids/riscontro_diagnostico_e.html and as established at the 7th SIDS International Conference in Florence, Italy (14). In fact, our autopsy data showed borderline lesions or lesions of a different nature in 15-20% of cases, leading us to exclude the diagnosis of SIDS.

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Tobacco and Smoking: Treating Tobacco Use among Pregnant and Parenting Smokers

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Despite evidence of increased risk to themselves, their pregnancies and their families, a significant proportion of pregnant women continue to smoke throughout pregnancy. Similarly, an even larger proportion of parents and caregivers continue to smoke around infants and children. Clinicians providing prenatal care for these women and pediatric care for their newborns and infants are in a unique position to offer clinically proven, effective interventions. The gap between what is known about how to treat pregnant and parenting smokers and what is actually done in clinical practice is large with fewer than half of all obstetricians surveyed in the US offering recommended treatment.

A growing volume of research since 1975 has demonstrated that clinically proven, effective interventions exist to produce long-term or even permanent abstinence from tobacco for all smokers. Achieving cessation is important for all smokers but especially for pregnant and parenting smokers since their smoking poses risks not only for themselves but also for their pregnancies and children. Treatments for smokers in general apply to parenting smokers but special considerations regarding treatment need to be made for pregnant women. Due to the harms associated with exposure to environmental tobacco smoke (ETS), or secondhand smoke (SHS), both parents and caregivers of young children should receive treatment to achieve cessation or counseling on how to eliminate exposure of children to ETS/SHS.

This presentation will provide a review of the recommendations made in 2000 by the United States Public Health Service regarding treatment for pregnant and parenting smokers, summarize recent findings that may impact treatment protocols and make recommendations regarding further research in treatment approaches for pregnant and parenting smokers. A summary of recommended changes in treatment approaches for clinicians based on this review and a description of factors affecting clinician adoption and use of proven treatments and systems supports found to increase the likelihood of clinician use of these treatments will be discussed.

Efforts underway in the United States to implement these recommendations will be presented with a

special focus on the work of the National Partnership to Help Pregnant Smokers Quit, the only national organization in the US working to mobilize health care systems and local communities to help pregnant smokers get the help they want and the support they need to quit smoking and remain tobacco-free. More than 60 member organizations of the National Partnership have joined forces to put evidence-based approaches to reducing tobacco initiation and use into practice at the national, state and local levels. The National Partnership's Action Plan, working group structure and evaluation process will be described including efforts to improve the healthcare system, use the media, promote community and worksite policies and programs, pursue federal and state policy initiatives and promote research, evaluation and surveillance.

Infection and Immunity

Researchers have long realized that the SIDS risk factors parallel those responsible for susceptibility to severe infections in infants and young children. They have noted, too, that both environmental and genetic factors affect anti-inflammatory responses to infection. Additional factors may well stem from inappropriate (perhaps heightened?) immune responses to otherwise innocuous common antigens and the resulting inflammatory processes. Dr Caroline Blackwell has put together the following session reviewing infection and immunity in the context of SIDS.

Evidence for inflammation and altered immune response in SIDS infants

Åshild Vege and Torleiv Ole Rognum, Norway

Signs of inflammation in SIDS infants are not a new finding. It dates back as early as 1889 when Arnold Paltauf (1) published his article about "Status thymolymphaticus" where he claimed that the SIDS infants had enlarged thymuses. He also stated that these infants had bronchitis. In the 1950s the notion of minimal inflammation in the airways was again brought to attention (2,3), and since then many authors (4,5,6) have reported that a large proportion of SIDS victims have signs of infection prior to death. In our first studies of SIDS infants and controls we found that the SIDS infants had significantly higher concentrations of hypoxanthine (Hx) in vitreous humour than the violent deaths (7), indicating that a significant proportion of the SIDS victims died after periods of hypoxia prior to death. Furthermore, we found that there were no significant differences between the Hx levels in SIDS and infectious deaths, indicating that there could be similarities in the death mechanism in SIDS and infectious death. We have also found increased concentration of interleukin-6 (IL-6) in the cerebrospinal fluid (csf) in half of the SIDS victims (8), as a sign of immune stimulation. This study demonstrated that there were two different populations of SIDS victims; one group with IL-6 levels similar to infants dying from serious infections; the other group with IL-6 levels comparable to violent deaths. Also other authors have pointed to the possibility of at least to different populations of SIDS victims (9). One such group could be related to sleep position and a possible thermal mechanism, the other to an uncontrolled inflammatory response to infection.

Overheating, as a result of infections and/or over-wrapping are well known risk factors for SIDS (10,11). Infections with bacteria or viruses can induce production of IL-6 (12,13) and in a rat model it has been shown that a passive warming of neonatal rat pups significantly increased IL-6 production and mortality. Many of the SIDS babies are overdressed and overwrapped and are inappropriately hot and sweaty when found dead (14). The IL-6 production that primarily may be a result of an overreaction to a slight infection may thus be aggravated by overheating imposed by external factors, which in

turn will increase the temperature further. There has been shown that a body temperature above 37°C almost always induces irregular breathing in infants (15), and that this gives an increased frequency of apnoeic episodes.

We have also found an increased immune stimulation in the laryngeal mucosa in cases with elevated csf IL-6 levels compared to cases with low such levels (16). The elevated IL-6 levels correspond to the levels found in infants that die from infectious diseases such as meningitis, septicaemia and pneumonia, although most of the SIDS cases only had slight symptoms of an infection prior to death. One third of the infants did not have any symptoms at all. These findings favour a hypothesis of an immunological overreaction to an otherwise harmless infection. This is also in accordance with the findings of Gleeson and co-workers (17).

We furthermore found that the babies found prone more often had symptoms of a slight infection and had a higher number of IgA immunocytes than babies sleeping on their side or back. A mild respiratory infection in an infant placed prone may thus induce laryngeal immune stimulation and exaggerate vagal reflexes, giving both bradycardia and irregular breathing. This may induce hypoxemia and in cases of inefficient gasping (autoresuscitation), the infant may develop severe hypoxia and finally coma and death. Failure of autoresuscitation may in part be due to deficits in the medullary serotonergic network, and interleukins may interact with this system.

A failure in the production of regulatory cytokines as interleukin-10 (IL-10), may further aggravate an over-stimulation of the immune system. This, acting in concert with different risk factors, could trigger the chain of events that constitutes the vicious circle (18)

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Development of Infant Mucosal Immunity in Relation to Vulnerability to Infections

Maree Gleeson, Australia.

In humans the systemic and mucosal components of the adaptive immune system, while interrelated, develop independently and at different ages. In association with innate immunity, the mucosal immune system provides the first line of defense against infections at external body surfaces. The human mucosal immune system comprises the lymphoid-associated structures of the nasal, bronchial, gastrointestinal and urogentinal tracts, the lachrymal, salivary and lactating mammary glands and the synovium of joints[1]. They form an interconnecting network through homing receptors that allow plasma cells activated at one site to be seeded to distant mucosal sites, thus providing extensive immune protection at mucosal surfaces[2]. Unlike the systemic immune system, the major class of antibodies in mucosal secretions is immunoglobulin A (IgA), which is secreted in a dimeric form (SIgA)[1]. The distribution of the two IgA subclasses of plasma cells varies at mucosal sites[3] reflecting differences in antigenic exposure: SIgA1 responds to protein antigens while SIgA2 responds to polysaccharide antigens. IgM is the other class of mucosal antibodies and is also transported across the mucosal epithelium in a secretory form (SIgM)[1].

Neonatal period

The anatomical structures of the mucosal immune system are fully developed *in utero* by 28 weeks gestation[4], but in the absence of intrauterine infection, activation does not occur until after birth. The gestational age at birth determines the degree of immune competence of the neonate, and the level of passively acquired maternal antibodies that provide effective defense against infections. The normal full-term infant is born virtually IgA-deficient. Mucosal immune responses occur rapidly in the first weeks of life in response to extensive antigenic exposure[5]. In addition to pathogenic or commensal bacteria and immunisation, the major modifier of the developmental patterns in the neonatal period is infant feeding practices. It takes several weeks before the mucosal lymphoid tissue responds effectively to antigenic challenge and the peak period for maturation of B cells at mucosal surfaces is from birth to 12 weeks of age[6]. The unresponsiveness of the mucosal immune system at birth is due to the combined effects of several factors[7]: hormonal influences during the birthing process are immunosuppressive (endogenous production of stress hormones); the immaturity of the neonatal antigen-presenting cells (dendritic cells) limits antigen processing[8]; and the maternally derived serum IgG antibodies and colostral IgA antibodies have immunosuppressive effects on neonatal antibody production.

The immediate postnatal period is also characterised by intestinal epithelium membrane permeability to intact macromolecules[9]. Ingestion of colostrum promotes membrane maturation in the gastrointestinal tract through regulatory factors[10] and results in closure within 48 hours of birth[11], an important process in limiting systemic exposure to antigens, which if handled inappropriately can lead to overwhelming infection, atopy, or subsequent tolerance to antigen recognition.

The First Year of Life

During the first 12 months of life the maturation of the mucosal immune system is dependent on the type and timing of antigenic exposure. The mucosal immune system is rapidly stimulated at birth by bacterial colonisation of the mucosal and external body surfaces. The development of effective mucosal immunity is essential for protection against infection in the postnatal period. In a healthy neonate, the pattern of appearance of antibodies in mucosal secretions is consistent with the rapid bacterial colonisation of the neonatal intestine. Oral feeding *per se* provides a stimulus for mucosal immune development and intravenously fed full-term infants are devoid of IgA- and IgM-containing plasma cells in the gut lamina propria[12]. SIgA appears in mucosal secretions between 1 week and 2 months of age[11]. Salivary IgA levels increase rapidly in the neonatal period to peak levels between 4-8 weeks of age and remain relatively consistent after 6 months of age until exposure to increased antigenic loads (*eg.* hospitalisation, childcare, schooling). The initial

bacterial colonisation patterns in the gastrointestinal tract differ between breast-fed and formula-fed infants. This significantly affects the degree and nature of antigenic stimulation of their mucosal immune systems. Colostrum deprivation also delays closure of mucosal membranes[12] and the protracted period of increased membrane permeability might contribute to the higher incidence of infections[13] and atopic diseases[14] observed in non-breast-fed infants. SIgM is also absent from mucosal secretions at birth in healthy full-term neonates but appears transiently between 1 and 6 months of age[15]. Total salivary IgM and specific IgM antibodies are occasionally observed in infants and adults in a pattern consistent with the concept that IgM antibodies reflect immune responses to novel antigens presented at mucosal sites. SIgM is usually present in high concentrations in mucosal secretions of IgA-deficient subjects in whom SIgM and IgG appear to play a compensatory role[16]. The appearance of specific SIgA antibodies in infants is dependent on the degree of vaccination or natural exposure to the antigen. Artificial colonisation of the intestine of newborns will also stimulate production of both SIgA and SIgM antibodies in stool and saliva and has been used as a mechanism to reduce infections and mortality in high-risk infants in intensive care units[17]. Malnutrition compromises host resistance to infection by reducing the availability of essential vitamins and trace minerals[18] and protein malnutrition is associated with decreased IgA responses to oral antigens and increased gastrointestinal infection rates[19].

Relationship to SIDS

A period of heightened immune responses occurs during the maturation process, particularly between 1-6 months, which coincides with the age range during which most cases of sudden infant death syndrome (SIDS) occur. A hyper-immune mucosal response has been a common finding in infants whose death is classified as SIDS[20], particularly if in association with a prior upper respiratory infection. Inappropriate mucosal immune responses to an otherwise innocuous common antigen and the resulting inflammatory processes have been proposed as factors contributing to SIDS. At the peak age of SIDS between 8-12 weeks, the mucosal antibody levels are often declining after the initial burst of response to commensal antigens, particularly in non-breast-fed infants[11]. It is also a time when many breast-fed infants are weaned, thus losing the protective effect of passive colostral antibody and the regulatory influence that breast-feeding exerts on the mucosal immune response[21]. It is possible during this period for an inappropriate uncontrolled inflammatory immune response to occur to an otherwise harmless antigen Interactions with other known risk factors for SIDS during this period could heighten the hyper immune response: the prone sleeping position (increase in airway temperature allowing colonisation); changes in night time cortisol levels associated with establishment of circadian rhythm (lack of suppression); and exposure to cigarette smoke (increase in bacterial load and adjuvant effect on immune response). The proinflammatory cytokine responses observed in SIDS infants[20] are most likely important links between the hyperimmune response, apnea and death. Uncontrolled mucosal immune responses to infections have been reported in a prospective case study of a SIDS death at 10 weeks of age[22]. In a recent study of infants admitted to hospital with acute life threatening events[23], suspected of being "near-miss SIDS", the induction of a hyperimmune salivary antibody responses to mild or unrecognized upper respiratory infections were a consistent finding. Genetically influenced control of the pro-inflammatory responses[24] induced by normally non-lethal infections, in conjunction with environmental factors, might explain why some infants are at-risk of SIDS and account for the ethnic differences in SIDS incidences throughout the world[20].

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The common bacterial toxin hypothesis for sudden infant death syndrome (SIDS)

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The common bacterial toxin hypothesis is that some cases of SIDS are due to the lethal effects of nasopharyngeal bacterial toxins that can act synergistically to trigger the events leading to death (1,2). The concept is consistent with the age distribution of SIDS, the winter excess of cases and the roles of prone sleeping and passive exposure to tobacco smoke. There is an increased isolation of staphylococci and gram negative bacteria, such as E. coli, in pernasal swabs from SIDS cases compared with age, gender and season matched healthy infants (3). Bacteria isolated from the nasopharynx of SIDS infants interact synergistically to cause rapid death in gnotobiotic weanling rats (4). Toxins from the same bacteria interact synergistically to cause death in chick embryos. Nicotine in very low doses potentiates the lethal effect of the toxin combinations (5). It has been shown, in both adults and infants, that secretions pool in the upper airways in the prone position leading to increased bacterial growth. In infants aged 12 to 18 months, suffering from viral upper respiratory tract infection, prone sleeping leads not only to increased bacterial growth in the nasopharynx but also to the appearance of gram negative bacilli, such as E. coli, as seen in SIDS infants at autopsy (6). Staphylococcal pyrogenic toxins (7), endotoxin and E. coli curli proteins (8) have been demonstrated in blood and tissues from SIDS infants. The most significant observation being the first in that staphylococcal pyrogenic toxins are only produced when the body temperature is elevated and therefore indicate a pre-mortem rather than an agonal or post mortem event.

The next steps should be to use the techniques of proteomics and genomics to search for evidence of bacteraemia and or toxaemia in fresh samples of fluid and tissue obtained as soon as death is ascertained. This will require considerable investment and needs to be undertaken on a national or international scale. Furthermore it is suggested that there is now good evidence that some cases of ALTE are near miss SIDS (9) and should be investigated for a bacterial and or toxaemic cause using similar methods. The advantage of investigating ALTE is that there are controls i.e. healthy infants; controls are never available in SIDS.

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Ethnicity, infection and SIDS

Caroline Blackwell, Australia

The risk factors for SIDS parallel those for susceptibility to severe infections in infants and young children. Among Indigenous groups in which the incidence of SIDS is high, there are also high incidences of serious respiratory and ear infections as well as meningococcal disease. We compared risk factors among groups in which there were medium, low and high incidences of SIDS: British Caucasians; Bangladeshi families in Britain; and Aboriginal Australians (Table) [1].

Risk Factor	Caucasian European	Bangladeshi	Aboriginal Australian
SIDS / 1000 live births	2	0.3	6.1
Prone sleeping	+	-	-
Mothers who smoke (%)	25	3	75
IgG levels at birth	+	++	++
Bed Sharing	+	+++	+++
Switch to circadian rhythm (age in weeks)	8-16	12-20	?
Breast feeding	+	+++	+++
Bacterial colonisation	+	?	+++
IL-10 "low responders"	+	+++	+++

-, +, ++, +++ = rare to common, ? = not known

There are 3 stages at which the risk factors could affect the infectious processes: 1) colonisation of mucosal surfaces by pathogenic bacteria; 2) induction of temperature sensitive bacterial toxins; 3) induction or control of the inflammatory responses to micro-organisms or their toxins. *Staphylococcus aureus* best fits the predictions of the common bacterial hypothesis [2], and staphylococcal toxins have been identified in over half of SIDS infants from 5 different countries [3]. The effects of risk factors for SIDS are assessed in relation to the three stages of infection.

Colonisation: Indigenous infants are colonised earlier and more heavily by potentially pathogenic bacteria [4,5,6]. This could reflect genetic or developmental differences in receptors for bacteria on epithelial cells, exposure to cigarette smoke or closer physical contact with older siblings or family

members. Smokers are more likely to harbour potentially pathogenic bacteria and to be more heavily colonised. Close prolonged contact during bed sharing could increase opportunities to pass bacteria from mother to baby. Cigarette smoke increases vulnerablity to respiratory virus infections and enhances bacterial binding to epithelial cells [7,8,9]. Prone sleeping and viral infection increased the numbers and species of bacteria present in the respiratory tract of infants [10]. Breast milk contains antibodies and glycoconjugates that could reduce colonisation by forming bacterial aggregates which are more efficiently expelled than individual organisms [11,12].

Induction of pyrogenic toxins: *S. aureus* toxins are induced only between 37-40°C, a range usually above the normal temperatures of the upper respiratory tract. Virus infection, the prone position or blockage of a nostril by bedding or mucus secretions could result in higher temperatures at which the toxin can be induced [1,13]

Induction and control of inflammatory responses: Both environmental and genetic factors affect levels of pro- and anti-inflammatory cytokine responses to bacteria and their toxins. Parents of SIDS infants had higher interleukin-1β (IL-1β) responses to staphylococcal toxic shock syndrome toxin (TSST) compared with unrelated control adults. Smokers in both groups had significantly lower responses to both TSST and endotoxin [1]. We examined differences in single nucleotide polymorphisms (SNP) in Europeans, Bangladeshis and Aboriginal Australians for three cytokine genes IL-1β (C-511T); IL-6 (G-174C); and the anti-inflammatory cytokine IL-10 (G-1082A). There were significant differences in distribution of allele frequencies for these SNPs between Europeans and Bangladeshis and between Europeans and Aboriginal Australians; however, there were no differences between the distributions of these allele frequencies between Aboriginal Australians and the Bangladeshi group. Smoking consistently reduced the production of IL-10, but the most significant effect ($p < 0.01$) was observed for the (AA) genotype. Cotinine levels in infants who live with smokers can be equivalent to those of active smoker [14]. Down regulation of IL-10 by cigarette smoke would reduce the infant's ability to control pro-inflammatory responses induced by infectious agents or their products. Compared with European populations, the AA genotype is significantly higher (>80%) for both Aboriginal Australians and Bangladeshis ($p = 0.00$) [1,15,16]. If genetic makeup were a major factor for susceptibility to SIDS, the incidence of these deaths should be similar for these groups; however, they are significantly different and most likely reflect differences in maternal smoking (Table). This hypothesis is supported by differences in the incidence of SIDS for groups of Indigenous Americans that reflect high and low levels of maternal smoking [17].

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July 5, Afternoon session

The Definition and Diagnostic Criteria of SIDS

The fist definition of SIDS is now 35 years old, as are the first diagnostic criteria. In this session, summaries of the most recent symposia on these topics — Soria Moria (Norway) in November 2003, and San Diego (United States) in January 2004 — will be presented together with a new definition and revised diagnostic criteria.

Sudden infant death syndrome (SIDS) and unclassified sudden infant deaths (USID): a definitional and diagnostic approach

Henry Krous, United States

INTRODUCTION

Beckwith's proposed the original definition of SIDS in Seattle 1969 as "the sudden death of any infant or young child which is unexpected by history, and in which a thorough post-mortem examination fails to demonstrate an adequate cause of death."[1] Twenty years later, it was redefined by an expert panel convened by the NICHD as "the sudden death of an infant under one year of age, which remains unexplained after a thorough case investigation, including performance of a complete autopsy, examination of the death scene, and review of the clinical history."[2] The NICHD definition of SIDS differs from the original definition by inclusion of the death scene investigation and limitation of the diagnosis to infants younger than one year of age.

Given the steady accumulation of new information about its epidemiology, risk factors, circumstances of death, pathology, and the importance of ancillary testing, refinement of the SIDS definition is overdue. Therefore an expert panel of pediatric and forensic pathologists and pediatricians deliberated these issues met in San Diego, CA in January 2004 to undertake this task. Drs. J. Bruce Beckwith (USA), Roger W. Byard (Australia), Torleiv O. Rognum (Norway), Thomas Bajanowski (Germany), Tracey Corey (USA), Ernest Cutz (Canada), Randy Hanzlick (USA), Thomas G. Keens (USA), Edwin A. Mitchell (New Zealand), and Henry F. Krous (USA, chair) comprised the panel.

After review of the original and NICHD definitions, a variety of topics were presented including the need for redefinition with its uneasy position between the health and legal systems, possible placement of risk factors in a new definition, the importance of clear definitions of study populations for research, the frequency of risk factors and the changes that had occurred since the "Back to Sleep" campaigns, the advantages and disadvantages of reporting on the death certificate those risk factors that may have been operative at the incident site in causing or contributing to death and the use of the terms "undetermined" and "unascertained" in flagging cases where significant parts of the investigation were lacking, or where there were doubts as to possible causes of death.

Following these presentations, vigorous discussions ensued and a definitional schema for SIDS and other cases of sudden infant death was agreed upon. It included a general definition for purposes of death certification and vital statistics, but recognizing its inherent limitations for research, subsets were also established. The new general definition of SIDS is intended to be more inclusive. The use of subsets of cases of SIDS other sudden infant deaths is intended to facilitate tracking of changes of epidemiological patterns, especially with respect to monitoring the effects of public health recommendations and alterations to infant care practices, at national and international levels. Finally, more precise definition of subsets of sudden infant death, with specification of requirements for diagnosis, should assist in the standardization of protocols to improve the evaluation of the circumstances of death and autopsy examinations and thereby bring investigations more in line with recommended guidelines.[3-5]

PROPOSED NEW DEFINITIONS

The panel has proposed the following definitions and criteria for subsets of SIDS and other cases of sudden infant death:

SIDS - General Definition

The sudden and unexpected death of an infant under 1 year of age, with onset of the lethal episode apparently occurring during sleep, that remains unexplained after a thorough investigation including performance of a complete autopsy, and review of the circumstances of death and the clinical history.

Category IA SIDS - Classical Features of SIDS Present and Completely Documented

An infant death that meets the requirements of the general definition and also all of the following:

Clinical:

- Older than 21 days and under 9 months;
- A normal clinical history, including full term pregnancy (≥37 weeks gestational age);
- Normal growth and development;
- No similar deaths in siblings, close genetic relatives (uncles, aunts and 1st degree cousins), or other infants in the custody of the same caregiver;

Circumstances of death:

- Investigation of the various scenes where incidents leading to death may have occurred, and determination that they do not provide an explanation for the cause of death;
- Found in a safe sleeping environment with no evidence of accidental death;

Autopsy:

- Absence of potentially lethal pathological findings. Minor respiratory system inflammatory infiltrates are acceptable. Intra-thoracic petechial hemorrhages are a supportive but not an obligatory or diagnostic finding.
- No evidence of unexplained trauma, abuse, neglect or unintentional injury;
- No evidence of substantial thymic stress effect (thymic weight less than 15 gms, and/or moderate to severe cortical lymphocyte depletion). Occasional "starry sky" macrophages or minor cortical depletion are acceptable.
- Toxicology, microbiology, radiology studies, vitreous chemistry and metabolic screening studies are negative.
-

Category IB SIDS – Classical Features of SIDS Present, But Incompletely Documented

- An infant death that meets the requirements of the general definition and also meets all of the above criteria for Category IA except that:
- Investigation of the various scenes where incidents leading to death may have occurred was not undertaken, and/or;
- One or more of the following analyses was not performed: toxicology, microbiology, radiology, vitreous chemistry and metabolic screening.

Category II SIDS
An infant death that meets Category I criteria except for one or more of the following:
Clinical:

- Age range - outside Category IA or IB, that is 0 to 21 days and 270 days (9 months) through first birthday;
- Similar deaths of siblings, close relatives, or other infants in custody of same care giver that are not considered suspicious for infanticide or for recognized genetic disorders;
- Neonatal and perinatal conditions (for example those resulting from preterm birth) that have resolved by the time of death;

Circumstances of death:

- Mechanical asphyxia or suffocation caused by overlaying not determined with certainty

Autopsy:

- Abnormal growth and development not thought to have contributed to death;
- More marked inflammatory changes or abnormalities not sufficient to be unequivocal causes of death.

USID (Unclassified Sudden Infant Death)
Deaths not meeting the criteria for Category 1 or II SIDS, but where alternative diagnoses of natural or unnatural conditions are equivocal. This includes cases where autopsies have not been performed.

Post-Resuscitation Cases
Infants found in extremis who are resuscitated and later die ("temporarily interrupted SIDS") may be included in the above categories depending on the fulfillment of relevant criteria.

COMMENT
These proposals are intended to incorporate accumulated knowledge to tighten definitions, and to assist in the more accurate investigation, diagnosis, and categorization of cases of SIDS and other cases of sudden unexpected infant death. The proposed framework is recognized as a "work in progress" that will have to be continually reformulated and filled in as more knowledge becomes available and our understanding of these complex and challenging cases increases.
The complete manuscript prepared after the San Diego meeting will appear in the July 2004 issue of *Pediatrics*.

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International consensus on exclusion criteria for the SIDS diagnosis – Is it possible?

Tortelv O. Rognum, Norway

SIDS definitions are definitions per exclusion. However, since there are different opinions with regard to which lesions that may be fatal, it has been extremely difficult to reach consensus with regard to diagnostic exclusion criteria. In the Nordic countries agreement was reached in 1992. A blind reproducibility testing, involving four countries and 10 forensic pathologists, has shown that the diagnostic practice concerning SIDS has become unified.

On a European level a similar approach has not been successful. The failure may also be evident by reading the literature. Different researchers investigating sudden unexpected death in infancy (SUDI), end up with totally different proportions of unexplained or SIDS cases (75 % - 2.5% of all SUDI cases). This great discrepancy makes it extremely difficult to compare SIDS rates and research results.

The discrepant views also have disastrous effects when it comes to the question of infanticide. For some years leading experts tended to say that: one sudden infant death in a family is a tragedy, two is suspicious, three is murder.

The pendulum has now moved to the other extreme, many experts stating that in most cases of repeated sudden infant death in a family there may be some genetic "risk factor" explaining death. Such genetic risk factors, are mitochondrial DNA mutations, IL-10 gene polymorphisms and long QT-syndrome. Thus it seems to be a great need for intense research on these issues.

In the frame of ISPID we want to stimulate the work for better investigations in all SUDI cases. A thorough death scene investigation is necessary in all cases and there must be an internationally accepted protocol for all investigations that should be included in the autopsy and how the results should be interpreted. This work started at the meeting in Oslo in November 2003. Agreement was reached with regard to brain-, heart- and lung pathology. The work will continue with regard to evaluation of findings on the death scene, evaluation of radiological findings as well as for interpretation of metabolic and genetic findings. The work will continue in Oslo in October 2004.

Concerning the overall definition, a new initiative was taken by Henry F Krous in San Diego in January 2004. The results of this meeting will be published this summer.

Future perspectives

The experience from a three year project investigation all SUDI cases in South-East Norway has shown that thorough death scene investigations by experts may disclose a few cases of neglect and abuse. An extended autopsy protocol including full microbiologic work up, radiology, toxicology, metabolic investigations as well as a case conference with experts from different fields, may exclude a few more cases from the pure SIDS group. However, the SIDS cases still make out approximately 50% of all SUDI cases. We feel that in the future the pure SIDS group will become smaller. Some syndromes and diseases that may be fatal will be sorted out: MCAD and other fatty oxidation disorders, lung QT syndrome and probably syndromes that are not yet described. However, the remaining 50-60 % of the SIDS cases may have one common denominator which is repeated episodes of hypoxia. These hypoxic events may be due to defect in gasping (autoresuscitation). The seretonergic network may be an important factor in this defect autoresuscitation, and the immune system with interleukins may trigger a fatal event in the course of an overreaction to a normal slight infection. Several research groups are now working on this hypothesis applying the three hit model for the understanding of SIDS:

1) Vulnerable developmental stage;
2) Genetic pre-disposition;
3) Trigger event i.e. slight infection, prone sleeping position.

The Avon multi-agency approach to the investigation of sudden unexpected deaths in infancy and the care of bereaved families.

Peter Fleming, Peter Sidebotham, Tracy Hayler, Peter Blair, United Kingdom

The approach that has been adopted in Avon is based upon the practices developed in several countries, recommended in the report of the CESDI study, and endorsed by the Foundation for the Study of Infant Deaths in the UK. (1-4).

As soon as possible after every sudden unexpected infant death a strategy discussion is held, involving the paediatrician, the police child protection team and the social services duty team. The purpose of the discussion is to plan how best to investigate the death and to support the family. This usually involves initial contact with the family in the Accident and Emergency department by the paediatrician and police officer, followed by a joint home visit, usually with the family doctor or health visitor. At these visits a full medical and social history is taken, with particular emphasis on recent events and a careful review of the circumstances and scene of the death. This information is passed to the pathologist, to ensure that appropriate and relevant post-mortem investigations are carried out.

Historically, the scene of the death of a baby was approached by police officers as a "scene of crime" and the same rules with regard to preservation of evidence were applied as at any suspected homicide. In developing the Avon Protocol, police Senior Investigating Officers were reassured that there was no risk of compromise of evidence for any potential criminal enquiry. Effective communication between all relevant professionals has allayed initial concerns. The attendance of a paediatrician together with a suitably qualified police officer at the death scene enhances the information gathering process for both agencies.

By visiting the home and seeing where the baby died, both the police and the paediatrician can gain further information, whilst the family are given the opportunity to talk through what happened in detail. Both police and paediatrician, in conjunction with the primary health care team, provide further support to the family. Families have expressed great appreciation of this co-ordinated approach, recognising the need for police involvement, but feeling that the joint visits have been helpful rather than intrusive. Information on the post-mortem is fed back to the family as it becomes available.

Finally, 2-3 months after the death, a case discussion meeting is held, involving all professionals who were involved with the family. This gives an opportunity to review the classification of the death, identify any contributory or associated factors, debrief those involved in the care of the family, and to plan for continuing support and care of the family, including informing them of the assessment of the cause of the infant's death. At this meeting the "cause" of death is classified according to the Avon clinicopathological classification scheme (2). This grid system of classification ensures the collection and recording of information that is of importance and potential value to the family, and to each of the agencies involved in the care of the family and investigation of the death, and forms the basis for planning the future support and care of the family.

A joint approach, with medical staff working closely with the Police Child Protection Team, ensures that all necessary information is collected sensitively and promptly, without the need for bereaved families to repeat their stories to multiple agencies. The paediatricians and the Police Child Protection team have broad experience of normal childcare practices in this community, and are thus perhaps less likely to draw unwarranted conclusions about the contributions of particular patterns of child care to the death. The continuing involvement of the paediatricians in research into current child care practices within the community further helps to inform their interpretation of information obtained after infant deaths.

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Australian protocol and definition meeting

Roger W. Byard, Australia

In an attempt to further improve standards, and to obtain a consensus among forensic and paediatric pathologists in Australia for a common definition of SIDS and recommended standardised autopsy guidelines for infant and early childhood deaths, a national workshop was convened in March 2004. The workshop was specifically oriented towards assisting pathologists who routinely perform autopsies on unexpectedly dead infants to help standardise their approaches and diagnoses. Representatives from forensic and paediatric pathology institutions from all Australian states and territories attended the two day workshop.

Presentations on the first day included an overview of problems in the investigation of infant deaths in Australia followed by a summary of the results of recent international workshops dealing with the diagnostic evaluation of sudden infant death (Oslo, Norway, November 2003) and issues of definition (San Diego, United States, January 2004). The latter presentation included the reasons for Beckwith's call for a re-evaluation of the definition of SIDS and the definition proposed from the San Diego meeting. Delegates then detailed the range of investigative approaches that are found in different jurisdictions around Australia. Concerns were expressed regarding recent trends in certain states not to have autopsies performed on all cases of unexpected infant deaths. On the second day details of the process of implementation of the international standardised autopsy protocol for sudden unexpected infant death in California were presented.

After some discussion and evaluation of the range of SIDS definitions available in the literature, delegates at the workshop endorsed the newly proposed San Diego definition of SIDS, and recommended uniform use and acceptance of the definition in centres around Australia. The new definition was thought to provide a good general definition of SIDS with recommendations for appropriate investigations, in addition to specifying characteristics such as an occurrence under one year of age and an association with sleep. There was also general, although not complete, support for the term USID, or unclassified sudden infant death, which had been proposed at the San Diego conference in an attempt to minimise the considerable confusion that surrounds the use of terms such as 'unclassified', 'unclassifiable', 'undetermined', 'undeterminable' and 'SUDI' (sudden death in infancy).

Review of both the Australasian SIDS Autopsy Protocol and the International Standardised Autopsy Protocol for sudden unexpected infant death was then undertaken. Given that both protocols had been formulated some time ago and are in need of re-evaluating, it was decided to update the Australasian protocol based on recent developments and current requirements for the appropriate investigation of these cases, using it as a template, while incorporating any features of the International Standardised Autopsy Protocol that were considered pertinent.

Although the specifications were regarded as a gold standard for the approach to unexpected infant and early childhood death, it was recognised that not all investigations could be undertaken in every jurisdiction or facility. For this reason the proposals were seen as guidelines that were freely available for modification to suit local circumstances. Any modifications would, however, need to be defensible to colleagues and the courts as being reasonable on

scientific, technical or logistical grounds. An example of the latter would be a decision not to undertake microbiological examination in a case from an isolated community in the far north where the body may have taken many days with suboptimal storage before it was available for autopsy. While the upper age limit for SIDS was taken as one year it was recognized that this is an artificial and somewhat arbitrary cut off point. For this reason the autopsy guidelines were considered applicable not only for infants, but also for toddlers and for children up to the age of five years.

The Canberra workshop has clearly demonstrated the effectiveness of collaborative work between forensic and paediatric pathologists and has provided Australian pathologists with a national definition of SIDS based on international recommendations. It has also provided guidelines for the pathological evaluation of cases of unexpected infant deaths that hopefully will further reduce the very significant medicolegal, ethical and societal problems that result from either incompletely or inadequately investigated cases, or from cases where there have been misinterpretations of findings.

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Future Research Directions

In the past decade, SIDS research findings have brought us a long way. Indeed, the "reduce the risk" campaigns can be seen as one of the great success stories of Pediatrics. But SIDS is still with us and the fight is still on. We have asked specialists from various fields to give us their perspectives on the challenges ahead.

Perspectives from the pathologist

Roger W. Byard, Australia

It is well recognised that cases of infant deaths caused by injuries or illness have been attributed incorrectly to SIDS in the past, and unfortunately it is likely that this problem continues at present. One way to ensure that opportunities for inaccurate diagnoses and misclassification are minimised is to follow protocols that provide frameworks for dealing with death scene and autopsy examinations in cases of unexpected infant deaths. Use of the Centers for Disease Control and Prevention (CDC) Guidelines for Death Scene Examination and the International Standardized Autopsy Protocol for sudden, unexpected infant death is, therefore, strongly encouraged. These protocols have been endorsed by the National Association of Medical Examiners (NAME) and the Society for Pediatric Pathology (SPP) in the United States, and provide some standardisation in approach. Recent meetings of SIDS experts have been held in Oslo, Norway and San Diego, California, United States to review the definition of SIDS and diagnostic criteria for other causes of unexpected infant death. However, although protocols provide guidelines for infant death investigation, certain difficulties persist.

One of the problems lies with the definition. Over the last decade at least four definitions have been proposed that may or may not include an upper age limit of one year, death scene examination, an association with sleep, autopsy examination, and review of the clinical history. The most widely accepted definition has been the one issued by the National Institute of Child Health and Human Development which defines SIDS as 'the sudden death of an infant under one year of age which remains unexplained after a thorough case investigation, including performance of a complete autopsy, examination of the death scene, and review of the clinical history'. Recently Beckwith called for a review of this definition with the introduction of stratification based on the level of diagnostic certainty, and the presence or absence of typical features. This has been undertaken by an expert panel who met in San Diego, United States in January 2004 and the results will be published soon in the journal *Pediatrics*.

The next issue concerns the use of the term 'SIDS' without fulfilling the criteria listed in standard definitions. Cases have been, and continue to be, classified as SIDS where the scene where the infant was found has not been investigated, and even where complete autopsy examinations by qualified pathologists have not been undertaken. Telephone interviews with caretakers should not be considered a death scene investigation. In Australia, infant deaths have been signed out as SIDS where autopsies were either not performed, or were incomplete and in other parts of the world autopsy rates for supposed SIDS infants have been as low as 0 to 40%. This occurs despite widespread acknowledgement that there are many diagnoses that can only be made with knowledge and findings obtained from death scene and post-mortem examinations. Examples include intracerebral haemorrhage, myocarditis and certain forms of congenital heart or inherited metabolic diseases. With improved investigation of infant deaths there has been an increase in the numbers of deaths being identified due to such causes as unintentional asphyxia from unsafe sleeping environments. In fact, the proportion of unexpected

infant deaths due to causes other than SIDS has reached 25% in some communities. Thus, without proper investigation as many as one in four infant deaths could be misdiagnosed as SIDS.

Isolated rural and indigenous populations are particularly difficult to study as it is likely that infant mortality is high due to acquired natural diseases such as gastroenteritis and pneumonia. However, such deaths may be incorrectly attributed to SIDS due to failure of adequate investigations. Interpretation of the significance of research results from such areas is problematic. Large national studies may also not have had mandatory death scene examinations in cases that have been classified as SIDS. Researchers involved in such studies may have particular difficulties in ensuring that their study population is appropriate, as they usually do not have control over the categorisation of cases, and often do not have a clear understanding of the need for scrupulous consideration of other possible causes of death or the thoroughness with which cases were worked up diagnostically. Too often a death certificate diagnosis of sudden infant death syndrome is all that is required for inclusion into studies. If a case does not fit diagnostic criteria in other areas of medicine it is generally not included in studies with cases that are diagnostically typical; why is SIDS different? Surely epidemiological and legal conclusions based on such research must be questioned.

It is possible that some of the contradictory research findings in the SIDS literature have been due to variable investigations and different diagnostic approaches to cases rather than to heterogeneous underlying causal mechanisms. It is time, therefore, for a re-evaluation of the way in which the term SIDS is being used.

One approach to the evaluation and formulation of SIDS research data would be to rank studies based on the completeness with which initial investigations were undertaken. Studies should clearly state the definition of SIDS that was used, the personnel involved in death scene and post mortem examinations, the extent of the post mortem examination and the number and type of ancillary investigations that were performed. The number of cases that failed to meet standard definitions should also be indicated. Stratification of SIDS case along the lines proposed by Beckwith and recommended at the recent San Diego meeting would provide a framework for this type of data presentation. Published research could then be evaluated on the completeness with which cases were investigated. Data based on cases with typical characteristics of SIDS, where the clinical histories were reviewed with full post mortem and death scene examinations by experienced individuals following accepted protocols would receive the highest grade. The significance of possible conclusions would be less reliable if cases did not have these steps performed, or were taken from archival material. Conclusions based on cases where death scene and post mortem examinations had not been conducted would receive the lowest grade, signifying that there were significant concerns regarding the credibility of the study results. In addition, if research relies heavily on material that was published before current definitions were formulated, this also needs to be acknowledged. Unfortunately, unless greater precision is used in determining that a death has been due to SIDS, research will be compromised and the term SIDS will fail to contribute in a meaningful and substantive manner to the study and understanding of unexpected infant death.

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Perspectives from the physiologists

Does the use of animal models assist our understanding of SIDS pathology?

Karen Waters, Australia

Studies in animals have been used to examine whether various clinical risk factors can induce death or other abnormalities that are typically associated with SIDS. Several groups have now used parallel studies in humans and in animals to provided broader insight into the mechanisms of disease than would be available from clinical studies alone. A balanced understanding of animal studies requires recognition of both the advantages and the limitations of such research. In this context, "Pitfalls" refers to "difficulties, or limitations" rather than "errors or mistakes".

Specific examples of the use of animal models by several research groups will be discussed, to illustrate that many of the conditions used to test animals are based on clinical conditions The advantages of animal models, is the use of controlled conditions, so that a number of animals can be exposed to the same conditions for determining their sequelae. Another benefit is the ability to undertake post mortem assessments of various organ systems, including the brain, after known environmental exposures.

Models have shown variable similarities to the human condition. For example, Cote's studies of piglets have shown that there is an age of vulnerability to hypoxic insults (Cote & Porras, 1998; Cote et al., 1996). Another approach has been to examine upper-airway function, and responses during different sleep stages as well as after commonly used drugs such as phenothiazine. Here, changes in human infants were shown to be equivalent with young piglets, and abnormalities then induced by the use of common clinical drugs were evaluated. Once prone sleep positioning had been identified as a risk for SIDS, studies of infants, and animal models in this position were used to identify the occurrence, severity, and sequelae of respiratory abnormalities that could be induced by this risk factor (Chiodini & Thach, 1993; Jacobi & Thach, 1989; Kemp et al., 1993; Sridhar et al., 2003)

Similarities in brain abnormalities have been studied by several groups. Having identified brain abnormalities in human infants who died from SIDS, recent studies by the group with Filiano and Nattie, have examined how lesions in the ventrolateral medulla of animals can produce respiratory control abnormalities that may underlie death in infants (Curran et al., 2000; Curran et al., 2001; Filiano & Kinney, 1995; Kinney et al., 1995). The effects of pre-natal hypoxic insults were compared in animal models, against brain abnormalities seen in SIDS infants (Mallard et al., 1999; Tolcos et al., 2000). Our own studies began with the identification of cell death markers in infants who had died from SIDS, and then explored whether noxious respiratory insults, such as those seen with prone sleeping, could induce equivalent abnormalities (Machaalani & Waters, 2003; Machaalani & Waters, 2003; Waters et al., 1999).

Another line of research, for which animals provide unique advantages, is the assessment of latent or delayed abnormalities after exposure to conditions from which the animals apparently show complete recovery (Gozal & Gozal, 2001). Subsequent studies (in life or after death) have then revealed ongoing abnormalities.

The most obvious, and main disadvantage of using animals to study SIDS, is that there are many differences in the characteristics and the development of animals compared to human infants. Findings such as the risks associated with prone sleeping, cannot be determined from animal studies. As more animal studies are undertaken, it becomes difficult to evaluate the true risks associated with the latent dangers implied by some of these study results.

With all aspects of research, each research method has a number of benefits to offer. However, proper understanding of the results of these studies also requires that the audience are aware of the limitations that the particular methods impose.

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Perspectives from the physiologists

André Kahn, Patricia Franco, José Groswasser, Belgium

From the standpoint of physiology, SIDS research still opens to a variety of fields of human physiology still poorly known. SIDS research includes new aspects such as the link between infant's physiology and new emerging risk factors and potential protective factors. More information is being collected on the characteristics of normal and deviant pre- and perinatal development. SIDS research could includes progresses in more classical study areas. Additional information is being collected on the physiology of vital control mechanism. Control mechanisms include such issues as the cardiocirculatory system; the respiratory system; the immune system; the autonomic controls; or the autoresuscitative control mechanisms. These control systems refer to the sleep and wake continuum, as well as other cardiorespiratory and autonomic responses to internal and external challenges. The infant responses and adaptation to both short-term and chronic stress is being evaluated. The contribution of the infant's emotional and sleep-wake status is progressively included in the description of normal physiology. Environmental factors in the child micro-environment that modify these mechanism are likewise taken into consideration in the explorations of infant physiology. The infant gestational age is an additional factor that is taken into consideration when exploring the physiology of very premature infants. The genetic basis of these aspects of developmental physiology and control mechanisms, are being collected.

The exploration of such complementary fields of human physiology requires adequate tools, and new ways to assess and measure subtle changes in the various research fields. For some, such as the waking system, definitions are being improved. Other research fields, such as the evaluation of the autonomic controls, lead to the definition of new and more precise non-invasive exploration tools.

These studies develop on the most efficient manner with the help of electronic communication tools, and common data-base accessible to all researchers world-wide. International research protocols likewise contribute to collect sufficient information. In addition to scientific journals and web sites, the new data are best debated and exchanged in scientific meetings such as the present one in Edmonton.

Perspectives from epidemiology

Peter Fleming and Peter Blair, United Kingdom

The rapid decline in the SIDS rate has been brought about by investigating the place in which the infant last slept and it is perhaps further detailed scrutiny of this environment that will bring about a subsequent fall in deaths. Although there is a consensus of opinion that infant sleeping position and exposure to tobacco smoke are part of some causal chain there is little understanding of how this causality works whilst for other factors such as bed-sharing and pacifier use there is not much consensus at all. The traditional approach to epidemiological investigation of using randomised controls and building a multivariate model which mixes up infants sleeping in different environments and factors that have different effects on these environments seems to confuse and limit the interpretation of our results. Future SIDS studies are likely to include smaller numbers of infant deaths, and to include a larger proportion of deprived families and infants sharing the parental bed. If we are going to meet these changes and overcome our polarised debates we need to re-design future SIDS investigations.

The South West Infant Sleep Scene (SWISS) study is an epidemiological investigation of SIDS deaths including a death scene investigation of each case and a similarly detailed sleep scene investigation of the control infants. The study area represents approximately 10% of the English population with 60,000 livebirths per year and an anticipated 30 SUDI deaths. The study began in January 2003, initially for three years but hoping to extend for a further two years.

When a death is notified a multi-disciplinary team including a paediatrician, a trained representative of the police and a SWISS researcher contact the parents and an interview takes place within hours of the death. This interview takes place in the family home and includes a detailed, structured death scene investigation, together with bereavement support and information for the family.. The death-scene investigation includes a standardised video recording of where the infant died, a plan of the room including measurement and north-south orientation, temperature readings and measurement of mattress softness. A follow-up interview is then arranged to formally go through a detailed study questionnaire. A life-sized doll was especially built to be used in this interview but problems in terms of how the bereaved parents may react to such a doll and the practical aspect of how to carry such an item has meant abandoning such an idea, a doll-like toy in the room is sometimes used to demonstrate the position and covering of the infant.

Approximately 300 randomised control mothers are being contacted via their midwife during pregnancy and details of infant health and sleeping environment collected by postal questionnaire at 2 weeks, 2 months, 4 months and 8 months old. Contact prior to the birth of the infant means we can try and determine the maternal preference of infant sleeping environment. Of these, 100 are being chosen, weighted to include a high proportion of bed-sharers, to take part in a sleep-scene investigation carried out in exactly the same way as the cases. The mother is contacted shortly before interview and asked not to make any preparation or change any routines for the visit. The mother is interviewed about infant sleep practices in the previous 24 hours to interview, a sleep-scene investigation is conducted and the same questionnaire as used for the index mothers is administered by the researcher..

A further control group has been enlisted for this study consisting of families at " high risk" for SIDS. This was formulated from previous study data (CESDI study), constructing a multivariate model of pre-natal factors obtainable easily from hospital records and significantly associated with SIDS families. From our data this included 4 factors; young maternal age, high parity, maternal smoking and social deprivation. Using these 4 factors 300 "high risk" control families are being included of which 100 will be selected for a sleep-scene investigation. It was anticipated that the ascertainment rate for this group would be extremely low and the first 8 months of data collection has borne this out. Of 192 families contacted via their midwife,19 were excluded because of very early pregnancies and only 31 (18%) consented to take part of which 10 have since dropped out. If this were the randomised control group there would be an insurmountable problem in terms of selection bias but because the very group we are trying to ascertain are those who usually fail to take part in any study this is not so much a problem. One of the solutions which appears to be working is to entice the families with small monetary vouchers for baby shops.

In the first year of the study there were 31 SUDI deaths of which 15 were SIDS, 12 explained SUDI and 4 still to be determined. Of the 15 SIDS infants, 4 were co-sleeping in the parental bed and 3 on a sofa. Of the 12 explained SUDI, 6 were due to unrecognised infection and 3 due to metabolic disorders.

In previous epidemiological studies pathological findings have often been ignored as too non-specific and anecdotal because of the lack of a comparison group. The large proportion of explained SUDI deaths in this study will be used as a further control group and the pathology findings integrated with the epidemiological information. Nearly 50% of the SIDS were co-sleeping in deprived households but what aspects of deprivation were important or how exactly the co-sleeping environment put the infant at risk may be unclear when just observing the cases but might become clearer when comparing them to similar "high risk" bed-sharing controls.

We anticipate the final number of SIDS infants in the study will be small (N=70), nearly half of whom will be found co-sleeping in mainly deprived households. However, using this new study design, we hope to have control infants matched for age, sleeping environment and similar housing conditions.

Health and Allied Professional – Parent – Indigenous People Program

July 3, morning session

A Key to Improved Social Network Support?

Kari Dyregrov, Norway

Recent research has shown that social networks are experiencing ineptitudes and insecurities when encountering bereaved populations, especially after traumatic losses. What are the ineptitudes?

How do the bereaved react to them? Why do network members feels so insecure?

How can the bereaved and network members communicate better in order to improve the support? In order to answer these and other questions, a double perspective is needed that examines the perspective of both the bereaved and the social network members. This is provided through unique qualitiative and quantitative data from two Norwegian nationwide studies with 232 traumatically bereaved parents (SIDS, suicide, accidents), and 131 social network members (SIDS, suicide). The encounter is analyzed as a relational communicational process, implying that social support must be explored as an interpersonal phenomenon. Thus, this paper explores the difficulties of the encounter as seen from both parties, and points to coping strategies of both groups in overcoming the difficulties. Moreover, it stresses the crossing viewpoints concerning what it takes to make the support more optional and concludes with advice from each group to the other. The paper concludes with some reflections on cultural aspects and the possibility of generalizing the findings across nations.

Aboriginal Leadership Forum on SIDS

Dwight A. Dorey, National Chief – Congress of Aboriginal Peoples
Kukdookaa Terri Brown, President, Native Women's Association of Canada

We have invited Aboriginal leadership to discuss action on addressing risk for our community. This session presents a unique opportunity to mobilize the community and develop consensus on health. Our Old People have often said that it is our leadership who have and are a huge part of the medicine necessary to heal our communities. SIDS is related to many of the factors that place our community at risk such as tobacco misuse, addictions, fetal alcohol spectrum disorder, and lack of pre-natal care and other culturally appropriate health services. Action on SIDS can have multiple positive effects and outcome for our communities. The Forum will include a discussion and questions and will inform the development of the Aboriginal Action Plan in SIDS.

Surviving the First Two Years

Joani Horchler, United States

Newly bereaved SIDS families attending the 2004 SIDS International Conference in Edmonton will be expecting to find a workshop that fills their specific needs as individuals bereaved for some two years or less.

Joani Nelson Horchler, author of the highly respected *SIDS & Infant Death Survival Guide* (of which more than 26,000 copies have been sold or donated) is well qualified to lead this workshop, having served as a moderator of a similar workshop held at the 1996 SIDS International Conference in Washington , DC, and having survived more than 12 years of bereavement herself. Joani is the mother of five daughters and lost her only son, Christian, to SIDS in 1991. Also, Joani has written much on the

subject of coping with recent bereavement, and as executive director of SIDS Educational Services, Inc. and as a trained peer counselor, she often counsels newly bereaved family members.

This workshop would allow each participant member time to talk with the group about his or her personal experience with a death, and it would offer peer support, comfort, and advice on how to cope with loss based on rime-tested experiences of people like Joani who have "been there."

July 3, afternoon sessions

SIDS: Sudden unexpected and unexplained… but what do we now know and how can we continue to learn and help prevent future deaths?

Peter Fleming, United Kingdom

Despite the dramatic falls in the number of infants dying suddenly and unexpectedly that followed the introduction of "Back to Sleep" campaigns, such deaths still comprise the largest single group of identifiable deaths in childhood in many countries. As the risk factors of prone sleeping position, parental smoking, and heavy wrapping have been identified, and in some social groups largely excluded, the pattern of factors contributing to sudden unexpected deaths in infancy has changed. Continued study of the factors that may contribute to unexpected infant deaths will allow the identification of additional factors, and the clarification of the role of the known factors in different environments. Increased recognition of the importance of metabolic conditions – some only very recently identified – and of unusual infections, together with an increased understanding of the way individual responses to infections may differ, has led to a fall in the proportion of sudden unexpected infant deaths that remain unexplained. In the current large scale study of such deaths in the Southwest of England, more than 50% of deaths are now fully explained, compared with 10% in the 1980's, and 20% in the CESDI study in the early 1990's.

In this talk I will present a review of current knowledge of risk factors, the complex issues raised by study of the infant's sleep environment – particularly the nature and importance of parent-infant interactions during sleep – and new developments in the fields of genetics, metabolism, immunology and infection that may help to understand why some infants are at particular risk of sudden death.

Pitapun's Traditional Healing Swing

Darlene Auger, Consultant

Pitapun's Swing is the Native Traditional Therapeutic Swing. To be swung is to be recognized in a spiritual way, to be nurtured in a healing way, and to be loved in a sacred way. The Native Traditional Healing Swing was born out of a powerful vision during a pipe ceremony in the fall of 2001, a vision that spoke about the essence of love that is needed to bring healing to all people. The message of nurturing our people back to health has been heard for many years now, all across the globe. As indigenous peoples we have been hiding our healing ceremonies and our grieving ceremonies to the point where generations of people have had to survive without them. The lack of traditional healing

ceremonies has caused our people double grief. Over time this grief has been accepted as a norm and the people have become numb with pain. It is time to heal. Traditionally as Indigenous peoples all over the world we have swung our babies above our beds or in corners of rooms. This simple but rather profound practice is about the respect we have for spirit. When babies are first born to us, we say they have one foot in Spirit World and one foot on Mother Earth. This is a crucial time for babies to decide whether they will stay to experience human-hood or if they will go back to Spirit World. This rite of passage is not complete until the baby learns to walk on their own two feet. Until then, we as parents or grandparents must nurture them by swinging them. We place them in a swing out of respect for their neutral place of being, not yet grounded on Mother Earth. The simple action of swinging our babies has profound benefits to the body, mind and spirit. Research suggests that the swinging motion balances the circulation of fluids of the left and right hemispheres of the brain. This induces emotional and physical balance, memory and also releases tension. Our ancestors were wise, they knew the healing benefits of the swing. There is a belief the when the body is swung in this side to side motion, that fluids in the brain release a hormone that acts as a natural healing agent to the rest of the body and thereby strengthens the immune system. Our people have suffered and survived many foreign diseases. The power of our ceremonies and our healing techniques should receive the credit they deserve and we need to reconnect with our traditional ways of nurturing our people back to health.

That's Not the Right Way to Grieve! -- Is There Really a RIGHT Way to Grieve?

Karen Martin, Canada

Talk about your grief.

There's no point in talking - it doesn't change anything. Remember and honor every birthday and anniversary. Try to ignore the significance of these days. Honor and celebrate your memories. Try to set them aside and move on. Turn your grief over to God. Turn your grief over to a support group or counselor. What is the right way to handle grief? More importatnly, is there a right way to grieve?

In this session, she will explore the origins and impact of rules that have defined the right way to grieve and how they can affect how bereaved parents feel about themselves, each other, their deceased child, and relationships with their own parents.

Helping Bereaved Parents Cope with Guilt: What You Need to Know

Bob Baugher, United States

One of the most common and enduring parental responses to the death of a child is guilt. While nothing professionals can say or do will eliminate the parents' feelings of guilt, there are ways to help. In this workshop, we will discuss the dimensions of guilt, reasons for guilt, and types of guilt. We will then explore a number of ways you can help parents cope with these feelings.

Indigenous Community Taking Action.
Forever sleeping: The Design and Development of Culturally Appropriate Ressource Material on SIDS

Kay Half

Kay developed a project to address SIDS in the Aboriginal community. In this presentation she will provide a project overview: The design and development culturally appropriate resource
material on SIDS. She will discuss the purpose and outcomes and the objectives.

The process she took involved:

- Incorporating a cultural approach in the development of an educational resource for Alberta region.
- Incorporating Elders assistance on Sudden Infant Death
- Involving parents whom have lost loved ones through SIDS in the overall development of the poster and pamphlet
- Coordinating Alberta Community Health Representatives in the development of a poster and pamphlet

Incorporate the assistance of a native artist in the development of the poster and pamphlet.

Sibling Grief

Janice Roper and Joani Horchler, United States

The workshop will include a brief overview of the age-related reactions to a sibling death, children's phases of grief, and informational/web resources for helping your living child heal. There will be facilitated group discussions, sharing of experiences and advice on how to cope with the emotions and answer questions. We will present age-appropriate arts & craft activities geared toward helping the expression of feelings toward their sibling's death. We will have the audience create some art and share it with the class (why they created it, what they were thinking or trying to say with it) to help them along their grief journey.

Janice Roper, author of the highly acclaimed Dancing on the Moon, a picture book to help children and their parents through grief, would open this workshop by reading the poetry of her book. Since this book typically evokes strong emotions, a reading of it is a good way to help children and their parents (who would also be invited to attend) to begin sharing with the group their feelings and questions about how to cope with their grief. Janice would co-lead this workshop with Joani Horchler, author of SIDS & Infant Death Survival Guide.

Both Janice and Joani would have their own children with them to participate in and help facilitate the workshop. Janice, a mother of three daughters, lost her only son, Daniel C. Roper IV to SIDS in 1995. She and her oldest daughter Selena, now age 9, posed for the artist who painted the 14 beautiful oil paintings that make up Dancing on the Moon. Selena has participated in other support workshops with her mother. Joani, who lost her only son, Christian, to SIDS, published Dancing on the Moon in 2001 through the nonprofit SIDS Educational Services Inc that she established in 1994.

July 4, morning sessions

SIDS and Kids in Australia: Community controlled organizations do make a difference

Rachel Atkinson

This presentation will look at the collaboration between SIDS and Kids and the peak body representing Aboriginal health, NACCHO; it will explain the structure of the Aboriginal community controlled medical services in Australia, and it will specifically look at the model of maternal and child health at the Townsville Aboriginal and Islanders Health Services (TAIHS), which demonstrates why we believe that we are making a difference, that we *can* reduce infant death in Aboriginal communities.

It will highlight the significance of the Memorandum of Understanding between SIDS and Kids and the National Aboriginal Community Controlled Health Organisation, NACCHO. This is a formal agreement between the two community controlled organizations to work in collaborative partnership to reduce perinatal and infant mortality in the Aboriginal and Torres Strait Islander communities; communities where families are six times more likely to suffer the tragedy of a SIDS death as compared to the rest of the Australian population (ABS 2000).

It will explain the community control structure from a national, state and *on the ground* perspective.

It will look at one Aboriginal Medical Service (AMS), the Townsville Aboriginal and Islanders Health Services Limited, (TAIHS); its governance, its programs and, in particular, the maternal and child health, *Mums and Babies,* project.

This is a collaborative program that has been run out of TAIHS since January 2000. Some 480 pregnant Aboriginal and Torres Strait Islander women have presented to the clinic for antenatal or pregnancy care (ANC) in the four years to December 2003. The number of pregnant women using the service has increased from 60 in 2000 to 150 in 2003; 60% Indigenous local women are now coming to TAIHS for ANC. The number of ANC visits has increased to 8 per pregnancy. These women are coming in earlier for a first visit. Women are having more ultrasounds and infection screening. Antenatal education has increased. The model is consistent with the WHO model of antenatal care. There has been a significant reduction in preterm births and increase in birthweight amongst Townsville-based women compared to historical controls. The proportion of preterm births amongst the Townsville-based women (7.3%) has fallen below that of non-Indigenous women in Queensland in 2000 (8.3%). However perinatal death rates remain high. This program has had a significant impact on perinatal outcome and empowered the patients, staff and service. We hope that these results will persist and improve further in the years to come. Our bottom line is to save more babies lives… and we believe that our model is the way to do this.

Talking Circle: Impact of SIDS on a Family

Brenda Jenkins, Theresa Whiskeyjack, Tess Jenkins, Albert Morin, Vivian Jenkins, Shauna Lapatak

This session will be conducted in a traditional Talking Circle format. The Jenkins/ Morin Family will relate the impact SIDS has had in their family and they will invite those present in the circle to speak of their experiences. The Traditional talking circle uses a talking stick or feather to allow each person to express, uninterrupted, all that they have to say on a given topic. Our Old People say that our ability to speak openly and honestly with one another is part of what it means to be a healthy community. This session will facilitate open and honest words to promote health and healing for families affected by SIDS.

Resilience

Sally Miller, United States

Discussion of grief, loss and dealing with change, coping strategies for bereaved parents after life changing events and how to find the strength to go on.

Teachings of the Moon: Women Elders' Circle on Parenting and Infant Health

Christine Daniels, Jenny Cardinal

In Plains First Nations culture, the women of the community were taught about womanhood and motherhood by the women Elders of the community in a place called the "Moon Lodge". The Moon Lodge was entered when a young woman had her first menstral cycle or "Moon Time". There she learned about sexual health, childbearing, relationships and many of the skills to ensure the survival of her children and her community. In the Moon Lodge, girls would gain the support of the entire community as they became woman, and as they became mothers. The Moon Lodge allowed women to gain the wisdom and strength of every generation that came before them. This traditional knowledge allowed women to give birth by themselves, and later catch up with her tribe. This session will allow participants to hear Our Women Elders speak on parenting and Infant health from a traditional perspective. It will be conducted in a circle format and facilitated by the Elders.

Tobacco… The Traditional Path

Jo-Ann Daniels, Canada, Aboriginal Community Support Co-ordinator
Roland Cotton – Elder, Blackfoot
Jerry Saddleback – Elder, Cree
Ernie Lennie – Dene

Most discussion on Tobacco and health focuses on the many heath problems associated with tobacco use, including SIDS. However, for many Aboriginal people, Tobacco is a very sacred medicine. Some believe that tobacco is a very powerful medicine that takes our prayers up to the Creator, the source of all health, and ensures they are heard. This session will focus on the traditional use of Tobacco and how it is used for health and healing. The perspectives and teachings of different Indigenous Peoples will be presented including, those of the Dene, Blackfoot, and Cree peoples.

Tobacco and Smoking: Treating Tobacco Use among Pregnant and Parenting Smokers

Cathy L. Melvin, United States

Despite evidence of increased risk to themselves, their pregnancies and their families, a significant proportion of pregnant women continue to smoke throughout pregnancy. Similarly, an even larger proportion of parents and caregivers continue to smoke around infants and children. Clinicians providing prenatal care for these women and pediatric care for their newborns and infants are in a unique position to offer clinically proven, effective interventions. The gap between what is known about how to treat pregnant and parenting smokers and what is actually done in clinical practice is large with fewer than half of all obstetricians surveyed in the US offering recommended treatment.

A growing volume of research since 1975 has demonstrated that clinically proven, effective interventions exist to produce long-term or even permanent abstinence from tobacco for all smokers. Achieving cessation is important for all smokers but especially for pregnant and parenting smokers since their smoking poses risks not only for themselves but also for their pregnancies and children. Treatments for smokers in general apply to parenting smokers but special considerations regarding treatment need to be made for pregnant women. Due to the harms associated with exposure to environmental tobacco smoke (ETS), or secondhand smoke (SHS), both parents and caregivers of young children should receive treatment to achieve cessation or counseling on how to eliminate exposure of children to ETS/SHS.

This presentation will provide a review of the recommendations made in 2000 by the United States Public Health Service regarding treatment for pregnant and parenting smokers, summarize recent findings that may impact treatment protocols and make recommendations regarding further research in treatment approaches for pregnant and parenting smokers. A summary of recommended changes in treatment approaches for clinicians based on this review and a description of factors affecting clinician adoption and use of proven treatments and systems supports found to increase the likelihood of clinician use of these treatments will be discussed.

Efforts underway in the United States to implement these recommendations will be presented with a special focus on the work of the National Partnership to Help Pregnant Smokers Quit, the only national organization in the US working to mobilize health care systems and local communities to help pregnant smokers get the help they want and the support they need to quit smoking and remain tobacco-free. More than 60 member organizations of the National Partnership have joined forces to put evidence-based approaches to reducing tobacco initiation and use into practice at the national, state and local levels. The National Partnership's Action

Plan, working group structure and evaluation process will be described including efforts to improve the healthcare system, use the media, promote community and worksite policies and programs, pursue federal and state policy initiatives and promote research, evaluation and surveillance.

Walking with the Newly Bereaved Parent
What Do I Do? What Do I Say?

Tina Langille, Canada

The death of an infant is one of life's most tragic events. The circumstances surrounding an infant death contribute to the uniqueness of each tragedy. There is no template, no road map or one right path for the newly bereaved family to follow. The first few days, hours and even minutes begin their journey of grief; it is important that they be helped by those who can guide them along this difficult and painful journey. Herein rests our challenge. As Health Professionals, First Responders, Coroners, Funeral Directors, Clergy, Bereavement Facilitators and many others, we are given the task of trying to help these families during what will likely be one of the most chaotic and painful times of their lives. Whether you are working in a hospital environment or in the community, this presentation is geared to nurture the personal and professional experience you may already have and empower you with information and new tools to help you help these parents.

Developing Partnerships With Tribes to Reduce SIDS Among Native Populations

Heather Shotton, Co-Founder Mikela's Miracles SIDS Connection

Sudden Infant Death Syndrome is a serious concern for the Native populations in both the United States and Canada. In the United States, the Native population experiences a disproportionately high rate of SIDS. According the Indian Health Service, the SIDS rate is 2 to 4 times greater for Native infants, depending on location in "Indian Country", than for the U.S. population. And, while the United States has witnessed a dramatic decrease in the rate of SIDS over the past decade, the rate of SIDS among Native people remains disproportionately high.

An important step in reducing the rate of SIDS among Native people is developing partnerships with Native tribes, tribal organizations, and Native organizations. It is important to understand the unique culture of Native people and the unique differences that exist between tribal groups. To effectively reach Native communities organizations must develop relationships with Native tribes so that they may best address their needs.

Mikela's Miracles SIDS Connection, a Native owned and operated SIDS organization, has been working with tribes in the United States over the past three years in an effort to reduce the rate of SIDS among Native people. This session will discuss the importance of developing partnerships with Native tribes, tribal organizations, and Native organizations as a means to reducing SIDS in the Native population. And the presenters will explore ways in which organizations can develop strong relationships and partnerships with Native tribes and Native organizations. This session should particularly benefit those who are interested in working with Native communities, and those who are concerned with the reducing the rate of SIDS among the Native population in both the United States and Canada.

How Men Grieve and How to Provide the Kind of Support They Want and Need

Oddbjorn Sandvik, Norway

I will begin with some remarks on how we understand grief in general, then discuss what we know about gender differences in grief. Then I will present some of my own experiences with grieving men and discuss some ways to organize grief support that is more appropriate to meeting the needs of grieving men than what we now offer.

July 5, afternnoon sessions

The Journey of our Spirit

Andy Blackwater

The Journey of Our Spirit session will guide participants on the journey as illustrated by the "Spirit of the Elk Teepee (Lodge) Design. The design was gifted to Mr. Blackwater's great great grandfather, "Keeper of Many Young Horses" by way of a dream vision. His Grandmother "Charges with Pride" transferred this teepee desing and the teepee's teachings to him when he was inducted into the Sacred Horn Society of the Blood Reserve in 1985.

Today there are many of these teepee desings on the Blood reserve and they are continually transferred down from generation to generation. Mr. Blackwater is the owner of two other teepees and he shares the teaching passed down from the distance past during this session. Teepp (lodge) desings carry special gifts for the owners and occupants. Every aspect of the "Spirit of the Elk" teepee (lodge) has a symbolic meaning in repsect to our reason and purpose for existence on earth. Mr. Blackwater is deeply honoured to share these traditional philosophies with you. For your interest, a pamphlet will be available to further illustrate the teepee's distinctions at the session.

Safe Sleep Practices To Reduce SIDS: It's Time for a Change

Tina Langille, Canada and Christine O'Meara, US

This workshop is geared toward a variety of professionals including Nurses, Nurse Practitioners, Physicians, Social Workers, Prenatal Instructors, Child Care Providers and others working with parents and infants. The workshop's goals are to present evidence-based SIDS risk reduction strategies and to engage health professionals in discussion around integrating the "Reduce The Risk" basics into the delivery of patient/client care and parent/caregiver education. Through interactive discussion participants will address concerns and difficulties with adhering to the current infant/toddler safe sleep recommendations, delve into controversial issues, examine barriers to change and explore possible solutions. A summary document reflecting the issues discussed and proposed solutions will emerge from the workshop

Part A: Basics

- *"Reduce The Risk" basics to include:* supine positioning for sleep, firm mattress with a tight fitting sheet, clutter-free sleep environment, smoke-free environment before and after birth, use of lightweight blankets for thermoregulation, and breastfeeding.

Part B: Professionals and SIDS

- *Your role as a professional caring for infants in lowering the risk of SIDS*: be informed about current research, help reduce parental anxiety by providing up-to-date evidence–based information about SIDS, practice and teach "Reduce The Risk" behaviors, access your

local/national resources and use educational tools effectively.

- *Monitor and SIDS*: discuss use of monitors in preterm infants in hospital and on discharge, the relationship between apnea/SIDS, and use of monitors with subsequent siblings (medically prescribed and store bought monitoring devices).
- *Institutional and Child Care policies*: learn about the application of a hospital safe sleep policy for term and preterm infants including how to transition to supine, review the standard of care and look at legal implications.

Part C: Safe Sleep Practice Barriers

- *Controversial issues*: aspiration and the supine position, plagiocephaly (flat heads), coping with a tummy sleeper, back versus side positioning, tummy time for awake babies, bed sharing/co-sleeping, and infant vaccinations.

Barriers to change: habitual practices, cultural/language diversity, alternate caregivers, grandparents, preterm infants, and inadequate public awareness.

Walking the Path to Responsibility

Debbie Dedam-Montour
Executive Director – National Indian & Inuit Community Health Representatives Organization

Tobacco cessation in Aboriginal communities is a challenging task that Community Health Representatives and other health workers are undertaking. There is no one easy way to get people to stop misusing tobacco that will apply to all communities. NIICHRO has developed a training manual that can be adapted to local needs and moves away from the heavy-handed messages presented by the mainstream media. This presentation will cover tips for health workers and bring forward ideas that could potentially motivate reduction or cessation of tobacco misuse. Presents the concept of having people with addiction to tobacco *Walk the Path to Responsibility* and work towards a smoke-free environment within their home and community by alleviating the feeling of guilt related to tobacco misuse as well as offer them a sense of action towards a healthier lifestyle.

"I have a headache" or "I just don't feel like it." Why Sex Is Suspended Following Child Loss?

Atle Dyregrov, Norway

A child is born as a consequence of love, sex, and intimacy. In the aftermath of a child's death there areas may symbolically become linked to the pain of the loss. As parents you find that invisible walls may find their way into the bedroom and create problems for the sexual relationship and with intimacy. Some (mostly women) associate sex with the pain of losing the child and they cannot even stand the thought of the sexual act. Others (mostly men) feel that the sexual act reduces the tension and provides the physical antidote to the pain that may be difficult to explain in words. Different emotional and physical needs may collide and the bed can become a the battle ground for these different themes. The aim of this presentation is to raise this "taboo theme" in such a way that it can prevent untoward blaming, intense arguments, and the sustained silence that can hamper the marital relationship.

July 6, Morning sessions

Living with Loss Over Time -- Bottle It Up Or Talk It Through?

Atle and Kari Dyregrov, Norway

Many studies of bereaved parents document the personal and family disaster of losing a child. Few studies, however, have focussed on what it is like to live with the loss over time. We will report on research results that pertain to the experiences of bereaved couples and how they deal with this loss over time. How do they commemorate and communicate about the dead child? What changes has the death created in their respective lives when it comes to values, life quality, and important decisions in life?

How can a couple or family best deal with the loss of a child over time? When the first active grief period is over, some family members (the majority of them men) use distancing as a strategy and want to get on with life and Look toward the future, while others (the majority of them women) want to continue to talk about the loss and cope best by approaching the loss. Is it best to bottle up feelings and thoughts about the loss or talk it through?
When strategies differ, how can couples handle this in such a way that both partners feel respected? These are the questions and themes that we will address in this presentation.

Nurturing Hope in the Face of Loss

Denise Larsen, Canada

How can we foster bereaved parents' hope in the face of devastating loss? How can we hold onto our own hope as clinicians when working with difficult situations like this? During this session, examine the nature and value of hope. Learn strategies to locate and enhance hope in your work with families. Take away new ideas to incorporate hope into your professional life.

Aboriginal Action plan on SIDS

Richard Jenkins, Canada

This session bring together Aboriginal stakeholders to discuss an action plan to address SIDS in our community. Aboriginal people in Canada endure a rate of SIDS that is anywhere from 3 to 8 times that of non-Aboriginal Canadians. Current research can only tell us how to reduce the risk – we must take action to empower our communities to reduce the rate of SIDS. This includes integrating SIDS awareness in current programs on infant health, addictions, and tobacco. We do not know what causes SIDS, so we should not conclude that our child care practices are to blame. The Action plan session will review the "Joint Statement on Reducing the Risk of Sudden Infant Death Syndrome in Canada" developed by The Canadian Foundation for the Study of Infant Deaths, The Canadian Institute of Child Health and the Canadian Paediatric Society. The goal of the session is for the community to produce a plan of action, and the commitments necessary to implement it.

Continuing Bonds with Your Deceased Child

Dennis Klass, United States

This is a presentation about how a continued relationship with an inner representation of deceased child is seen to be healthy, bring solace and resolution to the parents through spirituality, immortality and cultural rituals.
In this presentation we will look at the emerging model of grief used by researchers and clinicians. The presentation is based on two of my books: *Continuing Bonds: New Understandings of Grief* (1996) and *The Spiritual Lives of Bereaved Parents* (1999).

The dominant theory in the 20th century was drawn from Freud:

"Each single one of the memories and situations of expectancy which demonstrate the libido's attachment to the lost object is met by the verdict of reality that the object no longer exists." (1917).

In my role as the professional advisor to a self-help group of bereaved parents showed me that this did not describe what parents do with their bond with the child. I saw that many parents continue their bond, and that the bond with the deceased child plays important and often helpful roles in the parents' lives. Other researchers, working with other populations found the same thing. Indeed, we found that the model researchers and clinicians used was sometimes hurtful as bereaved parents tried to find their way to resolution.

In this presentation, we will look first at some of the ways continuing bonds function in the lives of deceased parents.

Solace:

Solace means pleasure, enjoyment or delight in the face of hopelessness and despair.

Solace comes into the pain, but does not remove the pain.
Solace is found within the sense of being connected to a reality that transcends the self. For bereaved parents, solace is connected with continued interactions with dead children.

Paths to Solace

- Linking objects
- Religious ideas and devotion
- Memory
- Identification

Rituals parents use to share their continuing bonds with their children

- Picture sharing
- Holiday candlelight service
- Cemetery tour
- Balloon and butterfly release

Samuel Marwit and I did some research asking what roles the deceased play in people's lives. The subjects were not bereaved parents. We found we could identify four roles.

Role model

- Situation-specific guidance
- Values clarification
- Remembrance formation

On the basis of our research, then there is an emerging consensus among bereavement scholars:
In the dominant 20th century "grief-work" model:
The *purpose* of grief is the reconstruction of an autonomous individual who in large measure leaves the deceased behind and forms new attachments.
The *process* is working through and resolving feelings.
In the contemporary model of grief:
The *purpose* of grief is the construction of a durable biography that enables the living to integrate the dead into their ongoing lives. The *process* is conversations with others who knew the deceased.

Conclusion:

We have only been talking about theory, but theory is the way we try to make sense out of the patterns we think we see in our experience. If we think more clearly, we can cope and help others to cope more effectively.

Scientific Program - Texts for Parents

We have asked all guests speakers presenting in the scientific program to provide a summary of their talk in non-scientific language.

They all made the effort to provide these additional documents for which we are grateful.

We have grouped the texts by order of presentation and date.

Fighting the highest SIDS rate

Ed Mitchell, New Zealand

In the early 1980s total infant mortality was high in New Zealand compared with other countries, and this was due to the high SIDS mortality rate. Furthermore there were marked ethnic differences in mortality rate with Maori being approximately twice the non-Maori, non-Pacific Islander (predominantly Europeans) rate.

While much can be learnt from overseas studies and retrospective review of SIDS cases locally there was a need to obtain New Zealand data with appropriate controls in order to find causes for the high SIDS mortality rate in this country.

This led to the development of the New Zealand Cot Death Study. This was a three year prospective case-control study, which compared 485 SIDS cases with 1800 controls. This research project commenced on 1 November 1987.

Because of public and health professional concern about SIDS we analysed some of the data from the first year of the study. As expected many risk factors for SIDS were confirmed. In addition, however, we identified risk factors, which were potentially amenable to modification. These were:

- prone sleeping position of baby,
- maternal smoking, and
- not breastfeeding.

The National Cot Death Prevention Programme was developed and was launched on 27 February 1991, although change in prone sleeping position occurred from the time the programme was being developed.

The SIDS prevention programme received a lot of interest both locally and overseas, partly due to the Thames television (UK) documentary "Every mother's nightmare" featuring our work, which has been shown in many countries. The prevention programme has been spectacularly successful. SIDS mortality rate in New Zealand has fallen from 4.4/1000 live births in 1988 to 2.3/1000 in 1992 and total postneonatal mortality from 6.1 to 3.6/1000 (1997). This equates to over 150 lives saved each year in New Zealand. Other countries followed our lead.

It is essential to monitor changes in SIDS mortality and changes in infant care practices to show a causal relationship between the two. We undertook a prospective study which collected data at birth and at 2 months of age on all infants born in New Zealand over a 2 year period. It showed that the prevalence of prone sleeping position decreased to less than 5% and confirmed the major risk factors identified in the New Zealand Cot Death Study.

A fourth risk factor, namely infants sharing a bed with another person, was added to the prevention programme in 1992. Further analyses found that the risk was predominantly among infants of mothers who smoked. The recommendation that infants of mothers who smoke should not bed share with their infants caused considerable controversy. Lactation consultants had been promoting bed sharing to encourage and support breastfeeding, and it was perceived by some Maori as an attack on their culture.

In 1995 a Maori SIDS Prevention Team was established under the guidance of Dr Tipene-Leach. Initially this focussed on the established risk factors but the social and health issues facing Maori are bigger than "risk factors" and much of the community health workers work evolved around dealing with social issues, such as housing. However, the importance of bed sharing as a cause of SIDS (more than 50% of SIDS deaths in NZ occur while

co-sleeping), became more apparent, and the Prevention Programme started addressing this issue. SIDS rates have continued to fall slowly, but more could be done.

New Zealand Government policy is that health funding is now done at district level. This means that trying to establish a national programme is just about impossible. There needs to be national agreement about health risk (and benefits) of co-sleeping, and for that matter pacifier use, and this needs to be prominently promoted.

Teen Health - STEPS to reducing the incidence of SIDS in the Teenage Demographic

Mary McCormick, Canada

Although many research studies have identified that teen mothers have a higher risk of SIDS, none of these studies actually address why. STEPS was developed in hope that we will be able to answer this question. The Canadian Foundation for the Study of Infant Deaths identified this need for educating this high-risk group and received a 2-year grant from the Trillium Foundation to develop and implement the STEPS program.

STEPS is a special communication and educational program that targets teen mothers and those that may be assisting in the care of an infant of a younger mother. This program responds to a unique target audience and at every step, involves this target group: teens and young adults. This program responds to an urgent need to educate young moms through a supportive educational training, teaching the steps to reducing the risk of SIDS and promoting good health before, during, and after pregnancy.

As stated previously, teen mothers have a higher risk of SIDS. A concern arose as to whether or not teens were receiving the back to sleep message. The belief was that they probably were, but perhaps not in a manner that they could relate to. Teenagers often have a carefree nature and sometimes seem disinterested in the medical environment. Some teens see nurses and doctors, parents and social workers as preaching and judgmental. Often teens avoid prenatal classes because of the fear that their limited knowledge may be grounds for having their child removed from their care. They can ask fewer questions and pretend to understand everything!

Mini focus groups were contacted in several areas to determine what type of materials should be developed to communicate the steps to reducing the risk of SIDS to teens.

Results of the survey led to the creation of a video accompanied with a guide and brochure, as well as a public service announcement (PSA). In addition, as teens are very technically inclined today, a special STEPS site was developed on the web with a chat area and a forum to post messages.

The PSA is a 30-second commercial for TV that makes the audience want more information and directs the viewer to call the 1-800 number for more information. A similar auditory PSA was developed for radio. In order to make the material more appealing and more impressionable to teens, we added elements similar to the fast paced formats currently used by music video producers to the look of the PSA and video. Our poster and brochure were designed in a color scheme chosen by teens with large graphics and as few words as possible. The graphics speak for themselves and communicate the message effectively.

Furthermore, although literature about SIDS is readily available, STEPS information concentrates on reducing the risk, not the SIDS event, so this literature is seen as less scary and threatening.

This program begins before pregnancy educating teens in the school system. All STEPS materials have been accredited by Curriculum Canada Services to be used as teaching tools for parenting classes or Family Studies. By reaching students before they have a child, we encourage good health practices, promoting healthy pregnancies and healthier babies. Students learn the steps to reducing the risk of SIDS, which can be beneficial if they are babysitters too. Nearly everyone knows pregnant women shouldn't take illicit drugs, but it's the legal ones--alcohol and tobacco--that are more commonly the source of pregnancy problems. Smokers put their

babies at a significantly higher risk of pre-term birth, low birth weight, and SIDS compared with nonsmokers. Students are taught good health is imperative to a successful pregnancy and parenthood.

STEPS also address the need of the expectant teen. Workshops are held educating the teens in the steps to reducing the risk of SIDS, stressing the importance of communication and a healthy lifestyle. Surveys are conducted to determine the needs and understanding of the participants.

The third aspect of the STEPS program addresses the teen mom and her baby. At this level, STEPS empowers the teen mom with the knowledge of reducing the risk of SIDS and increases her self esteem and confidence in dealing with those caregivers that assist her in childcare.

This training may have the added benefit of teens gaining presentation skills and in turn, higher levels of self esteem which can only positively affect all aspects of their lives.

In educating teens, it is important to address the need of educating all caregivers that may be involved in the care of the teen mom and baby. Grandparents, babysitters, teachers, social workers, prenatal instructors and more must be knowledgeable in these easy steps to reduce the risk of SIDS. This education must be a collaborative effort throughout the community to ensure that the message is the same from all involved.

In just one generation there have been significant changes in the way we parent our babies and children. It's not that grandma did the wrong things; she followed what were considered the best practices at the time. Grandparents may be confused about these new guidelines and insist that a teen mom is doing it wrong. STEPS empower teens with the knowledge necessary to reduce the risk of SIDS. Hopefully grandparents will have a healthy curiosity about the changes in baby care and will respect and support a teen's choices as a mother.

Currently, statistics are being gathered and in the future it is hoped that a reduction in the rate of SIDS in this target group will be seen. In order to measure the continued success rate of the STEPS program; trends will be monitored for several more years.

Together, teens and the Canadian Foundation for the Study of Infant Deaths explore the question of why teen mothers have a higher risk of SIDS, provide literature that is informative and non-threatening, and empower young mothers with knowledge that is science-based but friendly. This year, the program has provided support to nine teenage mothers whom have lost a child to SIDS. Of these nine, 3 babies were sleeping on their tummies and 2 on their sides. Age does not determine if someone will be a good parent or not, there are good and bad parents at all age levels, but education is key to reducing the risk of SIDS.

July 3, Morning Sessions

International Statistics on Infant Mortality and SIDS

Fern Hauck, United States

Dramatic declines in SIDS rates have occurred in virtually all countries in which these statistics are available. The reductions have exceeded 50% in most countries, and in some, approach 90% or more. While the final causal mechanism of SIDS is still uncertain, the decline of SIDS around the world has been attributed to reductions in prone infant sleeping position in countries in which this was the routine practice. Risk reduction campaigns, focusing on sleep position placement of infants among other risk factors, have succeeded in bringing about this change, which, in turn, has been clearly tied to the continuing decline in the number of SIDS deaths.

While these findings have been highly consistent, there is still considerable variability in SIDS rates across countries. Differences in rates could reflect a number of factors, including differences in: 1) risk factors for SIDS and their prevalence; 2) definitions; 3) autopsy protocols and rates of use; 4) death scene investigation protocols and rates of use; and 5) classification of death based on autopsy, scene investigation and other information about the infant. Additionally, concerns have been expressed that the reductions in SIDS are not as large as they appear, but may represent changes in classification within countries ("diagnostic shift") of some deaths that may previously been identified as SIDS more recently being identified as other causes of death, such as accidental asphyxia or "undetermined." A clue to this can be found in part by examining trends in postneonatal mortality rates in comparison to SIDS mortality rates within each country. Examination of cause-specific rates of sudden unexpected deaths in infancy (SUDI) can even better illuminate this question. In South Australia, for example, there has been a genuine decline in SIDS deaths, but there has also been a change in the diagnostic profile of SUDI, with an increase in deaths attributed to accidental asphyxia due to unsafe sleeping environments and cases designated as undetermined. The availability of extensive background information about complex issues (social and environmental, among others) may result in the greater use of the undetermined designation. So, while the overall postneonatal mortality rate fell, supporting a true fall in SIDS rates, it is important, also, to look at cause specific rates.

In this presentation, updated international data will be presented on SIDS rates and postneonatal mortality rates to examine and compare trends. Further, it will introduce the diagnostic and classification issues that play an important role in the interpretation of these statistical findings.

The changing profile of Sudden unexpected deaths in infancy
The Avon Experience

Peter S Blair and Peter J Fleming, United Kingdom

Since the acceptance of Sudden Infant death Syndrome (SIDS) as a registerable cause of death in 1971, the subsequent SIDS rate in England & Wales has followed a similar pattern to that observed in many other countries; a rise in incidence in the 1970's, peaking in the 1980's and a dramatic fall after the 1991 "Back to Sleep" Campaign. It has since been recognised that the initial rise in incidence was actually a diagnostic shift from death previously labelled as 'respiratory infant deaths' to the new classification of SIDS[1]. Thus, there is no evidence to suggest the high SIDS rates observed in the 1980's were substantially different in the previous decade, or for that matter, prior to the recognition of SIDS as a cause of death. The dramatic fall in SIDS rates over the last 15 years from 2 deaths per 1000 livebirths to less than 0.5 is directly related to the change in infant sleeping position over this time[2] . Given this change in infant care practice and decline in deaths we need to investigate how the epidemiological profile of SIDS infants has changed. National statistics show us that some factors such as gender remain unaffected, just over 60% of SIDS infants in England & Wales were males before and after the campaign, whilst the decline in deaths during the colder months and rise in younger SIDS infants suggest the profile has altered. However the limited detail of National statistics is restrictive and a more extensive collection of Regional data is required to provide the full picture.

We have been collecting SIDS data in Avon since 1984. Avon County is situated in the South-West of England with a population of over 800,000 people, predominantly white, with over 9,000 livebirths a year. The region is a mixture of rural and urban communities, the largest city being Bristol ranked 119th most deprived borough of 354 in the UK index of Local Deprivation[3]. The SIDS rate in Avon was higher than the national average during the 1980's (averaging 3 deaths per 1000 livebirths) and has fallen to around 0.5 deaths per 1000 livebirths subsequently. In the eight year period between 1984 and 1991 there were 256 SIDS deaths in Avon, whilst in the twelve year period since 1991 there have been just 69 SIDS deaths.

We will present changes in infant, maternal and social factors prior to and after the "Back to Sleep" campaign, a preliminary look at the data has revealed 3 important changes.

SIDS has traditionally been the largest component (80-90%) within the overall group of all sudden unexpected deaths in infancy (SUDI). The umbrella term SUDI also includes unexpected deaths that are subsequently explained such as death from unrecognised infection, unrecognised congenital anomaly, accidental death, non-accidental injury and death due to metabolic disorder. In Avon, prior to the "Back to Sleep" campaign 13% of SUDI deaths were explained; after the campaign this proportion has risen to 39%. Because it is usually not possible to differentiate between SIDS and explained SUDI until after the post-mortem the epidemiological investigation into SIDS in Avon has always been an investigation into SUDI, although information collected on the explained deaths has rarely been utilised. Given the proportional rise of explained SUDI future investigations can now use these deaths as a comparative epidemiological and pathological control group.

SIDS occurs in all social groups but is more prevalent in the socio-economically deprived groups. Recent studies suggest this economic divide between SIDS families and the UK population has widened.[4,5]. Since the "Back to Sleep" campaign the vast majority of SIDS families in Avon are from the socially deprived group with all that this entails in terms of low birth weight infants, short gestation, high parity, young maternal age, single parenthood and propensity to smoke and bottle-feed. Future SIDS studies can no longer just use a random control group from the population, a second control group of more deprived families is needed if we are to compare like with like.

The proportion of bed-sharing SIDS deaths has doubled from 14% between 1984 to 1991 to 28% in the subsequent 12 years in Avon. On top of that the proportion of co-sleeping deaths occurring on a sofa has also risen from 1% to 10%. However the actual number of bed-sharing deaths has declined from 4-5 a year from 1984 to 1991 to 2-3 deaths a year in subsequent years. This suggests that the number of SIDS deaths occurring in a cot has declined faster than those SIDS deaths occurring in the parental bed. Why this has happened is not clear but may be related to differences in how infants have been put down to sleep depending on whether the sleep is a solitary one or next to the parent. Again, however, this has implications on future studies in that we need not only to match for socio-economic status but also where the infant sleeps.

The epidemiological profile of SIDS infants has clearly changed and the future design of SIDS studies has got to change with it. The anticipated number of SIDS deaths has fortunately decreased whilst our knowledge of these infants and families has significantly increased. It is therefore important to utilise this information to construct groups of future control infants more closely aligned to the circumstances of SIDS infants.

Sudden unexpected infant death in Germany - case examination, diagnostic criteria, morphology and epidemiology

Thomas Bajanowski, Mechtild Vennemann, Germany

In 1990, 1,283 cases of sudden and unexpected infant death (SUDI) were registered in Germany corresponding to an incidence of 1.42/1,000 live births, but showing large local differences. Only about 50% of these cases were investigated by autopsy.

In the same year the Westfalian crib death study was started. This morphological/epidemiological case-control study was performed in the north-west part of Germany, in an area where about 4.0 million people live. Between 1990 and 1994, 238 SUDI cases (SIDS n=205, explained SUDI n=33) were investigated using a standardised protocol. During the same time period 23 cases of unnatural death (8 infanticides) could be identified.

The prone sleeping position, face-down position, smoking during pregnancy, complete covering, pillow in bed, lack of breast feeding, bedsharing with an adult and low socio-economic conditions could be established as the

main risk factors. Differences in the prevalence of these factors were present comparing the various groups of cases (typical SIDS, SIDS+, explained SUDI), but were not significant. At the beginning of the year 1992 a local prevention campaign was initiated and the incidence of sudden unexpected infant death decreased constantly until now. Simultaneously we observed changes in the epidemiological profile of the cases

In the German SIDS study (GeSID) which had been carried out between 1998 and 2001 in 18 centres in Germany representing about 50% of the population, 455 cases of sudden and unexpected infant death (8% typical SIDS, 80% SIDS+, 12 % explained SUDI) were investigated using a standardised protocol similar to that of the Westfalian study as well as 28 unnatural deaths. The groups defined by morphological criteria differed for 3 main variables only: breastfeeding, position placed to sleep and coughing the day before death. The epidemiological profile showed that the prone sleeping position further decreased (42%), the frequency of smoking mothers as well as of heavy smoking mothers (>10 cigarettes per day) increased (64% of the mothers smoked during pregnancy, 38% were heavy smokers). Important new factors are that 18% of the mothers were younger than 20 years old (1990-1994: 1%), and that a higher number of SIDS victims (50%) came from the lower socio-economic class.

Conclusions:

- Risk factors changed.
- A local and time-limited prevention campaign led only to limited success.
- It is neccessary to recognize new risks and to develop risk group-specific prevention methods.

Hypoxia, stressors, and vulnerability

Brain mechanisms that compensate for cardiovascular collapse

Ronald M. Harper, United States

The cause of death in the Sudden Infant Death Syndrome (SIDS) remains unknown, but involves a failure of breathing or a failure to supply enough blood to body parts where needed. Blood must be pushed through the body with enough pressure to reach important areas. Too much pressure may burst a blood vessel, but this is not the problem in SIDS. Too little pressure causes body parts to be starved of blood, and comes about when blood vessels relax (become larger) and the heart beats slower. We believe that in SIDS, a condition of "shock" occurs. In shock, the brain commands the blood vessels to relax and the heart to slow; the process lowers blood pressure so much that not enough blood flows to body parts. Unless the brain signals the blood vessels to constrict, and the heart to speed up, thus restoring blood pressure, death death will result.

We believe that brain areas which are responsible for restoring blood pressure when pressure goes too low have been damaged in some fashion, and do not operate correctly in SIDS victims. Breathing helps to raise blood pressure, so, if breathing stops as well, that help is lost. We use brain imaging to determine the parts of the brain affecting breathing, heart rate and blood vessel relaxation that might be damaged. We look at brains from children who have no breathing problems during sleep, and compare the images with those from children who have some breathing or blood pressure problems at night. We image the brain in adults who have trouble breathing during sleep and also the brains of very young animals to see if areas work in the same fashion in early life, since SIDS occurs in young infants.

We find damage in brain areas in both children and adults who have breathing problems during sleep. The injuries are in brain areas that determine whether blood pressure has gone too low, and which command other brain areas to raise heart rate and constrict vessels to restore pressure. In very young animals, some brain areas behave in an opposite way to that of adults when blood pressure is changed.

There are several questions remaining. How could the brain areas be damaged in the first place? Why would blood pressure drop so much, especially during sleep? What test might predict this damage other than an expensive image scan? The nature of the damage found in other patients with breathing problems during sleep could result from a severe exposure to low oxygen that the individual survived. The overwhelming evidence in SIDS cases is that some injury occurs before birth, perhaps from low oxygen delivery to the fetus. Blood pressure can fall for many reasons; pressure can drop (normally) to alarming levels during some stages of sleep. Pain from the intestines or other internal organs can cause a profound drop in blood pressure. Severe infection can also result in a shock-like condition. The issue in SIDS is obviously why some infants do not restore blood pressure; we suspect brain injury, largely from pre-natal causes. Several groups are attempting to develop a simple screening test that would find whether blood pressure is easily restored in a particular infant when pressure falls.

Is vulnerability a cause or consequence of Intermittent Hypoxia?

Karen Waters, Australia

A number of similarities have been observed between responses to hypoxia, and changes that are observed in post-mortem studies of SIDS infants. Once focus of recent research has been to find out whether clinical conditions that have been associated with SIDS can cause low oxygen (hypoxia) that is severe enough to lead to the types of changes observed in animal models.

It is also possible that small but repeated bouts of hypoxia insults can cause a cumulative damage that is more severe than any one insult could by itself.

In our laboratory, we have studied post-mortem tissue from infants who died from SIDS, and compared the results to the changes that we see in piglets after small and repeated exposures to hypoxia. Since other risk factors such as cigarette smoke exposure are also associated with SIDS, we have recently been examining whether exposure to nicotine makes the brain damage worse after our small, repeated insults to the piglets.

Our findings to date suggest that many similarities exist between the type and the pattern of brain damage that we see in our animals and in the SIDS infants. will result.

July 3, Afternoon Sessions

Translational SIDS Research: From Biome to Genome

Mechanisms of hypoxic cell death or tolerance: insights from genetic models

Gabriel G. Haddad, United States

One of the devastating clinical problems in early life (as well as in adults), is damage to the brain from diseases that limit oxygenation to the brain. We have used in the past mammalian tissues and now flies to understand the molecular mechanisms that form the basis of why injury occurs in mammalian tissues but not in other tissues such as in flies. If we understand the mechanisms in invertebrates we may be able to understand how to manipulate the system in mammals to render these tissues resistant to low O2 environments.

Proteomic and its application in basic and clinical sciences

Jennifer van Eyk, United States

Proteomic analysis refers to the ability to isolate and identify specific proteins within different tissue or cells of the body (humans or animals). Genes makes protein that execute the fonction programmed by the gene. The recognition that disease result not only in changes in specific genes, but also in specific proteins has long been recognized. However, proteins can also be altered or changed independently from genetic changes and it is now clear that specific diseases result in a pattern of change in proteins within the affected organ or nervous tissue. Dr. van Eyk will review the current techniques in proteomics to isolate and identify proteins. She will also review examples of the successful use of proteomics to identify changes in proteins that serve as markers of cardiopulmonary disease, as well as proteins that may serve to or predict the onset of disease. The development of sophisticated proteomic analysis, accompanied by the practice of assembling tissues banks of material from SIDS victims, should provide a unique opportunity to identify target proteins that play critical roles in SIDS.

Structural insights into neuroepithelial bodies: the link to hypoxia and SIDS.

Ernest Cutz, Canada

Our research is concerned with the study of recently identified specialized cell clusters in the lung called neuroepithelial bodies. Studies in our laboratory have discovered that neuroepithelial bodies represent oxygen sensors in the airway, monitoring oxygen concentration in the ait we breathe. The function of neuroepithelial bodies is to detect low oxygen (hypoxia) and send a signal to the brain to make appropriate adjustments including increasing breathing to compensate for the low oxygen concentration. We have been examining the ways how neuroepithelial bodies cells detect low oxgygen using a special equipment that can measure tiny electrical currents at the cell membrane. We have identified in neuroepithelial bodies an oxygen sensor comprised of a special signalling protein linked with an ion channel(small opening in the cell membrane).By manipulating the various components of the sensor we can increase or abolish the sensitivity and signalling of low oxygen by neuroepithelial body cells. For example,in a mouse model where the signalling protein in neuroepithelial bodies cells was made defective (so called knock out mice), neuroepithelial body cells show no response to low oxygen and their breathing is abnormal. We have also found that neuroepithelial bodies in lungs of SIDS infants are markedly enlarged, suggesting that these cells may not be functioning normally. This enlargment of neuroepithelial bodies could be the result of chronic low oxygen or abnormal development. Furthermore we have found that maternal smoking during pregnancy (a well known risk factor for SIDS) causes enlargement of neuroepithelial bodies, possibly resulting in abnormal function. Inflammation in the airway, as seen with mild respiratory infection(an other recognised risk factor for SIDS), could also interfere with the normal function of neuroepithelial bodies. Hence neuroepithelial bodies provide an important link between the external environment ("external stressors") and the Central Nervous System which controls respiration. The better understanding of how neuroepithelial bodies function in normal lung and in various disease states including SIDS may provide new information for devising new therapeutic and prevention strategies.

Metabolism and respiration: a comparative matrix - effects of temperature and hypoxia.

Peter Frappell, Australia

In small mammals and newborns, in general, a decrease in the rate of oxygen consumption occurs on exposure to moderately low levels of oxygen. This decline is mostly related, though not always, to a decrease in thermogenesis (production of heat) and hence is associated with a decline in body temperature. Most interestingly is that in all situations of moderate decrease in oxygen, ventilation (our rate of air taken up during one minute) is appropriately matched to changes in oxygen consumption. This suggests that supply is correctly matched to demand and this step in the O_2 cascade is therefore appropriately regulated. This talk adopts a comparative approach to understand the interplay between oxygen consumption and ventilation and to explore the effects of low levels of oxygen and changes in body temperature on this relationship and the possible implications for respiratory control.

Augmented sleep apnea in orexin knockout mice

Tomoyuki Kuwaki, Japan

Not only SIDS victims but also healthy humans experience respiratory arrest (apnea) during sleep. We are seeking brain substances that may increase or decrease apnea episodes for better understanding of the etiology of SIDS. For this purpose, we are using mice as the model animal because it is relatively easy to increase or decrease a specific gene product in mice's brain. Although it was not easy to measure respiration together with sleep/awake state in a tiny mouse, we have successfully developed a method to do so. In such a series of experiments, we found that deficiency of orexin, one of the neurotransmitters in the brain, resulted in an increase of sleep apneas. Thus, orexin seems to contribute for preventing sleep apnea and possibly SIDS.

Integrative cardiopulmonary control in mutant murine model

John Fisher, Canada

Scientists have long used animal models to test hypotheses about the cardiopulmonary control systems that may be involved in SIDS. However, the advent of the human genome project, as well as the sequencing of the genome of other animals such as the mouse, has provided an explosion of information and opportunity for research. Dr. Fisher's presentation will review the development of the mouse as a model for cardiopulmonary control and the strengths and weaknesses of the model to predict human physiology. The development of mutant and transgenic animals, which have specific genes "knocked out" or up-regulated, provide a highly novel development in the use of murine models and such approaches now impact on virtually every area of biological understanding. Several promising knockout murine models have been studied, as outlined by Dr. Kuwaki. This presentation will focus on other examples of genetically altered mice including some that display altered control of breathing relevant to SIDS. These include mice lacking the dopamine transporter (DAT), muscarinic receptor proteins, and the transient receptor potential vannilloid 1 (TRPV1) ion channel.

New Frontiers in Genetics and Neuroscience

Gene-Environment Interactions: Implications for Sudden Infant Death Syndrome

Carl E. Hunt, United States

Sudden Infant Death Syndrome (SIDS) has historically been associated with environmental risk factors. Most human conditions, however, are determined by genetic and environmental interactions, and both are essential. This presentation will review what is known today about genetic and gene-environment interactions for representative human diseases and about the relevance of gene-environment interactions to a broadened perspective on pathophysiology of SIDS.

There are several implied but incorrect assumptions that serve as barriers to accepting genetic risk factors and gene-environment interactions as causal in SIDS. They have persisted as barriers despite our rapidly expanding genomic knowledge base:

- SIDS is a unique and distinct disorder that does not follow the same "rules" as other human disorders
- Genetic disorders are caused by an abnormal gene; there is a 1:1 relationship between gene and disease, and having that gene predicts destiny
- If SIDS is a genetic disorder, then there must be a SIDS gene
- If there is a SIDS gene, then the parental source of this gene is "at fault"
- Diseases are either genetic or environmental in their origin, but not both
- The existence of major environmental risk factors, therefore, precludes the concurrent existence of causal genetic factors

Several genes have now been identified for which the distribution of variants differs in SIDS victims compared to control infants. These include a sodium channel gene (long QT syndrome), the serotonin transporter protein (5-HTT) gene, and several genes pertinent to development of autonomic nervous system. The list of genetic differences in SIDS victims may expand considerably as additional genetic studies are performed.

Several important environmental factors are associated with increased risk for SIDS and may indeed often be the life-threatening trigger. Susceptibility to SIDS in individual infants, however, is likely determined not by the trigger alone, but by genes interacting with challenges from the environment. Like other diseases, therefore, SIDS likely occurs when the environmental challenge leads to a lethal change in the presence or expression of one or more gene products affected by that gene variant.

There are multiple ways in which known environmental risk factors may be interacting with specific gene variants (genotypes). There appears, for example, to be an interaction between prone/side sleep position and impaired ventilatory and arousal responsiveness. There may also be links with modifiable risk factors such as soft bedding, prone sleep position and thermal stress, and links between genetic risk factors such as ventilatory and arousal abnormalities and temperature or metabolic regulation deficits. The increased risk for SIDS associated with exposure to cigarette smoke also appears at least in part to depend on genetic risk factors.

In summary, genes and proteins interact to produce complex networks which in turn interact with the environment to influence every aspect of our biologic lives. Failure to consider *both* genetic *and* environmental context impedes research progress whereas the study of dynamic gene-environment interactions constitutes a powerful strategy for enhanced knowledge of sudden unexpected death and SIDS.

Developmental neurotransmitters pathology in SIDS

Sachio Takashima, Yuri Ozawa, Japan

The campaign of sleep position in supine state has reduced the incidence of sudden infant death syndrome (SIDS), but SIDS is still the main cause of postnatal infant death. The causes and mechanisms in SIDS have not been elucidated. SIDS has the characteristics of occurrence time in early infantile period and in sleeping state. Previous studies on the brain of SIDS have revealed some minute lesions suggesting chronic or repeated low levels of oxygen. Also, the brainstem of SIDS infants has shown a particular type of scarring (gliosis) in respiratory centers, for instance, and abnormalitites of neurotransmitters, suggesting the possibility that abnormal development of the neuronal circuitry in the brainstem, the neurons of which regulate rhythmic breathing and arousal, underlies cardiorespiratory instabilities.

In the SIDS infants with brainstem gliosis, a neurotransmitter called substance P was increased in some regions suggesting the close relationship between chronic hypoxia and neurotransmitter development in SIDS.

Many neuron system have been studies in SIDS victims and in control infants in various regions of their brainstem. Several diferences have been found with in general a decrease in certain types of neurons in key areas.

In the pathogenesis of SIDS, there are predisposing and environmental factors. Recently genetic studies disclosed some responsible genes such as SCN5A channel and serotonin transporter as predisposing factors, while neuropathological studies revealed subtle developmental abnormalites in various regions.

The changes of neurotransmitters may be secondary ones induced by chronic low levels of oxygen in the blood or repeated low perfusion of certain areas of the brain in SIDS, but may be primary, reflecting some impairment of the developing neuronal system.

Larry Becker Memorial Lectures – Serotonin Symposium

Introduction to the Larry Becker Memorial Lectures

Hannah Kinney, United States

Dr. LE Becker was an extraordinary pediatric neuropathologist and colleague. He made many outstanding contributions to our understanding of brain tumors, mental retardation, inborn metabolic disorders, and muscle disease in children. He is most remembered by this community for his important contributions to SIDS brain research. In this memorial lecture, Dr. Becker's work concerning the neuropathology of SIDS will be reviewed in light of his many insights into deciphering SIDS.

Serotonin transporter gene polymorphism as a risk factor for SIDS

Maasaki Narita, Naoko Narita, Japan

Pediatricians who have ever seen SIDS victim can tell you how abnormal it is the completely healthy baby should die in that manner. We, pediatric-researchers believed that there must exist a certain pathology which makes SIDS happen, and that was where our SIDS study begins. In the symposium, I will talk about our recent discovery that a serotonin-related gene is significantly associated with occurrence of SIDS.

Serotonin is a neurotransmitter which modulates diverse brain functions including respiration. There had been reported dysfunctions in serotonergic neuronal system in some SIDS victims, however, no definitive mechanism has not been identified yet. There are several genes that function as regulator of serotonin activity, including serotonin transporter gene. With our careful study of comparing the genetic information obtained from 27 SIDS victims and 115 healthy controls, we were able to identify a critical difference in the distribution of a serotonin transporter gene polymorphism between SIDS victims and controls (Pediatrics, 2002). Since genetic involvement in SIDS pathogenesis had been denied until then, it was considered as a landmark discovery on SIDS as referred below: .

"Japanese researchers have uncovered a genetic variation that may underlie SIDS, the largely unexplained crib death that can strike otherwise healthy babies" (Reuters Health, Apr 02, 2001).

Our finding was further confirmed by US group recently (Weese-Mayer and collaborators) and it may pave the way for therapeutic and preventive strategies for SIDS.

Serotonin neurons as sensors of blood CO_2: Role in control of breathing.

George B Richerson, United States

A subset of SIDS cases has recently been linked to abnormalities of the chemical serotonin in the brainstem, but it is not known how these abnormalities could cause SIDS. We are studying serotonin-producing neurons in rats and mice. Since the function of these neurons in animals appears to be the same as in humans, defining their normal role in rodents may help to determine how a defect in serotonin could lead to SIDS.

We have found that serotonin neurons have the properties of sensors of arterial blood carbon dioxide (CO_2), and we propose that they initiate reflexes aimed at maintaining normal CO_2 levels. This CO_2 control is important, because when CO_2 rises it causes an increase in acidity of the, and even a small increase can be fatal. CO_2 is a normal byproduct of metabolism in the healthy body, and it is exhaled during breathing. Thus, the most important mechanism for controlling blood CO_2 is to change the depth and rate of breathing. Arousal is also important for CO_2 control in a sleeping individual with airway obstruction, because waking up allows one to relieve the obstruction. Our evidence from studies of rat neurons supports a role of serotonin producing neurons in both responses to CO_2: breathing and arousal. These neurons increase their electrical activity in response to a rise in CO_2. They also wrap themselves around large arteries in the brainstem, where they would be able to accurately monitor CO_2 levels in the blood after it has left the heart and lungs. In response to a rise in CO_2, these neurons appear to cause an increase in breathing and cause arousal, and would thus restore CO_2 back to normal.

This work points to a plausible hypothesis for some cases of SIDS. A defect in serotonin neurons due to environmental or genetic factors, when combined with the normal immaturity of the brainstem and the normal instability of breathing during sleep, may lead to a decreased ability to recognize the rise in CO_2 that occurs when the face is covered by bedding. Such an infant would not respond by waking up and breathing more. This could lead to a severe increase in CO_2, increase in blood acidity, and low oxygen. These data suggest that animal models with defects in the serotonin system may be appropriate for studying the mechanisms of a subset of SIDS cases.

Relationship between Serotonin related Factors and Neuronal Plasticity in Sudden Infant Death Syndrome

Toshiko Sawaguchi, Japan

Recently a role for serotonin in SIDS was proposed. On the other hand, a recent report suggests that abnormal neuronal plasticity was possibly associated with the arousal pathway in sudden infant death syndrome. In our studies, we look at serotonin-related factors and neuronal plasticity and examined whether the presence of sleep apnea was correlated with the pathological findings in the brain. In addition, the relationship between serotonin and neuronal plasticity in SIDS was investigated.

Our group consisted of 38 infants out of 27000 infants studied in a sleep laboratory where there respiration was carefully analysed during sleep.

Following analyses between physiological data and pathological data, some SIDS-specific significant correlation were found and will be described.

Our findings suggest that serotonin related factors and neuronal plasticity in the brainstem arousal pathway might be independently related with SIDS. Further investigation should be carried out for the correlation between serotonin related factors and neuronal plasticity.

Symposium on Upper Airways

Evidence for the Involvement of Obstructive Sleep Apnea (OSA) in SIDS

Bradley Thach, United States

Is acute upper airway obstruction a significant stressor causing SIDS? Upper airway obstruction in the nose, pharynx (throat) or larynx has long been a leading theory for cause of SIDS. Evidence for this has come from multiple studies. The evidence is summarized here: 1) The vast majority of SIDS infants are presumed to die during sleep and obstructed sleep apnea (OSA) is entirely a sleep dependant disorder. 2) Severity of OSA episodes is increased by increased nasal inflammation such as occurs with viral upper airway infection which is itself a risk factor for SIDS. 3) Certain facial features are strongly associated with increased risk for OSA (eg. small jaw or large tongue). Significantly, such abnormalities are reportedly increased in SIDS infants. Furthermore, sudden death from OSA is well documented in infants with these abnormalities. This, therefore links certain facial features to OSA and also to sudden death. 4) A large epidemiological study found that a family history of OSA is a risk factor for SIDS. 5) A prospective polygraphic study of sleeping infants found that episodes of obstructive apnea were more common in infants who ultimately died of SIDS compared to survivors. 6) The distribution of thymic petechiae in SIDS victims suggests that upper airway obstruction is a precipitating event in 70 % of SIDS cases. Finally, new evidence indicates that when infants are in the prone sleep position, their pharyngeal airway is vulnerable to collapse. That is the collapsible pharynx is more likely to initiate OSA episode. All in all, we conclude that there is strong, albeit indirect, evidence indicating that OSA either predisposes and infant to SIDS or actually acts as the final critical stressor causing death. Preliminary data from our lab provides direct evidence suggesting that complete airway obstruction immediately precedes death in some SIDS cases (see Pylipow M, Harris K, Thach BT, Abstract presented at this meeting). Whether or not this represents obstructive sleep apnea or some other entity such as blockage of the airway with gastric contents or secretions is unclear.

Anatomy of the Upper Airway in Infancy - and its Possible Relevance to SIDS

Shirley Tonkin, New Zealand.

Firstly, I would like to pay tribute to SIDS parents whose sad words have given me the ideas that may explain the mechanism of some of these tragedies - and thus suggest ways to lessen the chance of future deaths.

One parent told me that when she picked up her prone sleeping baby the bottom part of his face ' was all squashed in '

Investigations showed that this jaw movement was indeed possible in early infancy. How can the lower part of a baby's face become deformed?

The infant head at birth is about 1/4 of the whole body length – adults heads are 1/8 of the height. Infant spines are almost straight, the head sits on top like a toffee apple on a stick. To lie flat on the back the infant head must be flexed forwards.

Adult spines have two curves forwards - at the neck , and at the pelvis. Thus the back of the head is in line with the back of the shoulders in early adulthood and a person can lie supine without neck flection. The infant's cranium is almost adult size, but the lower face is very small and the mandibular articulation with the cranium is unstable as there is no mandibular head, and no glenoid fossa th limit backward movement.

The infant jaw has strong muscles for suckling with a biting motion. Within the infant jaw is situated the almost adult sized tongue which provides the infant with the ability to swallow liquids. It is in close proximity to the back of the pharynx, and thus forms a portion of the anterior wall of the upper airway.

The size of the airway will thus depend upon the size of the jaw - and the relative size of the tongue. Both are variable. Babies born with very small jaws (micrognathia) have been shown to have more breathing problems than normal babies.

So the size and thus the function of the upper airway is vital for the delivery of oxygen to the lungs. Upper airway size can be compromised by birth abnormalities of the passage - or of the mandible. It can be influenced by the muscle relaxation of sleep. It's size can be reduced by suction within the infant's airway - or by pressure from without. External pressure can come from outside agencies, or from the infant's own body

We have shown with imaging techniques that these circumstances do occur - and also the compromise of the upper airway can cause oxygen desaturation and severe bradycardia.

Could this be the mechanism of many SIDS deaths?

Genetic Diseases and Syndromes Affecting Upper Airways

Anat Shatz, Israel

Upper airways disorders may be responsible for sleep disordered breathing (SDB), stopped breathing (apnea) and Sudden Infant Death.

A large number of genetic disorders manifest SDB. Understanding how these genetic diseases and syndromes affect sleep and breathing may shed light on the mechanisms of SDB and increase our understanding of episodes of stopped breathing and Sudden Infant Death.

Genetic disorders that manifest SDB can be caused by anatomical defects, neurological defects, or by a combination thereof.

Several examples of such disorders, their clinically significant manifestations, as well as practical recommendations will be reviewed and discussed.

Cigarette smoke exposure and obstructive sleep apnea

André Kahn, Belgium

Maternal smoking during pregnancy is associated with an increased risk of Sudden Infant Death syndrome (SIDS). The frequency of SIDS victims increases with the amount of cigarettes smoked during gestation. The cigarettes smoked by the father add to the effects of those smoked by the mother. Smoking during gestation induces three main types of effects. First, smoking increases the frequency of repeated airway obstructions, obstructive apneas, during sleep. Such apnea are associated with drops in oxygen content in the blood, and drops in heart rate. Second, smoking during gestation also modifies the child's heart rate, that becomes rigid as if under constant stress. Finally, cigarette smoking during gestation reduces the infant's possibilities to wake-up from sleep. The reduced arousability diminishes the chance of the infant to react and to autoresuscitate when exposed to a life-threatening condition. These changes occur prenatally and contribute to increase the risk to die during the early months of life for a child from a smoking mother.

July 5, Morning Sessions

Tobacco and smoking

Introduction

Ed Mitchell, New Zealand.

There are many studies that have investigated the relationship between maternal smoking in pregnancy and sudden infant death syndrome (SIDS). Infants born to mothers who smoked in pregnancy have a 4.7 fold increased risk compared with infants whose mothers did not smoke. It is difficult to distinguish the effect of active maternal smoking during pregnancy from exposure to maternal smoking by the infant after birth.

Most attention has been directed at the mother's smoking behaviour, it would appear that smoking by the father increases the risk slightly. Infants exposed to smoking by the father where the mother is a non-smoker have a 1.4 increased risk of SIDS compared to infants not exposed to tobacco smoking at all.

A number of mechanisms by which maternal smoking may increase the risk of SIDS have been proposed, but so far none of them has been conclusively proven.

SIDS mortality might be reduced by between a third and a half if no fetus was exposed to maternal tobacco smoke.

In conclusion, smoking is causally associated with SIDS, although which of the many mechanisms is the most important is uncertain.

Cigarette smoke, infections and SIDS

Caroline Blackwell, Australia

Exposure of children to cigarette smoke is a major risk factor for SIDS and for serious chest and ear infections. The term passive exposure to cigarette smoke somehow implies that this route of exposure is less harmful than active smoking. Cotinine is a breakdown product of nicotine produced by the liver. Studies in Australia found that some infants exposed to cigarette smoke had levels of cotinine similar to those of active smokers. This

indicates that interactions between cigarette smoke and factors contributing to susceptibility to infection in active smokers might also apply to children who are "passive" smokers.

Many risk factors for SIDS parallel those that put children at greater risk of chest and ear infections or meningitis. In some Indigenous groups in which there is a high incidence of SIDS, there are also high incidences of infections. A study of Indigenous infants in Canada who died of SIDS found the majority had some recent symptoms of chest infections prior to death. Groups such as Asians (Bangladeshis, Pakistanis and Indians) living in Britain have had a low risk of SIDS and also low risk of infant deaths due to chest infections. Among Asian women, smoking is much less prevalent (3%) than that reported for Aboriginal Australian women (75%).

How does exposure to cigarette smoke increase risk of infectious diseases? Cigarette smoke can affect two important steps leading to disease: 1) attachment of the bacteria to the surface of the nose and throat; 2) balancing the body's normally protective responses that help to remove bacteria and viruses from the blood.

How do babies acquire bacteria and viruses? Babies get the bacteria that normally live on body surfaces mainly from their mothers. The mother has the closest and longest contact with the child, especially during the first few months of life. Smokers more often carry potentially harmful bacteria in their nose and throat than non-smokers. Smokers are also more likely to carry *Staphylococcus aureus,* bacteria that produce powerful toxins which we identified in over half of SIDS infants from 5 different countries. Close prolonged contact such as bed sharing could increase opportunities to pass bacteria from mother to baby.

In laboratory studies, cells from the mouths of smokers were stickier for many types of bacteria that cause infections in young children. Tar in cigarette smoke might be one of the "sticky" components responsible. Cigarette smoke makes adults and children more vulnerable to colds, coughs and "flu". The viruses that cause these can create new attachments sites in the nose and throat for bacteria or increase the ones naturally present.

How does cigarette smoke affect the body's defences against bacteria? The body's natural defences to bacteria and viruses play an important part in controlling disease. These defences are called the inflammatory responses. An example of local inflammatory responses is an infected cut that becomes hot, red, swollen and painful. These reactions can occur on a large scale throughout the body. There are two branches of this system: 1) **pro-responses** are due to a variety of messenger proteins that kill bacteria and viruses; 2) **anti-responses** are due to messenger proteins that switch off the pro-response messengers when they have killed the bacteria. If the pro-responses are not controlled, they can cause death as in meningitis or pneumonia. There is good evidence that some pro-responses are involved in SIDS.

There are environmental and genetic factors that affect the levels of both types of responses. Virus infection can turn up pr-responses and cigarette smoke can turn down anti-responses. There are small inherited variations in genes that can affect the basic level of the messenger produced. Cigarette smoke had variable effects on production of pro-responses; however, it consistently reduced one of the important anti-responses, regardless of the genetic makeup of the person. The greatest effect of cigarette smoke was seen for gene variants that are predominant among both Bangladeshi and Aboriginal Australian groups tested . If the genetic component were the most important factor, the risk of infection and SIDS should be the same in these two groups. The results suggest that exposure to cigarette smoke is the key factor.

Conclusions. We are beginning to understand why exposure of infants to cigarette smoke is a risk factor for infection and SIDS. We need the involvement of health educators, particularly those from indigenous groups in which many mothers are smokers, to develop effective education campaigns to keep infants "smoke free".

Environmental tobacco smoke exposure and SIDS in Germany

Thomas Bajanowski, Mechtild Vennemann, Germany

In the *German Study on Sudden Infant Death (GeSID)* 333 infants who died from SIDS and 998 living control infants were investigated to learn more about etiological and risk factors of SIDS including smoking. Furthermore in 135 SIDS cases and in 19 infants who died suddenly and unexpectedly from explained causes of death, nicotine concentration (NC) in hair as well as cotinine levels (CC) in body fluids were determined quantitatively. The NC in hair is a marker for an exposure to tobacco smoke over weeks or months while cotinine is a product of nicotine metabolism and gives information on tobacco smoke exposure over the previous few hours.

Results:

- Maternal smoking during pregnancy was associated with an increased, dose-dependent risk for SIDS (odds ratio 3.0).
- Infants of mothers who smoked during pregnancy had higher NCs in hair than infants of non-smoking mothers. The relationship between NC in hair and the daily maternal cigarette consumption was weak.
- There was no evidence that NCs and CCs differed between infants showing respiratory tract infections and so-called typical SIDS cases.

Conclusions:
Smoking during pregnancy is one of the most important (variable) risk factors for SIDS in Germany. Maternal smoking during pregnancy can be evaluated by determination of NC in the infant's hair. The NCs and CCs determined are lower than levels determined in active smokers. An association of parental smoking and respiratory tract infection in infants has been assumed, but could not be shown in our study.

Pathology and prevention of unexpected perinatal death and SIDS

Luigi Matturri, Italy

Among the many factors predisposing toward sudden infant death syndrome (SIDS), parental cigarette smoking has a predominant role. Indeed, the data in literature indicate that parental smoking is the major risk factor not only for SIDS but also for unexpected fetal death.

Our pathologic investigations of a vast series of unexplained perinatal deaths and SIDS have revealed frequent congenital anomalies both of the brainstem regions having a role in respiratory, cardiovascular and initial digestive tract activities and of the cardiac conduction system, in both SIDS and unexpected perinatal death victims. These findings seem to indicate a link between these congenital anomalies and a presumably genetic basis.

Study of the risk factors has demonstrated that the combustion products of cigarette smoke have a role in both atherogenesis (production of arteriosclerosis) and neural damage. We recorded a high incidence of initial atherosclerotic lesions in the coronaries of fetuses with smoker mothers. If the maternal smoking habit persists, true scarring plaques can be observed in the infant arteries, which are accentuated if the effect of nicotine is associated with that of artificial milk, due to a synergic action of the two factors.

Regarding the harmful effect exerted by the combustion products of tobacco, a significant association was found in our series between a very small size for one important region of the brainstem: the the arcuate nucleus and maternal smoking habit during pregnancy.

All the above lesions can be attributed to the direct action of the combustion products of nicotine on the smooth muscle cells of the arterial walls and/or on the neurons, interfering with the proper functionning of the cells.

These results suggest that the significance of the other risk factors considered important for SIDS may be revised, especially the question of the newborn's position in the cot, which has been assigned a fundamental importance in recent years that is not supported by anatomo-pathologic data.

Analysis of our series shows that it is essential for a complete autopsy to be performed, only by an experienced, reliable pathologist, in SIDS victims, including in-depth histological analysis of the cardiorespiratory innervation and specialized myocardium, as established at the 7th SIDS International Conference in Florence, Italy. In fact, our autopsy data showed borderline lesions or lesions of a different nature in 15-20% of cases, leading us to exclude the diagnosis of SIDS.

Tobacco and Smoking: Treating Tobacco Use among Pregnant and Parenting Smokers

Cathy L. Melvin, United States

Despite evidence of increased risk to themselves, their pregnancies and their families, a significant proportion of pregnant women continue to smoke throughout pregnancy. Similarly, an even larger proportion of parents and caregivers continue to smoke around infants and children. Clinicians providing prenatal care for these women and pediatric care for their newborns and infants are in a unique position to offer clinically proven, effective interventions. The gap between what is known about how to treat pregnant and parenting smokers and what is actually done in clinical practice is large with fewer than half of all obstetricians surveyed in the US offering recommended treatment.

A growing volume of research since 1975 has demonstrated that clinically proven, effective interventions exist to produce long-term or even permanent abstinence from tobacco for all smokers. Achieving cessation is important for all smokers but especially for pregnant and parenting smokers since their smoking poses risks not only for themselves but also for their pregnancies and children. Treatments for smokers in general apply to parenting smokers but special considerations regarding treatment need to be made for pregnant women. Due to the harms associated with exposure to environmental tobacco smoke (ETS), or secondhand smoke (SHS), both parents and caregivers of young children should receive treatment to achieve cessation or counseling on how to eliminate exposure of children to ETS/SHS.

This presentation will provide a review of the recommendations made in 2000 by the United States Public Health Service regarding treatment for pregnant and parenting smokers, summarize recent findings that may impact treatment protocols and make recommendations regarding further research in treatment approaches for pregnant and parenting smokers. A summary of recommended changes in treatment approaches for clinicians based on this review and a description of factors affecting clinician adoption and use of proven treatments and systems supports found to increase the likelihood of clinician use of these treatments will be discussed.
Efforts underway in the United States to implement these recommendations will be presented with a special focus on the work of the National Partnership to Help Pregnant Smokers Quit, the only national organization in the US working to mobilize health care systems and local communities to help pregnant smokers get the help they want and the support they need to quit smoking and remain tobacco-free. More than 60 member organizations of the National Partnership have joined forces to put evidence-based approaches to reducing tobacco initiation and use into practice at the national, state and local levels. The National Partnership's Action

Plan, working group structure and evaluation process will be described including efforts to improve the healthcare system, use the media, promote community and worksite policies and programs, pursue federal and state policy initiatives and promote research, evaluation and surveillance.

Infection and Immunity

Evidence for inflammation and altered immune response in SIDS infants

Åshild Vege, Torleiv O. Rognum, Norway

Signs of inflammation in SIDS infants have been noted since 1889 when it was claimed that the SIDS infants had enlarged thymuses and bronchitis. Since then, many researchers have noticed signs of inflammation and also that a large proportion of SIDS victims have signs of infection prior to death.

When comparing SIDS victims and babies dying from serious infections, we have found similarities between the two groups; both have elevated levels of hypoxanthine in vitreous humor, indicating that the babies may have experienced periods of oxygen deficiency (hypoxia) prior to death. We also have found increased levels of the signal substance interleukin-6 (IL-6) in the cerebrospinal fluid in half of the SIDS victims, in the same levels as in infants and children dying from serious infections. IL-6 is a small protein that belongs to the so-called cytokines. These proteins act as signal substances between cells in the immune system and different target organs, like the walls in blood vessels, the liver, bone marrow and the central nervous system. The cytokines are as important to the body as hormones and nerve signal transmitters. They are produced by many different cells in the body, and for instance bacteria and virus can stimulate their production.

The fact that the SIDS babies, in spite of the high IL-6 levels, only had experienced slight symptoms of infection prior to death favors the hypothesis that at least a part of the SIDS babies over-react to an otherwise harmless infection. IL-6 produces fever and this may induce irregular breathing, and also increased frequency of apnoeic episodes. Stimulation of neural reflexes in the upper airways may worsen this situation and may also induce disturbances in the heart rhythm (bradycardia). The infant may then develop severe lack of oxygen (hypoxemia) and ultimately coma and death.

Development of Infant Mucosal Immunity in Relation to Vulnerability to Infections

Maree Gleeson, Australia

In humans, the immune system has developed to provide a special form of protection against infections at external body surfaces, known as the mucosal immune system. It comprises a network of immune structures in the upper airways, gastrointestinal and urogentinal tracts and during pregnancy includes the lactating mammary glands. The interconnecting network allows immune cells to migrate from one site to the other mucosal sites, providing an extensive immune protection at all mucosal surfaces.

At birth the human mucosal immune system is fully developed, but in the absence of an intrauterine infection, the development of protective mucosal immunity by the baby occurs in the postnatal period. Researchers often test whether the mucosal immune system is working effectively by measuring antibody levels in saliva, called immunoglobulin A (or secretory SIgA). These antibodies provide protection against infections and appear between 2-4 weeks of age. Protective immunity is usually fully established by 3 months, however, the age of maturation varies between individual babies and the period form 1-6 months is critical to this process.

After birth the development of mucosal immunity is influenced by a number of factors, including the initial suppressive effect of the mother's own antibodies on the infant's immune development. The neonatal feeding practices; maternal and infant nutrition, vaccinations, natural exposure to infections and exposure to tobacco smoke all influence the how the baby's immune system develops.

The mucosal immune responses to infection during this development period can lead to inflammation in the airways and gastrointestinal tract if the baby's immune system has not developed to cope with the infections. Studies of SIDS infants indicate that uncontrolled inflammatory responses are a common finding and are often associated with prior respiratory tract infections. If this occurs in conjunction with other known risk factors for SIDS, the pro-inflammatory responses to the infection are potential contributing factors that lead to apnea (cessation of breathing) and unexpected death. Interactions with other known risk factors for SIDS during this period that could heighten the hyper immune response are: the prone sleeping position (the increase in airway temperatures allows the bacteria to grow); changes in night time cortisol levels (without adequate cortisol there is a lack of suppression of the inflammation); exposure to cigarette smoke (increase in bacteria and further stimulation of the immune response); and weaning from breast feeding (loss of maternal protective antibodies). The risk is even greater in some ethnic groups who have a genetic risk for pro-inflammatory responses to infections, particularly if the baby is exposed to tobacco smoke.

The common bacterial toxin hypothesis for SIDS

James A Morris, United Kingdom

In the last months of pregnancy antibodies (complex molecules that protect against infection) are pumped across the placenta from the mother's blood to her baby's blood. These antibodies protect babies against infection by bacteria and viruses in the first few weeks of life. As soon as babies are born they start to produce their own antibodies and the concentration slowly rises in the blood to reach 60% of adult levels at 12 months of age. The total amount of antibody in an infant's blood thus initially falls as antibody from the mother is used up to reach the lowest level at 2 to 3 months of age and then progressively rises as the infant generates its own antibodies. In SIDS the number of cases rises to a peak at 2 to 3 months and then falls and therefore it is tempting to suggest that death is in some way caused by a lack of protection against infection. A mathematical model shows that the rise and fall in the number of cases of SIDS in the first year of life can be explained by infection but only if the causative organism or organisms are very common. The organisms would need to be so common that 50% of the population would meet the organisms in any 50 day period.

As soon as a baby is born it becomes colonised by a wide range of common bacteria. These organisms are found on the skin surface, in the nose and throat and in the gut. Many of the bacteria are harmless but some are capable of invading the body and causing disease. A number produce toxic chemicals (toxins) that can enter the body and cause sudden death. Antibodies combine with bacteria and their toxins and neutralise their harmful effects and therefore infants will be most at risk when antibody levels are low.

These facts lead to the common bacterial toxin hypothesis of SIDS. The idea is that toxins produced by bacteria of the normal body flora enter the blood stream and precipitate sudden death. The risk of this occurring rises to a peak at 2 to 3 months and then falls as infant antibody levels change. Viral upper respiratory tract infections (common colds) do not cause death but they can cause a change in the bacterial flora of the nose and nasopharynx (the space behind the nose) and this can increase bacterial overgrowth and toxin production and increase the risk of sudden death.

Evidence is presented that SIDS infants have an abnormal nasopharyngeal bacterial flora with increased carriage of a number of bacteria including Staphylococcus aureus (Staph aureus) and Escherichia coli (E coli). Toxins from these bacteria interact synergistically (their combined action multiplies rather than adds) to cause death in animal models. The toxins also interact synergistically with tiny doses of nicotine, at levels found in passive smoking, to cause death. Secretions pool in the nasopharynx when infants sleep prone (on their front)

and this leads to increased bacterial growth. The combination of prone sleeping and a common cold leads to marked pooling of secretions and produces a bacterial flora similar to that found in SIDS. Staphylococcal toxins have been found in the brain and tissues of over 50% of SIDS infants and this is strong support for the hypothesis.

Further progress will depend on using modern and sophisticated methods of analysis to search for bacterial genes and bacterial toxins in fluid and tissue. Samples for microbiological investigation must be obtained as soon as death is ascertained; they should be analysed locally using standard methods but also submitted to regional and national laboratories for more specialised tests.

Ethnicity, infection and SIDS

Caroline Blackwell, Australia

Among Indigenous groups in which the incidence of SIDS is high, there are also high incidences of serious respiratory and ear infections as well as meningitis. These include many Indigenous groups in North America, Aboriginal Australians and the Maori of New Zealand. We compared risk factors among ethnic groups in which there were medium, low and high incidences of SIDS: British Caucasians; Bangladeshi families in Britain; and Aboriginal Australians [1]. The effects of these risk factors on susceptibility of babies to infections was then assessed.

Risk Factor	Caucasian European	Bangladeshi	AboriginalAustralian
SIDS / 1000 live births	2	0.3	6.1
Prone sleeping	+	-	-
Mothers who smoke (%)	25	3	75
IgG levels at birth	+	++	++
Bed Sharing	+	+++	+++
Switch to circadian rhythm (age in weeks)	8-16	12-20	?
Breast feeding	+	+++	+++
Bacterial colonisation	+	?	+++
IL-10 "low responders"	+	+++	+++

-, +, ++, +++ = rare to common, ? = not known

How do babies acquire potentially dangerous bacteria? Indigenous babies "pick up" dangerous bacteria earlier and in greater numbers [4,5,6]. This could be due to genetic or developmental differences in substances on the surface of the nose or throat to which bacteria stick, exposure to cigarette smoke or closer physical contact with family members. Smokers are more likely to harbour potentially dangerous bacteria and to carry greater numbers in their airways. Mother's smoking in combination with bed sharing increases the risk of SIDS; close prolonged contact like bed sharing could increase opportunities to pass bacteria from mother to baby. The means by which smoking could affect the way babies acquire bacteria are outlined in the accompanying abstract. Prone sleeping, virus infections and "stuffy" noses could also increase the temperature in the airways of babies and allow some bacteria to switch on powerful toxins; we have found these toxins in over half of SIDS babies from 5 different countries.

Do the risk factors affect the body's protective responses against infection? The body's natural internal defences against bacteria or viruses play an important part in controlling disease. These defences are produced

by white cells in the blood and are called the inflammatory responses. An example of local inflammatory responses is an infected cut that becomes hot, red, swollen and painful. These reactions can occur on a large scale throughout the body. There are two branches of this system; 1) **pro-responses,** messenger proteins that kill bacteria and viruses; 2) **anti-responses**, messenger proteins that switch off pro-responses when they have killed the bacteria. If pro-responses are not controlled, they can cause death (too much of a good thing).

There is evidence that some pro-responses are involved in SIDS. There are 4 ways in which the pro-responses are controlled: 1) antibodies that mop up bacteria, viruses or toxins; 2) hormones that damp down group 1 responses; 3) genetic factors that limit the levels of pro- and anti-responses; 4) environmental factors that can turn up pro-responses or turn down anti-responses. Total antibody levels high in Indigenous infants and remain so during the first year compared with babies of European origin; but there might be "holes" in these defences, absent or low levels of specific antibodies to particular germs or toxins. Asian babies stay for a longer period of time in an "immature" developmental stage in which night time levels of hormones that damp down inflammation remain high and could reduce the risk of SIDS which occurs predominantly at night. . We found significant differences for genes for both pro- and anti-responses among the three ethnic groups tested. Cigarette smoke had variable effects on pro-responses. Cigarette smoke consistently reduced an important anti-response, regardless of the genetic makeup of the person. The greatest effect of cigarette smoke was seen for gene variants that are predominant among both Bangladeshi and Aboriginal Australians. If the genetic components were the most important factors, the risk of serious infections and SIDS should be the same in these two groups. The low level of maternal smoking among Bangladeshis (3%) compared with the high proportion of smokers among Aboriginal Australian women (75%) indicate that the key factor is exposure of babies to cigarette smoke. This idea is supported by differences in the incidence of SIDS between groups of Indigenous Americans that reflect high and low levels of maternal smoking.

July 5 Afternoon

Definition and Diagnostic criteria

Sudden infant death syndrome (SIDS) and unclassified sudden infant deaths (USID): a definitional and diagnostic approach

Henry F. Krous, United States

INTRODUCTION

Beckwith proposed the original definition of SIDS in Seattle 1969 as "the sudden death of any infant or young child which is unexpected by history, and in which a thorough post-mortem examination fails to demonstrate an adequate cause of death." Twenty years later, it was redefined by an expert panel convened by the National Institute of Child Health and Human Development in the USA as "the sudden death of an infant under one year of age, which remains unexplained after a thorough case investigation, including performance of a complete autopsy, examination of the death scene, and review of the clinical history." The second definition of SIDS differs from the original definition by inclusion of the death scene investigation and limitation of the diagnosis to infants younger than one year of age.

Given the steady accumulation of new information about its epidemiology, risk factors, circumstances of death, pathology, and the importance of several additional testing, refinement of the SIDS definition is overdue. Therefore an expert panel of pediatric and forensic pathologists and pediatricians deliberated these issues in San Diego, CA in January 2004. Drs. J. Bruce Beckwith (USA), Roger W. Byard (Australia), Torleiv O. Rognum (Norway), Thomas Bajanowski (Germany), Tracey Corey (USA), Ernest Cutz (Canada), Randy Hanzlick (USA), Thomas G. Keens (USA), Edwin A. Mitchell (New Zealand), and Henry F. Krous (USA, chair) comprised the panel.

After review of the original and second definitions, a variety of topics were presented including the need for redefinition with its uneasy position between the health and legal systems, possible placement of risk factors in a new definition, the importance of clear definitions of study populations for research, the frequency of risk factors and the changes that had occurred since the "Back to Sleep" campaigns, the advantages and disadvantages of reporting on the death certificate those risk factors that may have been operative at the incident site in causing or contributing to death and the use of the terms "undetermined" and "unascertained" in flagging cases where significant parts of the investigation were lacking, or where there were doubts as to possible causes of death.

Following these presentations, vigorous discussions ensued and a definitional schema for SIDS and other cases of sudden infant death was agreed upon. It included a general definition for purposes of death certification and vital statistics, but recognizing its inherent limitations for research, subsets were also established. The new general definition of SIDS is intended to be more inclusive. The use of subsets of cases of SIDS is intended to facilitate tracking of changes of epidemiological patterns, especially with respect to monitoring the effects of public health recommendations and alterations to infant care practices, at national and international levels. Finally, more precise definition of subsets of sudden infant death, with specification of requirements for diagnosis, should assist in the standardization of protocols to improve the evaluation of the circumstances of death and autopsy examinations and thereby bring investigations more in line with recommended guidelines.

PROPOSED NEW DEFINITIONS

The panel has proposed the following definitions and criteria for subsets of SIDS and other cases of sudden infant death:

SIDS - General Definition

The sudden and unexpected death of an infant under 1 year of age, with onset of the lethal episode apparently occurring during sleep, that remains unexplained after a thorough investigation including performance of a complete autopsy, and review of the circumstances of death and the clinical history.

Category IA SIDS - Classical Features Of SIDS Present And Completely Documented

An infant death that meets the requirements of the general definition and also all of the following:

Clinical:

Older than 21 days and under 9 months;
A normal clinical history, including full term pregnancy (≥37 weeks gestational age);
Normal growth and development;
No similar deaths in siblings, close genetic relatives (uncles, aunts and 1st degree cousins), or other infants in the custody of the same caregiver;

Circumstances of death:

Investigation of the various scenes where incidents leading to death may have occurred, and determination that they do not provide an explanation for the cause of death;
Found in a safe sleeping environment with no evidence of accidental death;

Autopsy:

Absence of potentially lethal pathological findings. Minor respiratory system inflammatory infiltrates are acceptable. Intra-thoracic petechial hemorrhages are a supportive but not an obligatory or diagnostic finding.
No evidence of unexplained trauma, abuse, neglect or unintentional injury;
No evidence of substantial thymic stress effect (thymic weight less than 15 gms, and/or moderate to severe cortical lymphocyte depletion). Occasional "starry sky" macrophages or minor cortical depletion are acceptable.
Toxicology, microbiology, radiology studies, vitreous chemistry and metabolic screening studies are negative.

Category IB SIDS – Classical Features Of SIDS Present, But Incompletely Documented

An infant death that meets the requirements of the general definition and also meets all of the above criteria for Category IA except that:

Investigation of the various scenes where incidents leading to death may have occurred was not undertaken, and/or;

One or more of the following analyses was not performed: toxicology, microbiology, radiology, vitreous chemistry and metabolic screening.

Category II SIDS

An infant death that meets Category I criteria except for one or more of the following:

Clinical:

Age range - outside Category IA or IB, that is 0 to 21 days and 270 days (9 months) through first birthday;
Similar deaths of siblings, close relatives, or other infants in custody of same care giver that are not considered suspicious for infanticide or for recognized genetic disorders;
Neonatal and perinatal conditions (for example those resulting from preterm birth) that have resolved by the time of death;

Circumstances of death:

Mechanical asphyxia or suffocation caused by overlaying not determined with certainty

Autopsy:

Abnormal growth and development not thought to have contributed to death;
More marked inflammatory changes or abnormalities not sufficient to be unequivocal causes of death.

USID (Unclassified Sudden Infant Death)

Deaths not meeting the criteria for Category l or II SIDS, but where alternative diagnoses of natural or unnatural conditions are equivocal. This includes cases where autopsies have not been performed.

Post-Resuscitation Cases

Infants found in extremis who are resuscitated and later die ("temporarily interrupted SIDS") may be included in the above categories depending on the fulfillment of relevant criteria.

COMMENT

These proposals are intended to incorporate accumulated knowledge to tighten definitions, and to assist in the more accurate investigation, diagnosis, and categorization of cases of SIDS and other cases of sudden unexpected infant death. The proposed framework is recognized as a "work in progress" that will have to be continually reformulated and filled in as more knowledge becomes available and our understanding of these complex and challenging cases increases.

The complete manuscript prepared after the San Diego meeting will appear in the July 2004 issue of *Pediatrics*.

AKNOWLEDGEMENT

The CJ Foundation for SIDS provided the grant to undertake this work.

What can pathologists do to fight SIDS?

Torleiv O. Rognum, Norway

One main obstacle for the fight against SIDS is the different opinions among pathologists on what SIDS really is. In cases of sudden unexpected deaths in infancy pathologist have to find a cause of death. If they cannot find a cause, the diagnose is SIDS. However, pathologists have different opinion on possible causes of death. Whereas some experts think that modest inflammatory changes for instance in the heart muscle may cause death, others think that most of us have such changes from time to time without dying. If pathologists are going to contribute to the fight against SIDS, we have to agree on diagnostic criteria.

In Scandinavia, forensic pathologists have agreed on common diagnostic criteria for SIDS, and these criteria are used by all. We hope that an international agreement on common definition and common diagnostic criteria will help building a foundation for meaningful SIDS research.

To my opinion, perhaps 40 % of the today SIDS cases will in the future be explained by syndromes and diseases which we are only beginning to understand. The rest, more than half of the SIDS cases, I think can be understood by applying the three hit model:

1) Vulnerable developmental stage;

2) Genetic predisposition;

3) Trigger event: slight infection, prone sleeping position etc.

The Avon multi-agency approach to the investigation of sudden unexpected deaths in infancy and the care of bereaved families.

Peter Fleming, United Kingdom

The approach that has been adopted in Avon is based upon the practices developed in several countries, recommended in the report of the CESDI study, and endorsed by the Foundation for the Study of Infant Deaths in the UK..

As soon as possible after every sudden unexpected infant death a strategy discussion is held, involving the paediatrician, the police child protection team and the social services duty team. The purpose of the discussion is to plan how best to investigate the death and to support the family. This usually involves initial contact with the family in the Accident and Emergency department by the paediatrician and police officer, followed by a joint home visit, usually with the family doctor or health visitor. At these visits a full medical and social history is taken, with particular emphasis on recent events and a careful review of the circumstances and scene of the death. This information is passed to the pathologist, to ensure that appropriate and relevant post-mortem investigations are carried out.

Historically, the scene of the death of a baby was approached by police officers as a "scene of crime" and the same rules with regard to preservation of evidence were applied as at any suspected homicide. In developing the Avon Protocol, police Senior Investigating Officers were reassured that there was no risk of compromise of evidence for any potential criminal enquiry. Effective communication between all relevant professionals has allayed initial concerns. The attendance of a paediatrician together with a suitably qualified police officer at the death scene enhances the information gathering process for both agencies.

By visiting the home and seeing where the baby died, both the police and the paediatrician can gain further information, whilst the family are given the opportunity to talk through what happened in detail. Both police and paediatrician, in conjunction with the primary health care team, provide further support to the family.

Families have expressed great appreciation of this co-ordinated approach, recognising the need for police involvement, but feeling that the joint visits have been helpful rather than intrusive. Information on the post-mortem is fed back to the family as it becomes available.

Finally, 2-3 months after the death, a case discussion meeting is held, involving all professionals who were involved with the family. This gives an opportunity to review the classification of the death, identify any contributory or associated factors, debrief those involved in the care of the family, and to plan for continuing support and care of the family, including informing them of the assessment of the cause of the infant's death. At this meeting the "cause" of death is classified according to the Avon clinicopathological classification scheme. This grid system of classification ensures the collection and recording of information that is of importance and potential value to the family, and to each of the agencies involved in the care of the family and investigation of the death, and forms the basis for planning the future support and care of the family.

A joint approach, with medical staff working closely with the Police Child Protection Team, ensures that all necessary information is collected sensitively and promptly, without the need for bereaved families to repeat their stories to multiple agencies. The paediatricians and the Police Child Protection team have broad experience of normal childcare practices in this community, and are thus perhaps less likely to draw unwarranted conclusions about the contributions of particular patterns of child care to the death. The continuing involvement of the paediatricians in research into current child care practices within the community further helps to inform their interpretation of information obtained after infant deaths.

Australian protocol and definition meeting,

Roger W. Byard, Australia

Following recent meetings in Norway and the United States, a meeting of pathologists was held in Canberra, Australia to discuss the issues of standardising the definition of SIDS and the autopsy approach to unexpected infant deaths in Australia. The workshop lasted for two days and recommendations included the adoption of the proposed San Diego definition of SIDS for national use, and the formulation of an autopsy guideline/protocol for the uniform evaluation of these cases. The meeting demonstrated the usefulness of collaboration between forensic and pediatric pathologists and hopefully will provide guidelines that will improve the investigation of infant deaths.

July 6, Morning Session

Future Research Directions – Perspectives from the pathologist

Roger W. Byard, Australia

Diagnosing causes of sudden infant death is often a difficult process. Differences in the way that autopsies are performed and the use of different definitions for SIDS have led to considerable confusion. For example, the term SIDS has been used when the requirements of standard definitions have not been fulfilled. In an attempt to correct this situation, recent meetings of experts have occurred where steps have been taken to clarify the way in which cases are being classified and to institute protocols to provide frameworks for investigations. However, if research is to be meaningful, researchers must be extremely careful in assessing how extensively cases have been investigated and how closely cases fit with internationally recognised definitions and standards. Unless this approach is adopted, making sense of research findings in SIDS will be difficult and the literature will continue to be full of contradictions and unsupported conclusions.

Does the use of animal models assist our understanding of SIDS pathology?

Karen Waters, Australia

Studies in animals have been used to examine whether various clinical risk factors can induce death or other abnormalities that are typically associated with SIDS.

The advantages of animal models, is by using very controlled conditions for our studies, we can determine how the conditions associated with clinical abnormalities influence various organ systems, including the brain.

The strongest advantages of animal models are that they can be exposed to extreme conditions which could not be used in normal infants. As well as observing the changes that occur during life, tissue can be collected after death. Many of the conditions that are used for testing animals have been shown to occur with certain clinical conditions.

More recently, there has been additional interest in conditions that induce changes from which animals apparently show complete recovery, but where subsequent studies (in life or after death) have been able to show that damage had occurred in some organ systems.

The most obvious, and main disadvantage of using animals to study SIDS, is that there are many differences in the characteristics and the development of animals compared to human infants. Findings such as the risks associated with prone sleeping, cannot be determined from animal studies.

With all aspects of research, each research method has a number of benefits to offer. However, proper understanding of the results of these studies also requires that the audience are aware of the limitations that the particular methods impose.

Physiology of the human infant

André Kahn, Belgium

From the standpoint of physiology, SIDS research opens to a variety of fields of human physiology still poorly known. SIDS research includes new aspects such as the link between infant's physiology and new emerging risk factors and potential protective factors. More information is being collected on the characteristics of normal and deviant pre- and perinatal development. SIDS research could includes progresses in more classical study areas. Additional information is being collected on the physiology of vital control mechanism. Control mechanisms include such issues as the cardiocirculatory system; the respiratory system; the immune system; the autonomic controls; or the autoresuscitative control mechanisms. These control systems refer to the sleep and wake continuum, as well as other cardiorespiratory and autonomic responses to internal and external challenges. The infant responses and adaptation to both short-term and chronic stress is being evaluated. The contribution of the infant's emotional and sleep-wake status is progressively included in the description of normal physiology. Environmental factors in the child micro-environment that modify these mechanism are likewise taken into consideration in the explorations of infant physiology. The infant gestational age is an additional factor that is taken into consideration when exploring the physiology of very premature infants. The genetic basis of these aspects of developmental physiology and control mechanisms, are being collected.

The exploration of such complementary fields of human physiology requires adequate tools, and new ways to assess and measure subtle changes in the various research fields. For some, such as the waking system, definitions are being improved. Other research fields, such as the evaluation of the autonomic controls, lead to the definition of new and more precise non-invasive exploration tools.

These studies develop on the most efficient manner with the help of electronic communication tools, and common data-base accessible to all researchers world-wide. International research protocols likewise contribute to collect sufficient information. In addition to scientific journals and web sites, the new data are best debated and exchanged in scientific meetings such as the present one in Edmonton.

Perspectives from Epidemiology

Peter J Fleming and Peter S Blair, United Kingdom

The rapid decline in the SIDS rate has been brought about by investigating the place in which the infant last slept and it is perhaps further detailed scrutiny of this environment that will bring about a subsequent fall in deaths. Although there is a consensus of opinion that infant sleeping position and exposure to tobacco smoke are part of some causal chain there is little understanding of how this causality works whilst for other factors such as bed-sharing and pacifier use there is not much consensus at all. The traditional approach to epidemiological investigation of using randomised controls and building a multivariate model which mixes up infants sleeping in different environments and factors that have different effects on these environments seems to confuse and limit the interpretation of our results. Future SIDS studies are likely to include smaller numbers of infant deaths, and to include a larger proportion of deprived families and infants sharing the parental bed. If we are going to meet these changes and overcome our polarised debates we need to re-design future SIDS investigations.

The South West Infant Sleep Scene (SWISS) study is an epidemiological investigation of SIDS deaths including a death scene investigation of each case and a similarly detailed sleep scene investigation of the control infants. The study area represents approximately 10% of the English population with 60,000 livebirths per year and an anticipated 30 SUDI deaths. The study began in January 2003, initially for three years but hoping to extend for a further two years.

When a death is notified a multi-disciplinary team including a paediatrician, a trained representative of the police and a SWISS researcher contact the parents and an interview takes place within hours of the death. This interview takes place in the family home and includes a detailed, structured death scene investigation, together with bereavement support and information for the family.. The death-scene investigation includes a standardised video recording of where the infant died, a plan of the room including measurement and north-south orientation, temperature readings and measurement of mattress softness. A follow-up interview is then arranged to formally go through a detailed study questionnaire. A life-sized doll was especially built to be used in this interview but problems in terms of how the bereaved parents may react to such a doll and the practical aspect of how to carry such an item has meant abandoning such an idea, a doll-like toy in the room is sometimes used to demonstrate the position and covering of the infant.

Approximately 300 randomised control mothers are being contacted via their midwife during pregnancy and details of infant health and sleeping environment collected by postal questionnaire at 2 weeks, 2 months, 4 months and 8 months old. Contact prior to the birth of the infant means we can try and determine the maternal preference of infant sleeping environment. Of these, 100 are being chosen, weighted to include a high proportion of bed-sharers, to take part in a sleep-scene investigation carried out in exactly the same way as the cases. The mother is contacted shortly before interview and asked not to make any preparation or change any routines for the visit. The mother is interviewed about infant sleep practices in the previous 24 hours to interview, a sleep-scene investigation is conducted and the same questionnaire as used for the index mothers is administered by the researcher..

A further control group has been enlisted for this study consisting of families at " high risk" for SIDS. This was formulated from previous study data (CESDI study), constructing a multivariate model of pre-natal factors obtainable easily from hospital records and significantly associated with SIDS families. From our data this included 4 factors; young maternal age, high parity, maternal smoking and social deprivation. Using these 4 factors 300 "high risk" control families are being included of which 100 will be selected for a sleep-scene investigation. It was anticipated that the ascertainment rate for this group would be extremely low and the first 8 months of data collection has borne this out. Of 192 families contacted via their midwife,19 were excluded because of very early pregnancies and only 31 (18%) consented to take part of which 10 have since dropped out. If this were the randomised control group there would be an insurmountable problem in terms of selection bias but because the very group we are trying to ascertain are those who usually fail to take part in any study this is not so much a problem. One of the solutions which appears to be working is to entice the families with small monetary vouchers for baby shops.

In the first year of the study there were 31 SUDI deaths of which 15 were SIDS, 12 explained SUDI and 4 still to be determined. Of the 15 SIDS infants, 4 were co-sleeping in the parental bed and 3 on a sofa. Of the 12 explained SUDI, 6 were due to unrecognised infection and 3 due to metabolic disorders.

In previous epidemiological studies pathological findings have often been ignored as too non-specific and anecdotal because of the lack of a comparison group. The large proportion of explained SUDI deaths in this study will be used as a further control group and the pathology findings integrated with the epidemiological information. Nearly 50% of the SIDS were co-sleeping in deprived households but what aspects of deprivation were important or how exactly the co-sleeping environment put the infant at risk may be unclear when just observing the cases but might become clearer when comparing them to similar "high risk" bed-sharing controls.

We anticipate the final number of SIDS infants in the study will be small (N=70), nearly half of whom will be found co-sleeping in mainly deprived households. However, using this new study design, we hope to have control infants matched for age, sleeping environment and similar housing conditions.

History Section

2004 International conference on sudden infant death in Canada
Celebrating 30 years of Care, Education and Research

Aurore Côté, Canada

In July 2004, exactly 30 years after the Francis E. Camps International Symposium on Sudden and Unexpected Deaths in Infancy held in Toronto, participants are gathering at another International SIDS conference here in Canada. The scientific program of the 8th SIDS International Conference features a number of new themes in areas such as "translational biology" or "neurotransmitters," in addition to the usual recurring themes. Although we have learned a good deal about SIDS since the very first meeting held in Seattle in 1963, some of the early, apparently forgotten ideas have resurfaced. I thought it might be useful to present an historical perspective of the many themes discussed in the past that today, in 2004, are once again the focus of an international SIDS meeting.

It was after rereading the proceedings of the 1974 conference that I decided to revisit the old documents dealing with the topic of sudden infant death. In the 1980s, much before the identification of prone sleeping and various child care practices as risk factors for SIDS, I had read many of those texts. With the next perspectives in mind, I am impressed by the insights of investigators of the 1940s and 1950s. Often, the authors of the published case series were coroners or medical examiners working with pathologists. They put a lot of thinking into their publications, and their reviews of the then current literature are fascinating. The discussions that took place at the first few international meetings are of great interest as well.

I have therefore selected a few texts from the past and obtained permission to reproduce them in this document. These documents, found in the next section, speak more eloquently than any summary I could have put together. Because important documents may have been overlooked --I am limited by the fact that I read only French and English-- I invite all readers who know of significant documents of the past to share their knowledge with me at, or after, the meeting.

I would like to express my gratitude to Joanne Baird and Carole Pole of the medical library at the Montreal Children's Hospital. Their help was invaluable in locating many of the older documents. Special thanks also go to Robert G. Carpenter for his gift of the proceedings of the third international conference held in Cambridge. Special thanks are extended as well to the executive directors of the SIDS Foundation of Washington (Keri Wagenaar) and the Canadian Foundation for the Study of Infant Death (Geoff Richardson) for their help with historical documents.

International Conferences of the Past

The 8th SIDS International Conference hosted by Canada is not, in fact, the eight of a series of international meetings on the topic of SIDS. Before SIDS International was created as an organization, efforts were made to gather together scientists interested in sudden infant death. Indeed, Canada was the host of the fourth international meeting held exactly 30 years ago. But let us look at how it all started.

Seattle 1963

The first International SIDS conference took place in September 1963 in Seattle, in the United States under the chairmanship of Earl P. Benditt and Ralph J. Wedgwood.[1] The chairmen were from the departments of Pediatrics and Pathology at the University of Washington School of Medicine in Seattle. There were ten guest speakers, seven from the United States and three from England. Thirty additional participants came from the United States except for one from Canada, Dr Sydney Segal.[2]

After discussing such topics as virology, antobodies to milk, and cardiopulmonary reflexes, the scientists assembled realized that they knew little about the incidence and epidemiology of sudden death in infancy --not to mention the etiology, which still eludes us today. Dr Lester Adelson (of the Institute of Pathology at the Western Reserve University School of Medicine) commenting on the characteristics of Sudden Death Syndrome in the closing workshop of the conference, said: "My present inclination is to go home and try to digest

this tremendously rich diet of new and exotic foods and see what falls into place. I haven't had a chance to let things jell. I have heard a lot of new things today that I would like to think about."[3] In fact, all participants ask for more statistics pertaining to the different environments and races. At that point, the only available data came from case series that had pathologists performing autopsies. Clearly, the 1963 conference stimulated the production of more comprehensive and detailed studies.

Seattle 1969

The second international meeting took place in February 1969, again in Seattle.[4] The co-chairs, Drs. J. Bruce Beckwith, a pathologist, and Abraham B. Bergman, a pediatrician, were both with the Children's Orthopedic Hospital and Medical Center and the University of Washington, in Seattle. This time there were twenty-seven guest speakers from four countries (Canada, Czechoslovakia, Northern Ireland, and the United States), with additional observers from parents' and SIDS associations in the United States. Interestingly, the conference proceedings has an addendum describing the organizations in place for parents of victims of sudden death.

The purpose of the 1969 conference was, in the words of the organizers, to subject the work performed in the five years since the last conference to critical review and, again, to recommend future research priorities. The themes discussed were divided under four topics: epidemiology, pathology, virology and physiology. It was during the 1969 meeting that the name Sudden Infant Death Syndrome was adopted and a definition agreed upon.

In the addendum on national organizations, we learn of the National Foundation for Sudden Infant Death based in New York City (founded in 1962) which, in addition to providing services to families, had a medical board and allocated research money. This organism was to become SIDS Alliance. Mentioned, too, was the International Guild for Infant Survival (founded as the Guild for Infant Survival in 1964), a charitable and educational organization based in Maryland. Because of interest expressed in other countries and contacts in Australia, Canada, Indonesia, Israel, South Africa and Switzerland, the term "international guild" was felt to be justified. A local Seattle organization, the Washington Association for Sudden Infant Death Study, was also represented at the conference. Although officially launched in 1965, that group had parents who had been proactive since 1961 and had worked to have legislation adopted in 1963 that would refer cases of sudden deaths for investigation at the University of Washington.

Cambridge 1970

A short time later, in April 1970, the third international meeting was held in Cambridge, England, under the chairmanship of Sylvia Limerick. Francis E. Camp, a professor of Forensic Medicine at the London Hospital Medical College, and Robert G. Carpenter, from the Department of Medical Statistics and Epidemiology at the London School of Hygiene and Tropical Medicine, served as conference organizers.[5] The 30 guest speakers came for the most part from the United Kingdom, with the exception of the American professors Wedgwood (from Seattle) and Kempe (from Denver) in the United States, as well as Dr. Holy (from Charles University in Prague, Czechoslovakia). Two government officials were listed as observers. This meeting led to the creation of the Foundation for the Study of Infant Death in the UK.

Discussions focused on epidemiology, immunology --especially milk hypersensitivity-- respiratory reflexes, airways obstruction and inborn errors of metabolism.

It is interesting to note that the conference proceedings, eventually published in 1972, contained an addendum with information for the parents of victims of sudden unexpected death.[6] Apparently the first published document for parents, its language and tone closely resemble that still in use today.

Toronto 1974

The fourth international meeting was held in Toronto, Canada, in May 1974.[7] It followed the creation of the Canadian Foundation for the Study of Infant Death in 1973 and brought together 250 participants. Thirty-two guest speakers from six countries --Canada, England, Holland, New Zealand, Sweden and the United States—took part in the conference program.

The program was divided into four sections: pathology, epidemiology, developmental physiology

and human values. This was the first time that a section on the human aspects of SIDS was included in the program (a full day) at an international meeting on SIDS. Listed in the proceedings of the conference are the various SIDS-related national organizations based in Canada, England, New Zealand, and the United States.
Robert G. Carpenter, Ronald Harper and Shirley Tonkin --our guests in 2004-- presented at that conference as well. A highlight was the presentation of the noted Sheffield program by John L. Emery, a consultant pathologist in Sheffield, and Robert G. Carpenter of the London School of Hygiene and Tropical Medicine.

Baltimore 1982
The fifth international meeting was held in Baltimore in the United States in June 1982.[8] Ninety-four guest speakers from eight countries --Australia, Belgium, Canada, England, Italy, New Zealand, Northern Ireland and the United States—presented papers. Total attendance was nearly 300. The program included a session on "Evaluation of preventive approaches." Robert G. Carpenter presented on "The Sheffield program, a tool for prevention and evaluation," and Alfred Steinschneider presented on "The effectiveness of electronic home monitoring program in preventing SIDS."
Many of the research presenters at that conference are with us at the 2004 conference: Susan Beal, Robert G. Carpenter, James E. Fewell, Gabriel G. Haddad, Heather E. Jeffrey, André Kahn, Bradley T. Thach and Shirley Tonkin.

Bruxelles 1985
The next international meeting was held in Bruxelles in 1985. The story of the international meetings after 1982, aptly summarized by Kaarene Fitzerald in a recent book,[9] is reproduced here with permission of the publishers (please refer to page 139).

Recognition of the problem of sudden infant death

Several authors have already written at length about the reports from Antiquity and the Middle Ages concerning sudden unexpected deaths.[10,11] Although publications on sudden unexpected infant deaths were not numerous before the 20th century, the salient features of what was to become known as the Sudden Infant Death Syndrome were even then starting to be recognized. In his 1982 paper, Charles Templeman, Surgeon to the Royal Infirmary in Dundee, Scotland, described quite well the cases of healthy infants found dead in the morning.[13] It appears that all these infants were sharing a sleeping surface with at least one adult in the case series. The autopsy findings reported by Templeman are very closed to what we now know of SIDS victims with: "Frothy mucuous, often tinged with blood, is generally seen about the mouth and nostrils." "… a varying degree of congestion of the cerebral membranes--more or less engorgement of the internal organs, especially the lungs and kidneys. . ." "In about half the cases examined small punctiform haemorrhages were observed beneath the pleura and pericardium."[14]

A. Classification of sudden infant deaths

Starting in the 1930s, attempts were made to classify cases of sudden infant death. In 1938, pediatrician Alton Goldbloom and pathologist F.W. Wiglesworth, both with the Montreal Children's Hospital (then called the Children's Memorial Hospital), published what is probably the first Canadian paper on sudden infant death.[15] They classified the cases they had encountered (1933-1937) into three categories depending on the onset of symptoms (1 to 5 days, 24 hours, or negative previous history). In 1954, in a review paper, Dr A. M. Barret,[16] a pathologist from the United Kingdom, reported his practice of dividing sudden deaths into three categories according to the presence of symptoms (at least 12 hours, less than 12 hours, or none). He noted that the largest number of unexplained deaths, after investigation, were found in the last group (no symptoms prior to death). In the United States, Drs. Lester Adelson and Eleanor R. Kinney, from the Coroner's Office of Cuyahoga County and the Institute of Pathology at the Western Reserve University School of Medicine, were using a somewhat similar classification.
For the cases of infants with no previous symptoms, most authors suggested several possible diagnoses,

although they usually evoked infection or suffocation as a cause of death. After the discussion at the 1963 international meeting and the recognition that sudden infant deaths shared specific characteristics, there was general agreement that a special category had emerged with no established cause and no prior illness.
In the 1980s, two pathologists at the Hospital for Sick Children in Toronto, Canada (Drs. Ernest Cutz and Larry Becker), began using a subclassification. Dr. Bruce Beckwith of Seattle —the proposer of a new SIDS definition in 1992 at the second SIDS International meeting in Sydney—then proposed the use of three subclasses, categorised according to specific characteristics of the infants (age range, occurrence during sleep, family history), and pathological findings. However, the proposed classification was not accepted by the majority of the conference participants.
We have a session on the SIDS definition in 2004 and a revised version of that latter classification will be presented.

B. Causes of sudden infant death

In 1945, Paul V. Woolley, a pediatrician from the University of Oregon Medical School in the United States wrote an interesting review of the causes of sudden death,[17] citing the literature of the 1930s and 1940s. He reported infection (by bacteria or viruses), cardiac malformation and hypoglycemia from metabolic disorders as causes of sudden infant deaths. He concluded his paper with these words: "Complete autopsy examination with full recourse to modern bacteriologic technique should be demanded in every unexplained death of an infant."
Dr. Woolley is remembered by all SIDS historians as having done studies showing that infants cannot suffocate in ordinary bedclothes, nor even with their head totally covered. Although these studies were mentioned in only a brief paragraph in the 1945 paper, that little paragraph providing "evidence" that sudden infant death was not due to suffocation, seems to have had a profound impact on the medical and research community for decades after. However, a careful reading of his paper can teach us much more about his opinion on the subject of SIDS.
In a 1954 publication, Dr. Barrett (already cited for his review paper on sudden death) provided a detailed description of the causes of sudden death according to his classification mentioned on page 131. In the group of infants found dead in bed with no prior symptoms, he found congenital heart disorders in 9%, mechanical asphyxia in 13% and no explanation in 74%.

Defining the problem

Terminology

At the 1963 conference's opening session on causes of sudden death in infants ("Review of the problem") Dr Benjamin H. Landing, a pathologist at the Children's Hospital of Los Angeles in the United States addressed the problem of terminology: "Our clinicians call it *Sudden Death Syndrome*. Our virology department calls it *Sudden Unexpected Death*, which they abbreviate SUD. Our coroner's department calls it *Crib death*, and some of our British colleagues call it *Cot death*." [18]No doubt anyone knowledgable in the field of sudden unexpected death in infancy in 2004 can write something similar[19] For example, today we use the terms SIDS, SUDI, SUDS and unascertained sudden death, among others. Clearly, the problem of terminology started in the early days. This does not mean that nothing was achieved in the interim; it simply points to the difficulty of naming a condition that still has no identifiable cause.
We know that the participants at the 1963 conference decided, by consensus, to refer to the problem as *Sudden death syndrome*.[20] But it was not until after the 1969 conference that the term *Sudden Infant Death Syndrome* became widely, although not universally, used.

Definition

A tentative description was agreed upon at the first conference in Seatlle in 1963.[20]At the second International conference held in Seattle in 1969 the first "official" definition of SIDS was discussed and agreed upon.[21] Dr Bruce Beckwith, co-chair of the conference led the talks. The original discussions about the terminology to be used, the actual

definition and the constituents of a thorough investigation make very interesting reading (the discussion section is reproduced in the next section). The group agreed that the definition should be as general as possible, pending further research and more widespread recognition of the syndrome.

A second definition was agreed upon by a panel convened by the National Institute of Child Health and Human Development (NICHD) in the United States in 1989.[22] Although propositions to present two definitions or to have subclasses (as did the Canadian group) were advanced, it was agreed that a single definition would be more practical. Dr. Bruce Beckwith was a member of the panel of experts.

In 1992, this time at the Second SIDS International Meeting in Sydney, Australia, more discussions took place around the modifying of the 1989 definition. Panel chairman Dr. Beckwith again took the lead and suggested his definition with different subclasses (I, II, and III).[23] However, the majority of the participants were not ready for a change.

At the Stavanger SIDS International conference in 1994, another attempt was made to change the definition[24] and a "Stavanger" definition was even proposed.[25] However, the vote favoured returning to the 1969 definition. Although discussions about the SIDS definition have continued, the topic has been almost totally ignored at all ensuing SIDS International meetings.

Then in 2003, Dr. Beckwith published a notable paper in the *Archives of Pediatric and Adolescent Medicine* that proposed a change in definition and, again, the use of subclasses.[26] (Commentaries) He suggested that a relatively small ad hoc panel be appointed to review the 1989 definition currently in use. This led to another discussion about definition in a meeting chaired by Torleiv O. Rognum, professor at the Forensic Institute in Oslo, executive chairman of the European Society for the Study and Prevention of Infant Death, and organizer of the Oslo conference in June 2003. Again, Dr, Beckwith's proposed definition was discussed. This time it was agreed to hold a special meeting to finalize a revised definition. The meeting occurred in January 2004 under the chairmanship of Dr. Henry Krous of San Diego in the United States. A definition, one based on the 1992 definition originally proposed by Beckwith, was agreed upon and will be published in *Pediatrics* in July 2004. Note that the 2004 SIDS International meeting has scheduled a whole session devoted to the definition and diagnostic criteria for SIDS.

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Epidemiology

Incidence of SIDS

The first publications outlying mortality rates for sudden unexpected deaths in infancy started to appear in the mid 1960s.[27] They identified between two to three cases per 1000 live births. However, investigators realized that the rates in the United States were 2 to 4 times higher for African-Americans and Indigenous people, as compared to Causasians. These rates went practically unchanged for two decades after.

Relationship between SIDS and infant mortality rates

At the second Seattle meeting in 1969, Professor Joseph Houstek from Charles University (Prague, Czechoslovakia) presented the epidemiological aspects of Sudden Infant Death Syndrome in Middle Bohemia, a large Czechoslovakian territory.[28] His data covered the years 1952 to 1967. professor Houstek seems to have been the first scientist to correlate total infant mortality, and especially mortality rates excluding the neonatal period (which we now call postneonatal mortality), with SIDS. The higher was the postneonatal mortality, the higher was the SIDS rate.

In a preview of the Avon longitudinal study, to be presented at the 2004 meeting, Dr. Houstek had noted that "Since 1962, in the Middle Bohemian region, we have followed up systematically the question of morbidity, mortality, and general health in children from birth to fifteen years of age. In this region, … preventive and curative care are joined together"

First case-control studies of sudden infant deaths

Drs. Irene Garrow and Jacob Werne, from the Office of the Chief Medical Examiner in New York City and the Department of Pathology of St John's Long Island City and Flushings Hospitals (United States), wrote an interesting series of three papers in 1953 based on their 20 years' experience.[29,30,31] The classification of their cases was similar to that of Barret[16] but they went on to compare the histological findings between groups using an additional group of infants who had died immediately after violence. The authors made a critical point: "Only by study of such control material can one establish what is normal for this age group."

In 1965, Robert G. Carpenter, then at the Department of Human Ecology at the University of Cambridge (UK), and C.W. Shaddick from the Public Health Department at the London County Council published a study, conducted between 1958 and 1961,[32] that analyzed some sociological data in the light of three main hypotheses for sudden infant death (overwhelming infection, suffocation from soft bedding, and anaphylaxis from inhalation of cow's milk). Their methodology included a thorough questionnaire administered to the families of 170 sudden death victims and comparable questionnaires administered to the families of two live control infants as soon as possible after the corresponding death had occurred (301 controls recruited, 89% of intended sample). The control children were matched for age, sex, and area of residence, and time of year. The authors concluded: " The sociological data show that "cot death" is significantly associated with the use of a soft pillow, bottle-feeding, especially during the first two weeks of life, and symptoms of respiratory disease in the two weeks before death."

In 1971, Peter Froggatt, Margaret Lynas and G. Mackenzie from the Department of Social and Preventive Medicine at Queen's University in Belfast, Northern Ireland, published a study done between 1965 and 1967.[33] The objectives of that study were twofold: to describe the epidemiological aspects of sudden infant death, and to examine how the data collected relate to the main hypotheses of causation. Family interviews were performed on 163 consecutive sudden unexpected infant deaths (deaths that were sudden, unexpected and unexplained after a routine autopsy). There were three control groups. Control group A consisted of 950 infants chosen from a random sample of the 8,500 births in 1965 in Belfast. Control group B consisted of the chronologically next, like-sexed, surviving child born in Northern Ireland in the same administrative area to each of the interviewed 148 index cases. The other control group, "autopsy control," was used occasionally in comparisons and consisted of non-SIDS autopsy subjects aged 2 to 103 weeks.

The study identified an excess number of cases in twins, in socio-economically disadvantaged families, in crowded dwellings, among younger mothers in higher parities and, possibly, in premature infants. The study is also interesting because it provided data on usual and last sleeping positions. The paper, presented at the 1969 Seattle meeting,[34] concluded that there were too few infants sleeping on their stomach at the time to draw meaningful conclusions, there was a statistical difference between the position of the SIDS cases and the controls.

Many other studies followed, including the major national Institute of Child Health and Human Development (NICHD) study undertaken in the late 1970s

At the 2004 conference, we will learn more about the Avon longitudinal study and the German SIDS study, two major epideiology study

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Hypoxia and vulnerability

Vulnerability

In the first two Seattle conferences (1963 abd 1969), participants addressed the concept of vulnerability. In 1971, Peter Froggatt, then professor in the Department of Social and Preventive Medicine, Queens University, Belfast (Northern Ireland), wrote: "The characteristic age range is the most important factor, and there seems little doubt that these infants die because, while passing through this period of increased physiological vulnerability, some critical combination of extrinsic and intrinsic factors proves lethal"[33]

Other scientists have since slightly modified this original idea, creating among others the well-known Triple Risk model.[35]

Markers of hypoxia

Richard Naeye, a pathologist from the Milton S. Hershey Medical Center in Hershey (United States), seem to have been the first to write extensively about hypoxemia. He is remembered for his "markers of hypoxemia" in SIDS victims,[36] although some of these markers were dismissed in later studies.

In recent years, much emphasis has been placed on hypoxemia as indeed was always the case. In the 2004 program, apart from the symposium on Hypoxia, stressors and vulnerability, we have several sessions addressing this topic.

Airway Obstruction

Airway obstruction was one of the prominent theories advanced to explain sudden infant death in the 1940s and 1950s. Abramson's excellent paper, with its thorough review of the subject, was published in the *Journal of Pediatrics* in 1944.[37] However, earlier on, in the nineteenth century, Templeton[13] advanced the same hypothesis. Dr. Shirley Tonkin from Aukland, New Zealand, who was also an early promotor of the airway obstruction hypothesis factor in sudden infant death,[38] will be presenting on that topic again in 2004.

Recommendation for Public Health Education to decrease the risk of sudden death

In 1944, Dr Harold Abramson[37] published a study detailing what he then called "accidental mechanical suffocation." The study was carried out under the direction of a special Committee of Infant Mortality of the Medical Society of the County of New York. Abramson's cases were not matched to a control group and the study was later judged harshly in various SIDS review papers. The theory of suffocation was then more or less dismissed in the following decade. It is nevertheless striking today to read of the circumstances of death of the infants Abramson reported on, as well as his recommendations. Note how similar they are to those now used in our campaigns:

The crib and carriage: Greater attention should be paid to the preparation of the infant's crib and carriage.

1) No infant should ever sleep on a pillow, but always on a flat mattress.
2) Rubber sheets and undersheets should be spread smoothly and be sufficiently large to allow tight tucking beneath the mattress.
3) Quilted pads should be bound down or pinned down to prevent loosening.
4) Oversheets and blankets should be large enough to permit firm tucking beneath the sides and lower edge of the mattress, but at the same time not so tight as to prevent freedom of movement of the baby.
5) Unnecessary articles such as decorative pillows, add unattached blankets, strings or bars from which toys are dangled, and restraining straps which may slid up about the neck should be eliminated." [39]

Concluding Remarks

This very brief historical review has highlighted some of the work of past SIDS Investigators. Albeit small, this is a representative sample of the older research theories that today, as SIDS investigators, we are revisiting. I invite you to read the following historical texts to appreciate the insights of the various authors.

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Reproduced with permission from Arnold Publishers, In: *Sudden Infant Death Syndrome – Problems, Progress and Possibilities.* Eds. Byard & Krous, Arnold Publishers, London, 2001.

CHAPTER 21

The 'Reduce the Risks' Campaign, SIDS International, The Global Strategy Task Force and The European Society for the Study and Prevention of Infant Death

Kaarene Fitzgerald

SIDS groups and organizations

The development of international networks of clinicians, researchers, health professionals, bereaved parents and others interested in reducing SIDS and infant mortality has been and is continually evolving. There is no doubt that the highly motivated people who have worked together in groups and cooperatively are responsible for major changes in infant care practices leading to one of the largest drops in mortality recorded in modern times. This drop has been achieved with little or no financial support from governments in many countries. How did this wide-ranging group of advocates from disparate communities and backgrounds achieve so much with so little?

On 18th October 1985 a group of SIDS parents from eight countries, Belgium, Denmark, France, Germany Great Britain, Holland, Luxembourg and the United States, met at the University of Brussels following an international workshop organized by Professor André Kahn called 'The SIDS practical management of infants at high risk'. The group agreed to stay in touch and work towards another meeting.

The next meeting was held on 27th May 1987 at Lake Como, Italy following the New York Academy of Sciences meeting. This time representatives from thirteen countries, Australia, Austria, Great Britain, Canada, Denmark, France, Germany, Italy, New Zealand, Norway, Scotland, Sweden and the United States, met with the outcome being the formation of SIDS Family International (SIDSFI). The aims were to facilitate the sharing of information, improve services to families, increase awareness about SIDS, improve liaison with health professionals and encourage research activities.

Recommendations were developed covering the areas of autopsies, emergency responders, recruiting new national organizations, parents working with researchers, notification to SIDS organizations, non-SIDS bereaved parents and bereaved parent contacts. Agreement was reached to pursue the adoption of recommendations in each of the participating countries.

As each country presented, statistics were collected on populations, and information on when groups first started, the average number of SIDS cases occurring, whether autopsies were performed or not, and the incidence of SIDS per thousand live births. This first summary covered 20 countries. Since then, updates have been regularly prepared and circulated with the latest version, covering 35 countries, included in this chapter.

A further meeting of SIDSFI was held on 6th April 1989 in London, UK, this time with 15 countries: Australia, Austria, Great Britain, Canada, Chile, Denmark, Greece, Ireland, Italy, New Zealand, Netherlands, Norway, Scotland, Sweden and the USA. Topics for international support included using the term ‚apparent life-threatening event' (ALTE) rather than 'near-miss cot death', the use of publications from other countries, and development of a newsletter with the first editor, Felicity Price, coming from New Zealand.

The two most important topics were about the development of a more format system for international conferences including future venues and dates and a decision to advocate with the World Health Organization (WHO) to include information on SIDS in policy documents, especially 'Targets for Health For All'.

As part of the strategy to work towards achieving SIDSFI aims and increase communication it had been agreed to organize an international conference which followed the business meeting in London on 7-8th April 1989. About 350 delegates, who were primarily bereaved parents attended.
Meetings were subsequently held with WHO in Geneva and Copenhagen and it was agreed to work towards SIDSFI developing 'Official Relations' status as a non-government organization. Despite a great deal of effort from each of the SIDSFI Chairmen, including major submissions and articles for publication, at the time of writing this has not been achieved.

In 1990 WHO did agree to adopt a 'priority issue on the subject of SIDS' and acknowledged it was 'one of the major causes of infant mortality in the industrialized countries of the world'. (However any further major activity between WHO, SIDSI and the GSTF would not occur until the SIDSI conférence was held during 1998 in Rouen, France.)

A new group called the European Society for the Study and Prevention of Infant Deaths (ESPID), under the chairmanship of Professor André Kahn, held their Founding Congress 5 7th June 1991 in Rouen, France. It was at this meeting that the New Zealand research contingent very bravely presented the first posters opening robust discussions on the role of sleeping position.
Hazel Brooke from Scotland, the Secretary of SIDSFI, had also obtained funding to develop SIDS Europe and was coordinating efforts to support emerging interest and activities in Eastern Europe.
By the time the next SIDSFI conférence was held in Sydney, Australia in February 1992, about 28 national SIDS organizations were part of the network. At the business meeting preceding the conference it was decided to alter the name to SIDS International (SIDSI) to more clearly reflect the broadened activities. In addition, a register of SIDS researchers was commenced.
The Sydney conférence was targeted at researchers, clinicians, health professionals and bereaved parents with over 450 delegates from 18 countries attending. One of the major loci was the emerging research activity and health promotion programs looking at safe childcare practices and ways to reduce the risks of SIDS.
During informal discussions with researchers and health professionals whilst developing the Sydney conférence program, it had become apparent that efforts to advance scientific activity could possibly be enhanced by a more formalized networking process.
The concept of a Global Strategy Task Force (GSTF) was then developed by Kaarene Fitzgerald. Professors Adrian Walker and Caroline McMillen provided invaluable assistance preparing a draft scientific outline for initial discussions with Dr Marian Willinger from the National Institute of Child Health and Human Development (USA) and Dr Marsden Wagner from the Copenhagen office of WHO. Both of those organizations then agreed to support the new group.
The first Global Strategy Meeting (GSM) was held following the Sydney conference on 17-18th February 1992 with 70 representatives from 16 countries divided into four workshop groups: International SIDS Diagnosis, Epidemiology and Risk Factors, Physiology, Pathophysiology, and Predictive Tests, and Community and Organization Issues.
Each group met for a day and a half to identify issues that could be addressed through international collaboration, and to identify gaps in knowledge or resources. On the last day four international working groups with multidisciplinary participation were formed covering pathology, epidemiology, developmental physiology and education.
There was an enormous amount of energy in those early days. It was exciting, confronting but also salutary working on one of the major medical mysteries why should these babies die so suddenly and, apparently, without warning?
Theories and hypotheses abounded with, at any one time, 1 00 or more sensible sounding ideas. Anyone could obtain coverage no matter how far-fetched his or her idea might be. Parents were confused and frightened, the media were keen to publish articles and individual research teams were passionate about their project providing the answer.

SIDS organizations at a local level were trying to develop sensible answers and stay calm whilst perhaps looking after the families of six or more deaths in a day. And, at that time, it was known that about 30,000 infants were dying from SIDS in Western countries. Who was right? Would a drop in deaths attributed to SIDS ever occur?

In the meantime, through publication of the results of several USA research projects (subsequently round to be seriously flawed), apnea monitoring had become popular. Many saw the use of monitors as the only way to 'prevent' SIDS and thriving businesses commenced. In some countries the use of monitors was described as 'a risk reduction program' and large campaigns were mounted to have 'high-risk infants home monitored. Infant mortality did not fall significantly in most centers where these programs operated.

However, other researchers and clinicians who were visiting bereaved families, often within hours of the baby's death, realized common stories were emerging. Questionnaires were designed and case-control studies implemented. Another project looked at signs of illness in babies in the community. Stimulating papers were given on these subjects at the 1989 London conférence.

Research papers had been or were being published looking at the baby's environment including overheating and sleeping position. Drs M. Lee and D. Davies from Hong Kong had written a Letter to the Editor of *the Lancet* (1988) questioning the safety of prone sleeping and raised enormous interest. Susan Beal from Australia had also received significant publicity for her paper (1988). In the Netherlands, Dr Guus de Jonge and Dr Adele Engelberts were also gaining significant publicity and publishing relevant articles (1989).

More sophisticated questionnaires, particularly through the Avon Study (UK) led by Professor Peter Fleming, the NZ Cot Death Study with Professor Ed Mitchell; and the Tasmanian Prospective Cohort (Australia) with Professor Terry Dwyer, were designed and implemented.

SIDS organizations were also developing brochures and articles for parents and health professionals. It is believed the first 'reduce the risk/back to sleep' publication in English was produced in May 1987 by Stephanie Cowan from the Canterbury Cot Death Society in New Zealand, called 'Cot death: you cannot predict it, you cannot prevent it, but you can reduce risks'.

The first policy change recorded for health professionals was in July 1990 when Community Services Victoria (Australia) informed all their maternal and child health nurses to advise parents to place their babies on their sides or backs to sleep and to avoid overheating. One year later (July 1991) a 12 page color brochure in English and in 10 other languages was produced and distributed by the SIDS organization, incorporating the latest research and information from the Baby Illness Research Project (later to become known as Baby Check).

As a result of the 1992 Sydney Australia conférence presentations by many researchers, discussions at the SIDSI meeting, and discussants' papers at the GSM, 'Reduce the Risk/Back-to-Sleep' campaigns commenced over the next few months and years in many countries. Further discussions, papers and progress to reduce infant mortality have been the focus at subsequent SIDSI meetings, conferences and GSMs held in Stavanger, Norway 1994, Washington DC, USA 1996, Rouen, France 1998, Auckland, New Zealand 2000. Future meetings will be held in Florence, Italy March 2002, Edmonton, Canada 2004 and hopefully in Japan in 2006.

Reports from SIDSI and GSM meetings and GSTF projects including the International Autopsy and Event Scene Protocols, Core Curriculum (for health professionals), details about Project RIMI (Representation Minorities and Indigenous People), International Child Care Practice study, Standardized Mortality Reporting, position statements on smoking, and other issues, minimum standards for physiological, recording, state of the art reviews, WHO, International Statistics are available on www.sidsinternational.minerva.com.au or www.sidsglobal.org.

Due to the energy and commitment by many, at this stage a significant number of countries have seen a drop of between 50-80 per cent in infant mortality. Many parents have not had to deal with the tragedy of losing a child and the world has gained many thousands of citizens who will contribute in turn to its health and well-being.

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A Foreword

The Countess of Limerick*

My only excuse for accepting an invitation to speak this morning is that I regard this Francis E. Camps International Symposium as the successor to those symposia held in 1963, 1969 and 1970, and myself, as chairman of the last, I regard as the bearer of the Olympic torch. Just as Professor Wedgwood came hotfoot from Seattle to the symposium in Cambridge, UK, which, thanks largely to the generosity of Sir Max Aitken, resulted in the United Kingdom Foundation's being set up, so I now pass on the flame which kindled such interest in the United States and Britain to Dr. Harry Bain (chairman of this symposium) and to the newly founded Canadian Foundation. The theme of this symposium is "to associate together those who have specially studied the subject with experts in other fields and disciplines who have a limited knowledge of the problem".

I think it is most important to put the problem in its proper perspective and recognize its importance throughout the world. For instance, in the 67 year span of Francis Camps' life, total infant mortality rates fell dramatically - in the United Kingdom from 117 deaths pet 1,000 live births in 1905 to 17.5 in 1971 - but when these total figures are broken down the recent decline in the post neo-natal age group is much less than in the peri-natal or neo-natal group; and the most common manner of death in those aged between one month and one year is the sudden and unexpected one.

It is thus now a prominent and urgent medical problem; but it is much more than that. It is a problem which bas profound emotional and social consequences for the bereaved parents, and thus deserves the attention of all who care for those with whom we share this world. This aspect of the problem was poignantly highlighted by Mayor Crombie this morning.

It is not my task to outline what bas been achieved, as Dr. Eileen Hasselmeyer, I'm delighted to say, will be overviewing the research in a few minutes, but I would just like to comment on a few developments which are evidence of the value of symposia such as this.

In 1963 it was apparent that more comprehensive and detailed information on every aspect of the problem was required and this stimulated some very thorough epidemiological research.

From the 1969 conference we are chiefly indebted to Dr. Beckwith for his definition of the problem, then styled Sudden Infant Death Syndrome (SIDS), "the sudden death of any infant or young child which is unexpected by history and in which a thorough postmortem fails to demonstrate an adequate cause of death". This is not a definition reached by applying criteria based on preconceived ideas of what constitutes SIDS, but is one reached by exclusion of recognized causes of death. Identification therefore depends, of course, on the skill and techniques available to the pathologist and his judgment in interpreting minimal data. It is however in the best Francis Camps tradition, for Francis Camps, who acquired an outstanding international reputation as a forensic pathologist guided himself by the principle that "it is safer to proceed by excluding all possibilities than by taking the short cut of accepting what is obvious".

This brings me to the full title of this symposium and gives me an opportunity to pay a warm tribute to the late Francis Camps, who, the more he dealt with mortality, became increasingly and passionately concerned with saving life. It was Francis Camps who together with Sir Samuel Bedson inspired the British Ministry of Health to investigate this problem in the 1950s; Francis Camps attended the 1963 Seattle conference and he together with Mr. Carpenter convened the 1970 symposium in the United Kingdom which was intended to collate information to date and activate research in the United Kingdom.

I believe it was Pericles, after the battle of Thermopylae, who reminded us that the mark of greatness is that people's influence should outlive their mortality. I think there is no more fitting memorial to Professor Camps than that we have taken up one of his cherished concerns, that this symposium is named after him and that it

has been organized by his son, Peter Camps, who, having inherited his father's enthusiasm, vitality and stamina, deserves our deepest gratitude.

Perhaps as chairman of the Cambridge symposium, I am entitled to quote a passage from my summing up in 1970: "Few of us expected to learn at this seminar of a single simple answer to this tragic problem ... It is debated whether we are dealing with several conditions grouped under one name or a condition in which a single mode of death may be sparked off by different underlying causes, or thirdly, a condition which is composed of multiple factors working in various combinations some of which prove fatal."

Having acquired the name I have, perhaps I may be forgiven for trying to sum up our conclusion in a verse of appropriate metre.

When theories compete in profusion,
Then the experts conclude, in confusion:
"There'll be flaws in all laws
Of this unexplained cause
Till the problem is solved by exclusion."

But on a serious note are we right to proceed in our approach by excluding all other causes of death? To illustrate this, sometimes parents say to me "the Coroner told me my child had pneumoniae, but be died suddenly and utterly unexpectedly and I think he was a case of sudden infant death." Such a distraught mother may be trying to excuse herself for having failed to recognize that her baby was ill but, on the other hand, she may be right. Perhaps sudden infant death is the underlying cause of many deaths which are assigned to a familiar cause because the child was known to be suffering from this other disease at the time. Should not our enquiries and investigations extend to all sudden unexpected infant deaths? I fear I may have only added to rather than clarified the confusion!

In turning to the immediate future there are just three things I would like to say about this present symposium.

1) First is the importance of the fact that it is taking place at all -- knowledge has reached a point in all countries represented here where it is vital to exchange ideas and co-ordinate planning of future research and to approach the problem in a multi-disciplinary way. If we believe there is more than one cause, it is essential to sort out the ones which are different from the others and so identify sub-groups. If we believe there is a common factor, should we not widen the category of deaths we investigate?

2) I am delighted that included in the excellent program is a whole day devoted to the study of the physiological development of normal infants, and that a session bas been set aside to consider the human aspects - if we cannot yet prevent these deaths, we can at least try to alleviate the suffering of the bereaved.

3) I hope opportunity will be taken at this gathering of so many representatives from the various foundations sponsoring research and welfare projects, to plan greater and swifter communication of scientific material and progress and evaluation of welfare projects and fund raising methods, both nationally and internationally.

Of two outcomes, I am convinced - unwittingly we shall model ourselves on the somewhat topical figure of the politician who may be defined as a person who approaches every problem with an open mouth; and those who fail to get a word in edgewise should content themselves with Mark Twain's consoling thought: "There is no substitute for brains, but a man may achieve a similar reputation by keeping his mouth shut." Taking this hint, I shall now conclude, while wishing the symposium successful progress, which will enable Dr. Bain to present a much clearer picture in his concluding remarks on Friday.

**The Countess of Limerick, M.A. is a founding Member and Vice-Chairman of the United Kingdom Foundation for the Study of infant Deaths, and Chairman of the Sir Samuel Bedson Symposium held at Addenbrookes Hospital, Cambridge, England in April 1970.*

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Peter Froggatt

Dr. Steinschneider and Lady Sylvia Limerick, in their characteristically generous greetings to the Conference participants from the sponsors, correctly identified the importance of previous research conferences, namely those held in Seattle in 1963 and 1969, in Cambridge (England) in 1970, and in Toronto in 1974. First Seattle was a seminal occasion: it brought a degree of cohesion and order into what was an under-researched and to some extent an intellectually anarchic situation. Views were expressed; ideas exchanged; data presented; and available theories, such as they were, catalogued and provisionally evaluated. This new knowledge did not of course spring like Athene full-formed from Zeus's head: fundamental work existed from before World War II, and most especially from the 1950s, by pathologists, clinicians, and a group of immunologists who saw possibly lethal processes in certain feeding regimens. First Seattle in fact initialed what has proved to be virtually 20 years of uninterrupted and purposeful research. In the words of the editors, Ralph Wedgwood and Earl Benditt, the Conference "was pervaded by a stimulating spirit of enquiry . . . with a free exchange of ideas and much penetrating but friendly criticism" (2).

The contributors to First Seattle were all from the United States and Britain as was most, though certainly not all, of the comprehensive bibliography. A feature of these early days was the dominance of the English-speaking interest partly I believe because it was North America, Australia, and Britain which had experienced the greatest decline in infant mortality since 1900, and as this lethal tide receded so it uncovered this entity of sudden unexpected and unexplained death in infants which previously had lain unnoticed beneath the waves: personal tragedies but statistically unimportant. This does not explain the comparative disinterest of northern European countries - an interesting historical fact which I have not time to discuss but which to some extent (and with noble exceptions) exists to the present day. Another feature is the almost complete absence of surveys in Latin America and Mediterranean countries (except Israel), let alone in the Third World and Asia - a reminder that more numerous causes of infant death abound - though with many such countries now enjoying infant mortality rates of North American urban levels we can anticipate their joining the investigative fold. Scandinavia was early absent for another reason: with their post-neonatal death rates from infections causes tending to little more than zero, crib death - though it did occur - was rare, a phenomenon I will mention later.

Five-and-a-half years after First Seattle was Second Seattle - February 1969. Much progress had by then been made in many disciplines: hypotheses were formulated, new fields for study were introduced to stand along with the conventional ones, a "final common pathway of death" was adduced, the term "Sudden Infant Death Syndrome" was promulgated, and not just the unfailing optimists but those of a more cautious disposition departed feeling that a scientifically-based explanation of the tragic phenomenon was close at hand requiring only some thought, some more work and some more time to reveal it. There were of course sceptics and those of a more sombre caste of mind who could point to the bewildering proliferation of theories - never an omen of speedy resolution of any problem - but the mood of anticipation approached suppressed euphoria - or even unsuppressed euphoria in some cases - and helped buoy-up our spirits as we were buffetted by a brisk North-Wester in Puget Sound during the two-hour passage from Orcas Island in the San Juan's to Bellingham and dispersal. If the message of the First Seattle Conference was for more basic data, the achievement of the Second was to introduce coherence and systemisation into thought and facts and to demonstrate the need for researches into, particularly, cardio-pulmonary-laryngeal maturation processes and the nature and significance of immature responses. Few at Seattle in 1969 I think doubted that these labours would meet with anything but success.

The next decade was punctuated with exciting developments, much now under the whip of parent involvement and government concern. There was the fruitful meeting at Cambridge (England) in 1970, from

which sprang the United Kingdom Foundation for the Study of Infant Death - a sponsor of the present Conference; the series of workshops organised at NIH by the irrepressible Eileen Hasselmeyer; and the appropriately-named Francis E. Camps Symposium in Canada in 1974: but despite all the bustle and achievement the 1970s was nonetheless a decade of overall disappointment. In spite of much fundamental work of unquestioned authority, the breaking of new ground, the application of many fine minds and the best techniques, and the maintenance of a scientific rectitude and integrity in which all can feel proud, much of all this by persons here today, despite all this the overall perception of the condition still eluded our despairing grasp, while its cherished sequel - that is regimen for scientifically-based and socially acceptable prevention seemed increasingly an ever-remote goal if not an actual mirage. New trails were undoubtedly blazed in pulmonary and laryngeal development physiology; in cardiac conduction phenomena; in sophistication of epidemiological methods; and in maturation and their conception generally: but some are proving perhaps false trails and the relevance of others to sudden death in infants is still uncertain. The steady stream of fresh data seemed at times as much to confuse as to clarify. Some investigators of a more pragmatic caste of mind, frustrated by events, and despairing at the lack of progress towards a coherent aetiology, turned to history for its lessons and drew heart from what they learned, viz. deaths from many causes and at many ages had been reduced before science had uncovered the aetiology let alone devised any specific prophylaxis. Tuberculosis and scarlet fever are examples of diseases reduced by concomitant but coincidental environmental improvements, and cholera an example of a disease reduced by empirical action, and all before Koch ever saw a tubercle bacillus, Pettenkofer swallowed his swarm of cholera vibrios, or Hansen identified the lepra bacillus, or indeed before Pasteur had formulated the basis of bacterial action. Our own century has seen something like a tenfold fall in infant mortality rates in Britain, parts of Europe, and in the United States, much of it without recourse to specific but only coincidental action. Even some non-communicable diseases can be empirically reduced, whether those which fill the gap of the disappearing zymotic diseases or those actually produced by the changes in environmental or personal circumstances which themselves brought about the fall in the quantum of infection-disease deaths - like dietary related or urban sedentary conditions. Could not this happen with crib death? Could there be for researchers a remarkable irony in that by the time the aetiology and pathogenesis of those components constituting the crib entity are understood, empiric action or coincidental environmental changes will have of themselves produced its elimination or reduction to trivial levels? The work in Sheffield, England, provides some evidence for active empiric reduction; the results of incidence-monitoring in many advanced societies over the past decade and the seeming rarity of cot death in Scandinavia and even Israel, which have very low levels of post-neonatal infections deaths, provide hope for coincidental reduction as enhanced medical and social resources are distributed across an ever widening spectrum of society. But hope only: the sober realm of fact may well be different.

However, if we would wish to learn from history we must learn the right lessons, and the right lesson is that empiricism is a fickle ally, coincidental improvements are random ones not usually within the profession's control. There is no certain substitute for scientific understanding and soundly-based preventive programmes. History also teaches us that the pervading scientific philosophy must be receptive to the aetiological facts: work has often lain ignored because the scientific climate was not intellectually prepared for its reception - the work of Gregor Mendel for example, or perhaps Fleming among numerous examples. The bacterial age strengthened the adherents of the unitary concept of causation, and the ideas of multicausality and partial attributability are still not generally or easily conceived. But without such perceptions crib death may remain a disease of theories with virologists seeing a virological cause; respiratory physiologists seeing respiratory problems; immunologists incriminating immunological aberrations; and cardiologists with eyes only for the heart. Periodic symposia or workshops, like this week, are essential if the lonely furrows which all plough are ever to meet. History teaches us that medical men do not take readily to the concept of "chance;" yet I have no doubt that "chance" is a crucial factor in crib death, not of course in the sense that crib death infants are a random selection of all infants - which they are not - but in the sense that an ordinary or "chance" hazard of life - an infection, an ectopic beat, a prolonged apnoea episode unobserved - may reach the child during a hazardous period of normal development and turn into a fatal event. Some ten years ago, to conceal my

ignorance, I wrote that crib death infants die "because while passing through a developmental stage of physiological vulnerability some critical combination of extrinsic and intrinsic factors occurs which proves lethal in 2 or 3 infants out of 1 000 live births"(1). I hope this Conference will show that such a specious formulation is no longer an adequate epitome of our knowledge.

This conference brings together many of the subject's leading researchers and is made possible by our generous friends and sponsors whom we thank. Few, if any, areas of medical research have been so favoured of public support and enthusiasm: it is no hyperbole to say that without these our work would have been much compromised. The Conference might even be seen by a grateful posterity as ending the frustration of the seventies. Psychologists tell us that frustration can arise either from no progress at all or, on the other hand, from being blocked from a final goal as one approaches it. Psychologists also tell us that the closer one is to that goal without reaching it, the greater the frustration. This Conference could show which genesis of frustration we manifest: we all hope that it is the latter, and that the goal is indeed close because it is a goal worth achieving and striving for. And if real progress here is made it would be a fitting tribute to the work, pioneer and contemporary, which has pushed Baltimore into the first rank of cities whose citizens and institutions have carved for their city and themselves an indelible niche in crib death research. And now, Ladies and Gentlemen, to business.

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DISCUSSION OF TERMINOLOGY AND DEFINITION OF THE SUDDEN INFANT DEATH SYNDROME

DR. BERGMAN: Before going further in this conference, it seems important that we make some attempt to agree on uniform terminology and a standard definition of the entity under consideration. Even if we do not all reach final agreement on these points, a discussion of differences cannot help but shed some light. I will call upon Dr. Beckwith to start.

TERMINOLOGY

DR. BECKWITH: I would like to lead off a discussion of *terminology*. Those of us in this room have applied a great many names to this entity. Yet the terms we use are sufficiently alike that one might hope that some sort of consensus might be achieved. The need for this is perhaps self-evident. It would be a good thing indeed if, in future publications, in naming of parent groups, and in discussing this with the lay press, the same name could be used.

A good title for this entity should be short and euphonious, sufficiently descriptive to prevent confusion with other types of sudden death, and readily comprehendible by lay persons.

Since the 1963 conference, we in Seattle have favored the term "Sudden Death Syndrome." This has the important virtue of communicating to the medical profession the concept that this is, in fact, a distinctive clinico-pathological entity. The word "syndrome" also combines well with modifiers to form a collective noun and, hence, provides a better name for a disease entity than, for example, "Sudden Unexpected Death in Infancy." The latter is a fine name for a paper, but not for a disease. These advantages, however, are at least partially negated by the fact that the word "syndrome" is not in the vocabulary of most lay persons. I personally feel the term "Sudden Death Syndrome" should at least be amplified to include the word "infant." The reason for this is that a number of "syndromes" of sudden death occur at different ages, and papers entitled "Sudden Death Syndrome" alone could refer to any of these. (An additional, but perhaps temporary objection to this term is that the initials "SDS" are at the present time popularly used for a militant student group.)

The popular term "Sudden Unexpected Death" has the important disadvantage of embracing unexpected death due to a variety of clearly defined diseases.

I should, therefore, like to cast my vote for the term "Sudden Infant Death Syndrome" (SIDS) as the best of many possible expressions for this entity.

DR. GARDNER: What is wrong with "crib death"?

DR. BECKWITH: I rather like the term "crib death." We are going to have to defer some of the arguments on this point until tomorrow, but our evidence suggests very strongly that this is a phenomenon related to sleep. We have not seen a single case in the category we term "Sudden Infant Death Syndrome" that bas not occurred in sleep, but other studies seem to be in opposition to us concerning this point. Therefore, I don't want to be the one to force the issue by advocating the use of the words "crib death."

DR. STEELE: There are just two points I would make. As one of the users of the abbreviation "SUD," let me say that this was done simply because this was the common usage in the particular area in which I live.

DR. GARDNER.' What do you mean by the letter "U"?

DR. STEELE' "Unexpected." I also think this is one of our responsibilities with regard to education of the

medical profession. I would be happy to change my usage and adopt the "Sudden Infant Death Syndrome." This is the first point I make.

The second one is that I think the use of "cot" and "crib" death implies that death always occurs only when infants are asleep and are horizontal. However, we have had cases where the infant was awake and being carried around in a supermarket, or was in a cart, when he suddenly expired. Therefore, I think there are certain disadvantages in using this particular expression.

DR. BECKWITH: Was the sudden expiration unexplained by a complete autopsy?

DR STEELE: Yes,

DR. PATRICK: I think there are children who die suddenly and unexpectedly for which we have rational reasons, and then we have some other children for which we have no good explanation.

DR. BERGMAN: Therefore, what is your preference?

DR. PATRICK: "Sudden Unexplained Death in Infancy," or "Sudden Unexplained Death." We have to relate it to infancy, because medical examiners find that there are adult people who die suddenly and unexpectedly without adequate explanation.

DR. MARSHALL: Insofar as my personal experience goes, some of these deaths are not entirely sudden, and whether they are expected or not depends on who might expect the death. They are certainly unexplained, and I would prefer the term "Unexplained Death in Infancy" and leave it there.

DR. BECKWITH: For some time I was in the habit of terming these cases "Sudden Unexplained Death Syndrome," and this term appeared frequently on the obituary pages. Unfortunately, the word "unexplained" led to important misconceptions on the part of families and acquaintances, who assumed that because death was "unexplained" that there was the possibility of foul play. The resultant problems were of such magnitude that we avoid the word entirely; there was too much confusion between the concepts of unexplained death, and death due to a clearly defined disease of unexplained cause. The word "unexplained" also has the disadvantage that what is unexplained in 1969 might not be unexplained in 1979.

DR. MILLER. I am wondering if the whole question of terminology is not, after all, going to have its biggest impact on us in trying to explain this to the lay individual. We need terminology that the layman can understand. Maybe we should go back to "crib death."

DR. WEDGWOOD: I believe there is value in having a single name for something we recognize as a common entity, if for no other reason than that one looks up a name in *Index Medicus* or in the index of a textbook and can find what he is looking for.

A "hang-up" with any descriptive name is that we tend to imply unintended meaning to the words used of which we are not sure. There are very few things we know for sure about SIDS other than that it happens in infants. I agree totally with Dr. Beckwith, that in 1969 we can no longer say that it is "unexplained." If the disease were called "King's Syndrome," nobody would be disturbed at all because it is a syndrome, and, surely, a syndrome means constellation, if you wish. If we put somebody's name on it, everybody will be happy. On the other hand, I think it is also important to use a single meaningful clue to reduce confusion in the literary and educational processes.

DR. BERGMAN: The objection that several of us have had to using the word "unexplained" is the aura of mystery, and the guilt that families suffer after their children die. I think this is compounded by the existing terminology, because "unexplained" means something different to physicians than it does to lay people.

I sense that the group is not ready at this point to decide on a single name. It will probably have to evolve through usage. Let's try for consensus on a uniform definition. Again, I will ask Dr. Beckwith to lead off.

DEFINITION

DR. BECKWITH: Of greater importance than uniform terminology is uniform definition. We have passed the point in time when "sudden unexpected death" can be considered an epidemiological entity. Sharper criteria must be applied. As Dr. Marshall and I shall bring out later in this conference, the Sudden Infant Death Syndrome is, at the present time, necessarily a diagnosis of *exclusion.* While absolute uniformity between

reported series is not possible because of inherent difference in the interpretation of lethality of given lesions between pathologists, we can at least attempt to agree on what steps should be taken to ensure that important definable causes of death have been excluded. Editorial boards reviewing manuscripts on SIDS should critically examine the criteria used for assignment of cases in "explained" and "unexplained" categories, and should no longer permit publication of series in which sudden unexpected death" is equated with "sudden unexplained death."

For purposes of discussion I should like to present the working definition we have adopted in the Seattle study. When examined critically, this definition is too brief and has a number of loopholes. However, an adequate definition would be so lengthy as to be ridiculous. Our definition goes as follows:

The sudden death of any infant or young child, which is unexpected by history, and in which a thorough post-mortem examination fails to demonstrate an adequate cause for death.

One of the problems, of course, is in the last clause. What is an adequate cause for death? What qualifications should one have in order to make this judgment?

It is unlikely that we can resolve the latter issue. We should, however, be able to agree on the central issue of what constitutes a minimum adequate investigation of a case. Obviously, a death in which the brain was not examined cannot confidently be said to be "unexplained." On the other hand, there are relatively few kinds of disease that can cause sudden unexpected death, and one need not insist upon a tremendously sophisticated work up before accepting a case. For example, I would not insist upon routine gamma globulin levels, viral isolation attempts, or plasma cortisol levels before accepting a case as SIDS. The following list sets forth my concept of a minimal acceptable work up:

PROPOSED MINIMAL ACCEPTABLE INVESTIGATION NECESSARY FOR A DIAGNOSIS OF SUDDEN INFANT DEATH SYNDROME

1. Adequate history.
2. Gross examination, *including* thorax, abdomen, brain, entire larynx, and spinal cord.
3. Blood culture.
4. Histological examination, *including:*

Brain — Liver
Heart — Kidneys
Lungs — Other organs as indicated by Nos. 1 and 2 above.

5. Ancillary studies (toxicological, chemical, special cultures, virological studies, and so forth) as indicated by results of above.
6. Counseling of family.

CRITERIA FOR INCLUSION

DR. GARDNER: Why do you include blood culture?

DR. BECKWITH: Blood culture is crucial only if one is going to define cases and explain death by a positive culture in the absence of other findings.

DR. PATRICK: This is one of the things we are going to get into a hassle about because the meaning of autopsy bacteriology is not clear in this particular group of children.

DR. GARDNER: So much depends on how long the body has lain from the time of death to autopsy.

DR. BERGMAN: The question came up earlier as to how each series of cases was studied. A fair number of cases will be presented during the next two days. We should resolve the question of what criteria are either acceptable or unacceptable in deciding whether a case can be diagnosed as SIDS.

DR. BECKWITH: The issue is, for example, whether one can accept as SIDS a case that bas had a complete

autopsy but not a blood culture.
DR. LANDING: Isn't the question, "I-low often the phenomenon happens?" In other words, whatever you may find, how often does it happen? Then you know whether it matters or not.
DR. STEINSCHNEIDER: It would seem to me that the fundamental issue of this discussion revolves around the explained, not the unexplained-the implications are that if you find an organism in the blood then you have explained the death. It seems, in terms of the proposed definition, that we are dealing with a diagnosis by exclusion.
What we must do is fail to demonstrate adequate explanation for death. If a given case is excluded from this category on the basis of a blood culture, the implication is we have explained the death; that we have made a positive statement, that we have said this organism is responsible for the death of this individual. This is how I read what is being proposed.
DR. WEDGWOOD: I think we can go at this another way. Suppose you are reading a paper on a hypothetical cause for SIDS and the author presents a series of patients, say five hundred, and lie has not done a single autopsy. Would you believe the conclusion from the paper? I think we would probably all say "No," and so an autopsy is a minimum criterion. Now, if lie had not done blood cultures would you be willing to accept the conclusions?
DR. GARDNER: No.
DR. WEDGWOOD: Well, if the answer is "No," then a minimum criterion for our interpretation of any study is culturing the blood. I believe that this is what Dr. Beckwith was trying to get at, and that interpretation of the blood culture is a secondary issue. If blood cultures have not been done, if autopsies have not been performed, and if histories have not been taken, then I don't think any of us would accept the results of that particular series for discussion.
DR. BECKWITH: This is the key point, because the same argument could be carried on when talking about the histological examination, or about looking, at the lung. We are not saying how we interpret it. However, I was trying to do exactly what Dr. Wedgwood said; that is, to define the criteria we would insist on before listening to an epidemiologist talk about his series.
DR. ROBBINS: I would suggest that we not require blood cultures for certain statistical analyses and also that we not accept positive blood culture as a clear explanation in every instance. I say this because I have seen babies in the hospital with positive blood cultures to whom we did nothing, and the next day they looked as rosy as could be. Of course, this doesn't mean that a positive blood culture cannot be the explanation of the disease, but it is almost as nebulous as a positive viral culture in certain instances. We are somewhat in the same boat in relation to doing viral cultures, because we don't always know how to interpret Our results.
DR. RAY: I don't know of any proof that bacteremia in the absence of gross or histological lesions is, in itself, lethal. I think we should have to say that bacteremia is not an adequate explanation for crib deaths.
DR. PATRICK: I think this is a perfectly reasonable position to take -that we decide here and now that in the absence of histologic lesion or any evidence of infection, children with positive blood cultures not be included in the syndrome.
DR. BERGMAN: Dr. Landing, what would it take for you to accept a case from, let us say, Dr. Patrick's laboratory or Dr. Marshall's laboratory? What type of minimum workup?
DR. LANDING: In general, I would accept an adequate gross autopsy and the histologic proposals Dr. Beckwith made.
DR. BERGMAN: Dr. Marshall, will you accept a case or a series of cases where blood cultures were riot performed?
DR. MARSHALL: At the moment I would, at least until I can be convinced that I am wrong.
DR. BERGMAN: How about the other criteria? Does anyone want to comment on the other minimum acceptable criteria for inclusion?
DR. DRAKE: You mention "counseling of family." Why was this included?
DR. BECKWITH: It probably isn't germane to this specific issue, but I could not help getting the pitch in there.

DR. BERGMAN: We might as well expand the pitch a little. When one visits the home and talks with families of crib death victims in order to determine the history of what happened, one cannot fail but become involved in counseling.
DR. FISHER: Do you just mean the medical history a day or two before, or are you going to trace the history of the child from birth and think of things that may have happened *in utero?* In other words, where do you start?
DR. BECKWITH: I mean a perceptive and experienced medical historian to get the circumstances of death and the circumstances preceding death. He should not be "taken in" by an inexperienced policeman or a parent or baby sitter who is concealing facts. I cannot define what an adequate historian is. I cannot define what an adequate history is, but I think an attempt above and beyond the ordinary police report ought to be made.
DR. FROGGATT: But you would use it again for exclusion?
DR. BECKWITH: Correct.
DR. PETERSON: It seems to me that we are still floundering epidemiologically, and perhaps we always will, and, further, that it is better to have a more flexible definition than a really tight one.
DR. BECKWITH: Is this adequately flexible?
DR. PETERSON: I would certainly agree that an autopsy would be a necessary requirement in order to rule out any sort of nefarious deeds. Also, you might be doing something by way of field inquiry. To me that would be sufficient.
DR. BERGMAN: Diagnosis in medicine is a phenomenon of relative probability, as is so well illustrated by Feinstein (28). It involves a series of factors. If enough factors intersect, perhaps a diagnosis will result. Rarely do physicians feel 100 per cent certainty in all their diagnoses. There is always the possibility of being wrong. However, the majority of cases of sudden infant death are not that difficult to diagnose.
The discussion tonight has been useful in opening up some of the problems of the studies to be presented tomorrow, because they all bear on the reliability of the collected data.

ADDENDUM: RECONSIDERATION BY PATHOLOGISTS

DR. LANDING: After the above discussion, some of the pathologists (Drs. Landing, Valdés-Dapena, Marshall, Patrick, Fisher, Drake, and Beckwith) met and reconsidered Dr. Beckwith's proposed minimum acceptable autopsy work up, and decided that some of the criteria he had suggested could be modified and still leave patients who were acceptable for epidemiologic purposes.
We felt that *gross examination,* including brain and larynx, was most important. The spinal cord, although it happens to be routine in our autopsy service, and we are not about to change it, is probably not that critical, and patients could be accepted as legitimate instances of the entity we are talking about even if their spinal cord had not been examined. The probability of finding a lethal lesion of the cord is very small. The same thing, applies to blood culture. We felt that blood or spleen cultures, or both, should be recommended but not required in terms of willingness to include patients in certain studies.
Dr. Beckwith had recommended brain, heart, lungs, liver, and kidneys as the minimum histologic work up, and other organs only if they appeared to be abnormal or abnormality was suggested by history. The group felt that this was acceptable and could not think of any other specific organs that would have to be studied histologically before one would be willing to accept a patient as a legitimate instance of the sudden death phenomenon.

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ROLE OF INFECTION, SUFFOCATION, AND BOTTLE-FEEDING IN COT-DEATH

AN ANALYSIS OF SOME FACTORS IN THE HISTORIES OF 110 CASES AND THEIR CONTROLS

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Infant mortality rates for England and Wales continue to decline. However, this does not hold good for sudden deaths reported to coroners, and the cause of a large proportion of these cases remains obscure (Banks, 1958).

Three hypothesis have been offered in explanation by Barret (1954): (i) an overwhelming infection, possibly of virus origin; (ii) suffocation from soft bedding; (iii) an anaphylactic type of reaction from inhalation of cow's milk by a milk sensitized child.

These hypotheses have been discussed with inconclusive results (Banks, 1958; Parish, Barrett, Coombs, Gunther, and Camps, 1960; Parish, Richards, France, and Coombs, 1964).

The purpose of this paper is to present the results of an analysis of some sociological data in the light of the three hypotheses.

MATERIAL

In twelve London Boroughs and in the Administrative County of Cambridge, deaths reported to the coroners in children aged 2 weeks to 2 years were investigated as described by Banks (1958). Between September, 1958, and May, 1961, 170 deaths were investigated, sociological inquiry forms being completed by coroner's officers and health visitors.

In addition, in each case, comparable questionnaires were completed by health visitors for two live control children as soon as possible after the corresponding death occurred. The method of selecting control children was to take the two nearest names on either side of that of the dead child in the Medical Officer of Health's Register, matching for sex and accepting only those children who were still living in the district. Thus the control children were matched for age, sex, and area of residence, and their clinical histories were matched for time of year. Controls wore not selected for cases which had been resident for less than one month at the address at which they wore taken ill or died and this, coupled with a few refusals, made only 301 controls available, representing 89 per cent of the intended sample.

CLASSIFICATION OF THE CASES

Some of the deaths resulted from recognizable causes, e.g. congenital malformations of the heart. Such deaths, although sudden, are not the object of this investigation, which is primarily concerned with those cases in which the information available do« not reveal or strongly suggest the true cause or causes of death. It is convenient to use the term "cot death" to describe such cases.

Pathological and bacteriological investigations have been carried out on some of these cases, but for others they are not complete. For the purpose of this study a provisional pathological classification was made by Dr R. W. Ainsworth on the data available. In 80 per cent of the cases this classification was based on the clinical history and macro*scopic post mortem* findings (Table I[1])

[1] Subsequent histological examination of lung sections of 33 cases provisionally classified as "cot death" revealed a cause of death in only 2 (6 per cent). In six out of the thirteen cases which were provisionally classified as probably due to pneumonia, tracheo-bronchitis, etc., the cause of death could not be demonstrated histologically on the material available. Hence the provisional classification shown in Table I almost certainly underestimates the proportion of "cot deaths".

TABLE I

CERTIFIED CAUSE OF DEATH AND THE PROVISIONAL CLASSIFICATION OF 170 CASES OF SUDDEN DEATH IN INFANCY

Certified cause of death			Respiratory*				Non-Respiratory				Total
Time in hospital before death (hrs)			Nil	<12	12+	Total	Nil	<12	12+	Total	
Provisional Classification	Cot deaths		107	2	-	109	1	-	-	1	110
	Other deaths	Respiratory	21	6	3	30	-	-	-	-	30
		Non-Respiratory	2	-	-	2	21	2	5	28	30
Total			130	8	3	141	22	2	5	29	170

*The following causes were regarded as respiratory

Cause	International classification of disease No.
(a) Upper respiratory infections, influenza, pneumonia, bronchitis, and other diseases of the respiratory system	470-475, 480-483, 490-493, 500-502, 510-527
(b) Pneumonia of newborn	763 and 763.5
(c) Accidental mechanical suffocation	E921-E925

Table 1 shows the cause of death certified by the coroners divided into respiratory and non-respiratory causes, and the length of time the child was in hospital before it died. It also shows which of the deaths were provisionally classified as "cot deaths", and which could be attributed to some definite respiratory or non-respiratory cause. All but one of the 110 cot deaths were certified as due to respiratory causes, and 77 per cent (109) of the 141 deaths certified as due to respiratory causes were provisionally classified as cot deaths.

Cot deaths are likely to be certified differently in different areas, but it seems clear that in most instances the circumstances of death and the *post mortem* findings are such that they are certified under one or other of the respiratory causes of death shows at the foot of Table I.

NATIONAL STATISTICS

There are no published national statistics of the numbers of cot deaths. However, the General Register Office has kindly prepared special tabulations relating to the number of deaths among children aged between 2 weeks and 2 years in England and Wales in 1955, 1958, and 1960. The basic data are given in Table II, which shows that, despite a 5 per cent. Rise in the number of birth between 1955 and 1960, the total number of deaths in the age group fell from 6,993 in 1955 to 6,572 in 1958 and rose only by 3 per cent. to 6,781 in 1960. During the same period the number of respiratory deaths certified by the coroners, of which on the evidence of Table I about four-fifths would have been cot deaths, steadily from 1,432 to 1,674. Thus, although total death rate and total respiratory death rate significantly for both sexes between 1955 and 1958 and continued to fall, although not significantly between 1958 and 1960, rates of respiratory deaths. certified by the coroners showed no significant variation. Many factors may affect these figures, but as they stand they suggest that cot deaths are not decreasing in frequency as are other forms of infant mortality.

In 1960, 1,322 of the 1,674 respiratory deaths certified by the coroners occurred in the home. If the ratio of cot deaths to coroners' respiratory deaths (excluding hospital deaths) observed in the survey is applied to this figure, it produces an estimate of about 1,090 cot deaths in England and Wales in 1960.

TABLE II

TOTAL NUMBER OF DEATHS, RESPIRATORY DEATHS, AND RESPIRATORY DEATHS CERTIFIED BY CORONERS IN IFANTS AGED ROM 2 WEEKS TO 2 YEARS BY SEX ENGLAND AND WALES, 1955, 1958, AND 1960

Sex						
Year	1955	1958	1960	1955	1958	1960
Total Deaths	3,952	3,716	3,784	3,041	2,856	2,997
Total Respiratory Deaths	1,725	1,608	1,681	1,197	1,216	1,254
Respiratory Deaths Certified by Coroner	851	902	960	581	635	714

TABLE III

COT EATH BY AGE AND SEX

Age (months)	Sex		Total
	Male	Female	
Less than 1	1	5	6
1-	5	12	17
2-	17	12	29
3-	16	7	23
4-	4	7	11
5-	2	1	3
6-	4	6	10
9-	3	2	5
12-	1	3	4
18-23	1	1	2
Total	54	56	110
Average Age (mths)	4.3	4.4	
Standard Error of Meane	± 0.53	± 0.54	

TABLE IV
MONTH OF COT DEATHS

Month	Jan.	Feb.	March	April	May	June	July	Aug.	Sept.	Oct.	Nov.	Dec.	Total
No. of Cot Deaths	13	16	18	10	12	5	2	5	2	9	8	10	110

Note: The period of this survey was September 1, 1958 to June 30, 1961

SOCIOLOGICAL SURVEY

Age.-Table III shows the age-sex distribution of the 110 cases classified as cot deaths and indicates that the greatest numbers occur in the 2- to 3-month age groups. The peak appears to occur at a slightly earlier age for females than for males, but the difference in the two distributions is not statistically significant.

Sex.-There were more female than male cot deaths in the survey, but the age-sex distribution of these does not differ significantly from that of all coroners' respiratory deaths.

Time of Year.-Table IV shows the calendar month in which the 110 deaths occurred. The survey does not cover quite three years, so that 9.7 deaths are expected each month, except for July and August, when only 6.5 deaths per month are to be expected.
An excess of deaths during the winter months, which is statistically significant (0.05>P>0.01). This finding is in agreement with other surveys of sudden death in infancy (Banks, 1958; Jacobsen and Voigt, 1956).

There is a seasonal variation in the percentage of births per month. In England and Wales, this variation is less than 10 per cent. The percentages are above average between March and July, and below average from August to February, except for January, when they are usually close to average. The proportion of births is lowest in November. Since 68 per cent. of the cot deaths occur between the ages of 2 weeks and 4 months, the seasonal variation of births does not explain the excess of cot deaths in January, February, and March.

TABLE V
SYMPTOMS OF ILLNESS IN LAST TWO WEEKS BEFORE DEATH

Symptoms	Cases		Controls	
	No.	%	No.	%
None	20	20.6	102	61.5
Symptoms of Respiratory Infection	66	68.0	53	31.9
Symptoms of Gastric Infection	2	2.1	-	-
Other Symptoms Only	9	9.3	11	6.6
Total	97	100	166	100
Not Known	13		30	
Grand Total	110		196	

Symptom of Illness in the Last Two Weeks. Table V and Fig. 1 show the numbers and percentages of cases and controls who had symptoms of illness in the last two weeks before death. The information regarding symptoms of 100 of the control children relates to the last two weeks of the dead child's life. For the remaining 66 controls for whom definite information is available, the replies relate to the last two weeks before the form was completed, which was generally a week or two later.

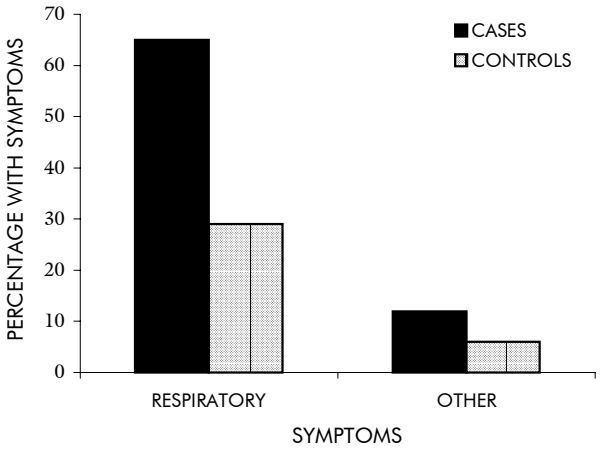

Fig. 1. Percentage of cases and controls who had respiratory or other symptoms of illness in the last two weeks before death.

Included under symptoms of respiratory infection were.
Colds, otitis media, ear discharge, influenza, snuffles, catarrh, sore throat, whooping cough, bronchitis, pneumonia, and rneasles. Diarrhoea was taken as a symptom of an intestinal infection and gastro-enteritis had sometimes been diagnosed by a medical practitioner in the last fortnight.

Included as other symptoms were:
Pale colour, constipation, off feed, colic, anaemia, thrush, vomiting, pains, sores, discharging eyes, and eczema.
Napkin rashes, poor development, prematurity and physical or mental defects were not included under the headings of Table V but were coded separately. Two of the cases were mentally defective, one being a mongol, and one had a cardiovascular defect.

It should be noted that only a definite reply "no illness" or a dash against the questions relating to symptoms was accepted as indicating that the child had no symptoms. It is probable that most of those sown as "not known" in fact had no symptoms.

Children with respiratory and gastric or other symptoms are shown as having respiratory symptoms.

Symptoms of respiratory infection occurred more than twice as frequently among the cases as among the controls. The difference is statistically highly significant (0.001 >P). The cases also included a higher percentage of babies with non-respiratory symptoms than the controls, but this difference is not statistically significant.

Although there is this marked difference between the percentage of cases and controls with respiratory symptoms, it could be argued that bereaved parents, seeking to find some explanation for the tragedy, are more likely than the mother of a living control to recall minor symptoms. There may have been a little biais of this kind, as 50 per cent. of the cases with respiratory symptoms had been seen by a doctor compared with 56 per cent. of the controls with respiratory symptoms. However, had there been a marked degree of over-reporting of minor symptoms among the cases, a much greater difference might have been expected.

Sleeping Conditions. Of the 110 cases, 86 (78.2 per cent.) were discovered dead, nineteen (17.3 per cent.) were discovered ill, and five (4.5 per cent.) were taken ill while under observation. Among those discovered ill were several who may in fact have been already dead.

Since the death or the onset of the fatal symptoms in 95 per cent. of the cases while the child was in its cot or pram, the nature of the bedding has been tabulated in Table VI. The bedding of the cases when List found was very similar to that normally used, but pillows were more often used, and both pillows and mattresses are more often soft for the cases than for the controls. All three differences are statistically significant, the levels of significance being 0.001, 0.05, and 0.0l respectively.

The hardness of the pillow or mattress is a matter of opinion and differences in this respect is due to the fact that a coroner's officer assessed the bedding of the cases and a health visitor that of controls. However, the presence or absence of a pillow is a far more objective measure, and the difference between the normal sleeping conditions of the cases and controls is highly significant respect.

Fourteen of the cases were in bed with others at the time of discovery, although for only six was this normal. Only two of the controls normally slept with others. Many of the cot-death cases were taken into bed with their parents because they were restless. Twenty of the cases were found with mouth and nose completely covered by bedding.

It bas been suggested (Brown, 1960) that, if cot death results from a modified form of anaphylactic reaction after the inhalation of vomit, then it could be avoided if babies were put to sleep face downwards. However, cases included in this study were found face downwards rather more frequently than would be expected from their normal sleeping habits or from those of the controls, the differences being statistically significant (0.05 and 0.01 >P respectively). It seems, therefore, that sleeping face downwards is unlikely to prevent cot death. Agonal movement may, of course, affect the position in which the child is found. The percentage of cases and controls normally sleeping face downwards was very similar.

Breast-Feeding. -Histories of feeding from birth were provided for cases and controls from health visitors' records. These data are not necessarily a complete

TABLE VI
SLEEPING CONDITION

Type of bedding		Cases				Controls	
		When last found		Normal conditions		Normal conditions	
		No.	%	No.	%	No.	%
Pillow	None	51	47.2	51	48.1	135	70.7
	Hard	18	17.7	19	17.9	33	17.3
	Soft	39	36.1	36	34.0	23	12.0
	Total	108	100	106	100	191	100
	Not known	2		4		5	
Mattress	None	1	0.9	1	0.9	1	0.5
	Hard	61	56.0	63	58.4	147	77.0
	Soft	47	43.1	44	40.7	43	22.5
	Total	109	100	108	100	191	100
	Not known	1		2		5	

record up to the age at death because of the irregular contact between the health visitor and the child; there is, however, no apparent bias between cases and controls. Using all complete records, the numbers of cases and controls wholly and partly breast-fed at various ages have been tabulated and express as percentages of the available histories (Table VII and Fig. 2). Only 56 per cent. of the cases were wholly breast-fed for the first two weeks of life compared with 70 per cent. of the controls. Thereafter, the percentage breast-fed falls rapidly with age in both groups. By means of a modified form of life table analysis, it may be shown that the duration of complete breast-feeding is significantly shorter (0.01>P) in cases than controls. This difference also holds for those cases and controls which were initially breast-fed.

Combined analysis. -From the preceding three sections it appears that, for these 110 cases and their controls, cot death is associated with a history of respiratory symptoms, the use of soft pillows and mattresses, getting the mouth and the nose covered by bedding, and a history of early bottle-feeding. These various factors are interrelated.

Numerous cross-tabulations could be made, but it was decided to investigate thirteen factors in a discriminant analysis, restricted to cases and controls with sociological histories which included answers to thirteen questions. There were 82 cases and 146 controls meeting this condition. These 146 controls were tested as regards age and sex to see if they could be regarded as a random sample of the theoretical number of 164 and this hypothesis was not discredited; in case there were any discrepancies, however, sex was added as a co-variance factor to the thirteen factors.

Comparisons of doubtful validity (e.g. how cases were last found compared with how controls were normally found, and histories of symptoms) were deliberately included in this analysis to see whether differences between cases and controls in the use of pillows and bottle-feeding could be explained, how ever implausibly, in terms of these other factors. The analysis showed that they could not, and that the following factors were statistically significant:

TABLE VII
HISTORY OF BREAST-FEEDING AT VARIOUS AGE

Age	Cases					Controls				
	Available Histories	Breast-fed				Available Histories	Breast-fed			
		Wholly		Partially			Wholly		Partially	
		No.	%	No.	%		No.	%	No.	%
Birth	103	58	56.3	18	17.5	186	131	70.4	25	13.4
2 wks	103	40	38.8	18	17.5	186	108	58.1	22	11.8
1 mth	97	24	24.7	15	15.5	177	74	41.8	13	7.3
2 mths	80	7	8.8	8	10.0	151	40	26.5	11	7.3
3 mths	54	3	5.6	2	3.7	97	21	21.6	3	3.1

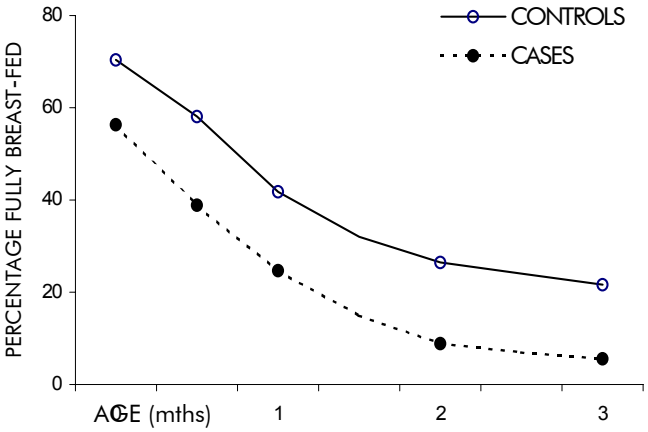

Fig. 2. History of breast feeding of cases and controls based on all available histories up to each age.

Factors	Statistical Significance
Soft Pillow	0.01 > P
Shared Bed	0.05> P
Bottle-feeding during First Two Weeks of Life	0.05> P
Found with Bedding covering Mouth And Nose	
Completely	0.001> P
Partially	0.01> P
History of Respiratory Symptoms	0.001> P

The remaining factors (namely a history of non-respiratory symptoms, the use of a pillow, the use of a soft mattress, sleeping face downwards, partial bottle-feeding in the first two weeks of life, and partial or complete bottle-feeding immediately before death) were not statistically significant.

Other Comparisons. -Cases and controls have been compared with regard to all questions included in the sociological questionnaire. They enable a number of possible causes of the unexplained deaths to be excluded, *e.g.* using paraffin heaters; lack of adequate ventilation; viruses carried by birds, other pets or vermin; vaccination and immunization.

Histories of eczema and asthma and hay fever were also compared in cases and their families with those in controls and their families. There was no evidence to suggest that cases or their families had an increase incidence of allergic reaction.

Analysis of the data also shows the cases of death unexplained sudden death generally come from a poorer type of home than the controls, although living in the same area. This is brought out by the health visitors' opinion of the general standard of mothering and of the home, by the number of visits paid to the home, and the number of visits to the infant welfare clinic. It is also suggested by the fact that a higher percentage of cases were illigitimate (18 per cent. compared with 8 per cent.) and that their mothers were younger (14 per cent. being under 20 years of age compared with 2 per cent.). Also, the social class of the chief wage-earner was lower and there were more persons per room in these homes than there were in the homes in cases than of controls.

DISCUSSION

The sociological data collected have been examined for evidence regarding three hypotheses as to the cause of cot deaths: infection, suffocation, and hypersensitivity reaction.

The infective hypothesis is supported by the finding that the prevalence of respiratory symptoms in the last two weeks before death is a significant factor differentiating cases from controls. The difficulty of obtaining reliable data on symptoms is obvious, but the fact that relatively twice as many cases as controls had been seen by a doctor in connection with respiratory symptoms strongly suggests that the association is real. But although respiratory symptoms were common among the cases there is nothing to suggest that they were generally all severe or that they heralded an overwhelming infection. Twenty cases had no symptoms of illness before death, and in seven of these cases there was no history of illness of any kind in the household.

The suffocation hypothesis is supported by the finding that the use of a soft pillow is a significant factor differentiating cases from controls. This difference may be explained by the fact that the hardness of the pillow was assessed by different people in different circumstances, but the fact that

Cases were more often put to sleep with a pillow than controls cannot be so explained. In addition, these cot-death cases were last found with mouth and nose partly or completely covered by bedding and in bed with others, significantly more frequently than was the control children. But, as already stated, these last two comparisons are of doubtful validity.

Realizing how difficult it is to sum up the risk of suffocation, the coroners' officers were asked to express a confidential opinion as to whether there was *any risk at all* of the child having suffocated. Of 99 answers, in only 33 was the answer in the affirmative.

Jacobsen and Voigt (1958) similarly reported that the face was found covered in 32 per cent. of unexplained sudden and unexpected infant deaths. This is remarkably similar to findings reported in the last paragraph. Jacobsen and Voigt, however, discuss these finding as an attempt to rationalize the event by the parents. They quote Woolley (1945), who demonstrated that a diminution in the oxygen tension of the inspired air is producible only when the infant's face is covered by a close-woven rubber fabric, not with an ordinary porous fabric. Barrett (1954) also thought suffocation unlikely to be the cause of cot death, but conceded that the histological changes seen in three cases in which death was almost certainly due to asphyxia were identical with those seen in cot death.

In favour of the hypersensitivity reaction to cow's milk hypothesis is the fact that m only four cases was the last feed before death reported to have been from the breast. There is also a marked difference between cases and controls in the duration of breast-feeding. Also in favour of hypersensitivity being in some way involved is the fact that the symptoms of hyper sensitivity to cow's milk are vomiting, failure to thrive, gastro-enteritis, and chronic respiratory infection *(Brit. med. J.,* 1963). These symptoms commonly occur in the clinical histories of these cases.

It must be admitted that the lower general standard of mothering among the cases would be expected to be associated with early bottle-feeding and the use of soft bedding and the presence of more persons per room, a higher proportion of beds, and more children in the household, with an increased incidence of infections. However, a poorer type of home, more sibs,

prematurity, and poorer general development do not in themselves provide a cause of death.

The data do, however, provide some evidence in favour of each of the three hypotheses as to the cause of cot death. It is likely that cot deaths may arise from a variety of causes, and that no single hypothesis can fully explain them all. A possible explanation of our findings, however, may be that cot deaths result from a long chain of events. For instance, respiratory infection might cause a blocked noses. Then, if for some reason the child's mouth became obstructed, it would begin to suffocate, which in turn might cause it to vomit. Finally, aspiration of vomit containing cow's milk might set off a fatal anaphylaxis reaction if the child were hypersensitive to it.

SUMMARY

1. Tabulation provided by the general Register Office, and sociological data relating to 110 "cot deaths" and 196 matched controls are analysed.
2. It is suggested that there were probably about 1,100 "cot deaths" in England and Whales in 1960.
3. The sociological data show that "cot death" is significantly associated with the use of a soft pillow, bottle-feeding, especially during the first two weeks of life, and symptoms of respiratory disease in the two weeks before death.
4. Cases and controls are also compared with regard to several other factors.
5. Three hypothesis as to the cause of "cot death" are discussed.

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Short Biographies

We asked our guest speakers to tell us about themselves, their professional life, and, most importantly, about who they are outside the workplace. There were no other guidelines. You will learn much in the next few pages.

Our guest speakers: Who are they?

Luigi Acerbi
Luigi Acerbi is a paediatrician. He was born and lives in Italy and had his school education in Milano. He has worked at Melegnano Hospital, collaborating with the University of Milano, in the Social and Preventive School of Paediatrics. Actually he manages the Community Paediatrics Service of the Local Health Unit, south east of Milano. Since 2001 he is a member of the Regional Health Administration Working group on SIDS. In collaboration with the Italian Seeds for SIDS Association he has promoted the first SIDS awareness campaign in Lombardia region. He has never been previously in Canada and this is the best opportunity to meet friends and visit this country. With his wife (Paola), two daughters (Elena and Francesca), cats and tortoises live in a fine village (Lacchiarella) near Milano.

Atkinson, Rachel
Rachel Atkinson is a proud Aboriginal woman of the Yorta Yorta tribe from Rumbalara in Victoria, Australia. She graduated with a Bachelor of Social Work and an Associate Diploma in Community Welfare from the James Cook University, Queensland, in 1990.
Rachel's early professional background was in social work, child protection, and juvenile justice. For the past seven and a half years she has been the Chief Executive Officer of the Townsville Aboriginal and Islanders Health Services (TAIHS) Limited, an Aboriginal community controlled medical service which employs in excess of 90 staff; three-quarters being of Aboriginal or Torres Strait Islander descent. Rachel was instrumental in the formation of the Australian SIDS and Kids Aboriginal and Torres Strait Islander Advisory Committee. She is the Board Chairman for the Queensland Aboriginal and Islander Health Forum, QAIHF, and a member of the national executive of the National Aboriginal Community Controlled Health Organisation, NACCHO. Rachel sits on a number of health advisory committees that set national health priorities. Rachel is a strong advocate for equal rights and for Aboriginal health advancement. She believes in the Aboriginal concept of health as holistic and encompassing the individual, the family and the community. She recognizes that ill-health and sickness are often caused by poverty and poor living conditions. Rachel's work in Aboriginal health has encompassed child protection, family violence and sexual abuse, maternal and child health, social and emotional health and wellbeing, and health promotion. Rachel is a mother and a SIDS grandmother.

Auger, Darlene
Darlene is a Canadian Aboriginal Women of the Wolf Clan of the Plains Cree Tribe. Originally from Wabasca, Alberta, she now resides in Edmonton. First she is a mother of two, Fawn and Kistin and secondly she is a scholar, researcher, writer, actress, singer and facilitator. Darlene has a degree in psychology and plans to obtain her doctorate in traditional healing. She has been working with people using her hands and intuitions to bring about healing and well-being for a long time.

Bajanowski, Thomas
Thomas Bajanowski was born in Jena, Germany. He is married since 1982 and has two daughters, Juliane (21 years old), Barbara (17 years old).
He graduated in medicine from Friedrich-Schiller-University, Jena. He became a specialist in legal medicine in 1990 and he is presently Director of the Institute of Legal Medicine, University of Duisburg-Essen. He is a member of the International Academy of Legal Medicine and a member of the Executive board of the European Society for the Prevention of Infant Death.

Baugher, Bob
Bob Baugher is a psychologist, author of books on grief, including "Understanding Guilt During Bereavement," and a 17-year professional advisor for the Seattle chapter of The Compassionate Friends.

Blackwater, Andy
Blackfoot name: SHOT ON BOTH SIDES. Andy is a member of the 'Fish Eater Clan" of the Blood Tribe (KAINAI) one of the tribes of the Blackfoot Confederacy. He is in his sixty fifth year and has resided on the Blood Reserve all of his life. He is a parent, grandparent and husband to wife Phyllis.
Andy has served his people as tribe leader (Councilor) for several years and he now serves as a special advisor to them and others in many aspects of life. He is very active in culture and tradition and participates in transfers of Sacred Bundles and other sacred items. (Example: Brave Dog Society Rattle Owner).
Today, Andy is acknowledged as a traditional grandfather (traditional Elder). He is a Traditioanlist, Ceremonialist, Historian and Philosopher in the Blackfoot culture. Andy has become the most ACTIVE unemployed individual around!

Blackwell, Caroline

Currently Caroline Blackwell is working at the University of Newcastle in Australia , a very long way from "home" as she was born in southern Louisiana. Her introduction to SIDS was in the late 1960s when the first baby of a childhood friend died of "crib death". At that time she was a postgraduate student in medical microbiology at Stanford University, and was asked to apply her research skills to find out why the baby had died. This is a question heard many more times when she started working on SIDS at the University of Edinburgh in Scotland in the late 1980s. She had been working on genetic and environmental risk factors for infections, and the risk factors for SIDS emerging from the epidemiological studies appeared to parallel those they were investigating in relation to diseases caused by bacteria and viruses. The parallels between infection and SIDS became clearer as data for cot deaths in different ethnic groups was published. Populations with the highest incidence of SIDS such as Indigenous groups in Australia, North America and the Maori of New Zealand also have high incidences of childhood infections. This led to her last academic move in 2002 to the University of Newcastle in Australia where she has become part of a multidisciplinary team dedicated to improvement of the health of Australia's Indigenous people.

Blair, Peter

Peter Blair was born in the industrial North of England and he was rarely seen without a football at his feet playing the cobbled-street pitches in Manchester. After realising he was not going to be given the number 9 shirt to play for his beloved United he took solace in other numbers. First obtaining a Mathematics degree and then studying for a Masters in Medical Statistics at the University of Leicester.

He moved South to Bristol in 1992 joining a thriving team of SIDS researchers led by Peter Fleming. In 1998 he completed his Doctorate entitled "*Assessing the changing risk factors associated with SIDS"* and was responsible for the analysis of the large UK CESDI study. Bristol is also home to the Avon Longitudinal Study of Parents and Children (ALSPAC) which is still collecting data from 14,000 mothers and infants enrolled since 1992-3. This has given him the opportunity to look at several epidemiological aspects of infant morbidity as well as mortality including how poor infant growth impacts on later life. His main interest however is still SIDS and he is currently helping to manage the South West Infant Sleep Scene (SWISS) study, one of the first studies to control for death-scene investigations by conducting a similar sleep-scene investigations of control infants.

He has only briefly visited Canada for one day, en route to a busy New Zealand Conference in 2000, but this was literally a high point of the trip staying on the 24th floor of a Vancouver Hotel with a beautiful view.

Outside SIDS research he has 2 young sons (4 and 6 years), both of whom seem much more interested in football than numbers, but no doubt they'll learn.

Byard, Roger

Roger Byard currently work at the Forensic Science Centre in Adelaide, Australia, as a Specialist Forensic Pathologist. He qualified in medicine in Tasmania, Australia in 1978 and then worked in Canada (Hamilton, Ottawa & Toronto) for a number of years where he obtained Canadian qualifications in family practice (CCFP) and anatomical pathology (FRCPC). His wife is originally from Ottawa, Ontario. His research interests are in SIDS and in 'Preventative Pathology' part of which involves coordinating childhood accident prevention through the 'Keeping Your Baby and Child Safe Program' in South Australia. He has two dogs, Alice and Sophie.

Cardinal Jenny

Jenny Cardinal is a well respected and knowledgeable Elder from Saddle Lake First Nation. She has vast experience on the variety of issues including addictions, parenting, and traditional knowledge and values.

Cutz, Ernest

Ernest Cutz is a Professor of Pathology in the Department of Laboratory Medicine and Pathobiology, University of Toronto, Senior Staff Pathologist in The Division of Pathology, The Department of Paediatric Laboratory Medicine and Senior Associate Scientist, The Research Institute at The Hospital for Sick Children (HSC) in Toronto. He is a graduate of Charles University Medical School in Prague. He completed his post graduate medical education in Czechoslovakia, France and Canada. He has a long term interest in SIDS both as a diagnostic Pathologist and a member of Paediatric Forensic Unit at HSC as well as performing basic research relevant to the pathobiology of SIDS. Together with Dr. L. Becker he has established a SIDS Research Program at HSC with studies on the respiratory control mechanisms. This joint work has resulted in the discovery of catecholamine abnormalities in the carotid bodies of SIDS victims. His current work funded by grants from Canadian Institutes for Health Research is focusing on the role of airway oxygen sensors, described in his laboratory. His is a chairman of SIDS research committee at HSC and a member of scientific advisory committee of Canadian Foundation for the Study of Infant Deaths.

Daniels, Christine
Christine Daniels is a well respected and knowledgeable Elder from Saddle Lake First Nation. She has vast experience on the variety of issues including addictions, parenting, and traditional knowledge and values.

Daniels, Jo-Ann
Ms. Daniels is Cree/Metis and has been working in community development for the last 25 years on various issues concerning education, housing, social justice, health, social welfare and presently on crime prevention.
Ms. Daniels has developed, presented and facilitated workshops at many major conferences; and also trained the trainers, teaching professionals community approaches and philosophies to community-driven action and responsibilities.
Ms. Daniels worked extensively with Elders across North America and brings their knowledge to the work she presents and shares. She is currently with Aboriginal Community Support as a Co-ordinator, and with Alberta Community Crime Prevention Association.

Dedam-Montour, Debbie
Ms. Dedam-Montour is a Mi'kmaq from the Listuguj First Nation in Quebec, Canada. In February 2004, she was appointed Executive Director of the National Indian and Inuit Community Health Representatives Organization (NIICHRO). Her involvement with aboriginal health issues began in the year 2000 when she accepted the position of Trust Administration Officer for the wage parity compensation awarded to Community Health Representatives (CHR Settlement Trust). Through her involvement in distributing the compensation, Ms. Dedam-Montour has become very aware of the numerous challenges faced by Community Health Representatives thus appreciates opportunities to inform others of the integral role they play in aboriginal communities. In 1989, Ms. Dedam-Montour held the position of Employment Recruitment Officer for the Quebec Native Women's Association where she developed an Aboriginal Human Resources Directory.
Her involvement in the field of Aboriginal employment and training continued into the 90's with the *Pathways to Success* Strategy where she developed policies as a board member and later as Executive Director of the Aboriginal Workforce Association of Montreal. As well as being an Executive Board member of the First Nation Human Resources Development Commission of Quebec, her experience has given her knowledge and expertise necessary to achieve NIICHRO's goal of developing standards of practice and accreditation for CHRs. Ms. Dedam-Montour is committed to community and human resource development for the enrichment of healthy aboriginal communities and to the support of Community Health Representatives.

Dorey, Dwight Allister
Dwight Allister Dorey is national chief of the Congress of Aboriginal Peoples (CAP). He was re-elected to the position at the organization's annual general assembly on November 8, 2002 for a second three-year term of office. The Congress -- formerly Native Council of Canada -- is the national advocacy organization for more than 800,000 off-reserve Aboriginal peoples living in urban, rural and remote areas throughout Canada. Chief Dorey, a Mi'kmaq from Lunenburg County, Nova Scotia, has more than 25 years of experience in Aboriginal politics at the provincial, national and international levels. Prior to his first election as National Chief, he was senior policy advisor to the Congress. He served as elected Chief and President of the Native Council of Nova Scotia from 1989 to 1997. First elected in May, 1977, he also served as Vice President of the Native Council of Nova Scotia until 1989, with the exception of a two year term (1984-85) as Vice President of the Native Council of Canada. His extensive political experience has been strengthened by his senior management career as president and general manager of the Mikmakik Development Corporation in Nova Scotia, and as operations manager for the Associated Management Group in Toronto. Chief Dorey holds a Master's Degree in Canadian Studies from Carleton University in Ottawa. He is the co-editor of "The Aboriginal People's Movement Off-Reserves -- a book published in 2003. He has served on several boards of directors nationally and regionally, and has travelled widely internationally as a participant and delegate at several United Nations and Organization of American States meetings and conferences.

Dyregrov, Atle
Dr. Atle Dyregrov is the director of Center for Crisis Psychology in Bergen, Norway. He is a clinical and research psychologist, holding memberships in the Norwegian Psychological Association as well as an associate membership in the American Psychological Association, and he is a board member of the International Critical Incident Stress Foundation and one of the founders of the European Society for Traumatic Stress Studies. He lectures both at the Faculty of Medicine and Faculty of Psychology at Bergen University. His former positions include 4 years of clinical and research work at the Children's Hospital in Bergen, and 4 years at the University of Bergen. He has been the director for Center for Crisis Psychology since its formation in 1988.
Dr. Dyregrov is the author of numerous publications, journal articles and books including "Grief in children" published in 10 languages, "Disaster psychology", "Grief

and care within schools" (co-author: Magne Raundalen), «Children and war « (co-authored with Magne Raundalen), «Children on the Intensive Care Unit» (co-authored with several others). and «Children and Trauma». His book on sleep "The little sleep book" was published in Norwegian in 2001 and is also published in the other Scandinavian languages. His book with the title: "Psychological debriefing: a leader's guide for small group crisis intervention" were published by Chevron Publishing Company in 2003.
He has lectured extensively in the Scandinavian countries, as well as in Europe and the US. He has worked extensively as a consultant to different UN organizations, especially UNICEF in their work for children in war in Africa, the Middle East and former Yugoslavia, and UNHCR in developing staff support routines around the world.
His clinical work has covered areas such as: grief reactions in parents following the loss of a child, grief and trauma in children, organizing psychosocial disaster assistance, Critical Incident Stress Debriefing work with disaster-workers and other helpers, and work with children in war situations.

Dyregrov, Kari
Kari Dyregrov is the mother of three children; she is married to Atle Dyregrov. She worked as a physiotherapist for several years until she completed her masters and PhD degrees in sociology at the University of Bergen, where she specialized in qualitative research methods and in health and social policy. Since 1995, she has been working as a researcher of traumatic bereavement at the Centre for Crisis Psychology in Bergen, Norway, where she has been the project leader for several research projects.
Her research topics have covered areas such as: grief reactions and self-help strategies in bereavement, social network responses to traumatized people, the organization of psychosocial assistance in the community, and the ethical and methodological aspects of research on adverse life situations.
In November 2003 Kari Dyregrov finished her doctorate degree: "The Loss of a Child by Suicide, SIDS, and Accidents: Consequences, Needs, and the Provision of Help." In 1999, based on the results, which showed a need for an organization for the suicide bereaved, she took the initiative to establish LEVE - The Norwegian Organization for Suicide Survivors. She has worked as a member of the National Board of LEVE since its establishment.

Fisher, John
John Fisher received his Ph.D. in Physiology from McGill University, followed by post- doctoral training and a faculty position at the University of Texas Medical Branch, Galveston. He subsequently returned to Canada to join the Hospital for Sick Children Research Institute in Toronto, and then the Department of Physiology at Queen's University, with cross-appointments to the Departments of Medicine and Paediatrics. Dr. Fisher's research is funded by CIHR and the Ontario Thoracic Society and focuses on airway sensory receptors, the regulation of airway caliber and control of breathing pattern in the newborn.

Fleming Peter
After Medical School in Bristol (UK) Peter Fleming worked as an intern in Paediatrics in the University hospital in Saskatoon before returning to the UK where he initially qualified in Adult Internal Medicine. He subsequently trained in Paediatrics in Bristol and at Great Ormond St Hospital in London. He then returned to Canada for a further two years, at the Hospital for Sick Children in Toronto. It was in Toronto that he developed a particular interest in the causes of unexpected infant death, and in developmental physiology. He qualified as a paediatrician in Canada, and maintain his Canadian links through a Fellowship of the Royal College of Physicians of Canada.
He returned to Bristol in 1978, and set up a laboratory to investigate the developmental physiology of infants and young children, with a particular interest in sleep. In 1980 he set up a service to provide help and support to families bereaved by the sudden death of their infant, and in 1984, with Dr Jem Berry, Paediatric pathologist, he set up the Avon Infant Mortality Study.
Since 1984 he has led a series of research studies into unexpected infant deaths, in Bristol and throughout the UK. In 1989 he implemented, with his team, the first "Back to Sleep" campaign in Bristol, and the results of this were the impetus to the UK Government to mount such a campaign 2 years later. In the 1990's he led the CESDI study of Unexpected Infant Deaths, and he is currently head of the South West Infant Sleep Scene (SWISS) Study which aims to identify the ways in which infants' sleep environments affect growth, health and development as well as the risk of unexpected death.
Outside medicine, his main interests are spending time with his family (he has four sons), running marathons and mountain biking, though now he has great trouble keeping up with his boys

Frappel, Peter
Peter Frappell is Reader and Associate Professor, Adaptational and Evolutionary Respiratory Physiology Laboratory, Department of Zoology, La Trobe University, Melbourne, Australia.

A Tasmanian by birth, Peter completed a B.Sc. (Hons) at the University of Tasmania and his Ph.D. at Flinders University of South Australia under the supervision of the late Professor Russell Baudinette, where his interest in marsupial pouch young, metabolism and thermogenesis was fostered. This was followed by a Medical Research Council of Canada Fellowship at McGill University working with Professor Jacopo Mortola on comparative aspects of ventilation and metabolism in mammals. Peter returned to Australia in 1992 to take up a lecturing position in the Department of Zoology at La Trobe University. His laboratory is mainly concerned with various aspects of the oxygen cascade and what effects body size, development and environmental stress have on the various parameters involved in the delivery of oxygen. Areas that are currently under investigation include: the determinants of aerobic capacity in varanid lizards; ecophysiology of *Daphnia* in particular adaptation/acclimation to low oxygen levels; hypoxic hypothermia and the resetting of the thermal set-point in mammals; adaptation to altitude; inter-individual variability in physiological parameters; the effects hypoxia and temperature may have on metabolism, ventilation and oxygen transport, and the control of breathing. Peter is currently Head of Department and adopts an eclectic approach to life.

Gleeson, Maree

Maree Gleeson is fortunate to have dual professional roles in immunology that allow her to be the Director of the diagnostic immunology pathology services in a large teaching hospital in Newcastle, Australia, as well as medical researcher in the Faculty of Health at the University of Newcastle (Australia). Newcastle was where she was born and although she studied at the University of Sydney she is pleased to be back in her hometown providing a service to the community. Her introduction to SIDS research was very much like the syndrome - sudden and unexpected. In the early 1980s she was studying for her PhD thesis on a project that was looking at how the immune system develops in children and how it provides protection at external body surfaces. Her research involved studying babies from the day they were born until they were 5 years old to determine what factors altered their immune protection and caused the subsequent development of asthma and allergy. Unfortunately one of the babies in her study died unexpectedly at 10 weeks. This was her first exposure to SIDS. At the end of the 5 years of research theye realised that this baby had a very different pattern of immune response to a mild respiratory tract infection than any other child in the 5-year study. The research was able to provide insight into the risk factors for SIDS and the role of respiratory infections. The group has since expanded this research to assess the role of infections in the "near-miss' SIDS infants who present to hospital with acute life threatening events, to investigate the infective causes and mechanisms that could lead to death. Their recent research has been in collaboration with Caroline Blackwell to determine the genetic and environmental risk factors for infections, particularly in Australian Indigenous populations where the incidences of SIDS and infections are high. They work as part of a multidisciplinary team at the University of Newcastle dedicated to improvement of the health of Australia's Indigenous people.

Haddad, Gabriel

Dr Haddad is presently University Chairman of the Department of Pediatrics at the Albert Einstein College of Medicine (New York); and Pediatrician-in-Chief at the Children's Hospital at Montefiore. He has 3 wonderful children (Chris 22, Diana 21 and Justin 16) and a very understanding wife Karen. His new job has consumed most of his time but he is enjoying it. He jogs 3-4 times a week and at the moment is preparing for a half marathom for the labor day race in New Haven.

Half, Kay

Originally from Whitefish Lake First Nations, Band 128. Second youngest of seven girls, daughter of Mary Half and the Late Mr Jules Half of Good fish lake. Worked 8 years in the area of health for the community and 16 years as the CHR Coordinator for First Nations Inuit Health Branch, Health Canada. She is presently Regional CHR Coordinator First Nations Inuit Health Branch, Health Canada.

Harper, Ronald

Ronald Harper was born in Newcastle (now Miramichi), New Brunswick (Canada), and received his undergraduate degree at Dalhousie University in Halifax, Nova Scotia (Canada). He obtained his Master's at Tufts University in Boston, USA, and his doctorate at McMaster University in Hamilton, Ontario (Canada). He is currently a Professor in the Department of Neurobiology, University of California at Los Angeles and a member of the UCLA Brain Research Institute. He is past-President of the Sleep Research Society, and is a member of several other scientific societies. His interest in SIDS stems from research into brain mechanisms underlying sleep. Early in his studies, it became apparent that what should be periods of rest in reality are often times of great physiological stress for many individuals; the action of brain processes during sleep on breathing and cardiovascular control can endanger survival. The objective of his research program is to determine the means by which brain structures act to maintain vital

functions; to this end, he examines infants who are at risk for sudden death in infancy, children who are unable to sustain ventilation during sleep (Congenital Central Hypoventilation Syndrome), and adults with obstructive sleep apnea (OSA) or heart failure.

Hauck, Fern

Fern R. Hauck is currently Associate Professor of Family Medicine and Director of Research in the University of Virginia Department of Family Medicine. Dr. Hauck's medical degree is from St. Louis University School of Medicine and her residency training was conducted at the Maine-Dartmouth Family Practice Residency in Augusta, Maine. She received postgraduate research and health policy training through the Robert Wood Johnson Foundation Family Practice Faculty Development and Research Fellowship at Case Western Reserve University School of Medicine, the Centers for Disease Control and Prevention's EIS Program, and as Senior Scholar in Residence at the Agency for Healthcare Research and Quality, Center for Primary Care Research.

Dr. Hauck's primary research focus has been Sudden Infant Death Syndrome (SIDS) and other Sudden Unexpected Deaths in Infancy (SUDI). She was principal investigator of a large case-control study of postneonatal infant mortality in Chicago, the Chicago Infant Mortality Study. The primary objectives of the study were to examine risk factors for SIDS and other infant deaths, assess parental knowledge of sleep position recommendations, and to develop and evaluate standardized scene investigation protocols. Further interests that evolved from this research are the development of SIDS risk reduction strategies focusing on African-American and other minority communities, and determining criteria for diagnosing sudden infant death. She has been a medical and scientific advisor to a number of SIDS/SUDI organizations and federal agencies, and serves on the American Academy of Pediatrics Task Force on SIDS.

Dr. Hauck has considerable international health experience and maintains an active interest in this area. She has worked in Southeast Asia, Africa and the Caribbean, including serving as a refugee camp doctor for a year in Thailand, where she provided training and care for Cambodian refugees. In October 2002, Dr. Hauck founded and currently directs the International Family Medicine Clinic in the University of Virginia Department of Family Medicine, where she and colleagues provide comprehensive primary health care for the growing number of refugees and other immigrants in central Virginia.

On a personal note, Dr. Hauck was born in New York City and currently resides in rural Virginia with her husband and 4-year old twins. She enjoys travel, gardening, theater, music, and reading, but most of all, she enjoys spending time with her family.

Horchler, Joani Nelson

Joani Nelson Horchler, a journalist by training and experience, felt compelled to create for herself a new career--trying to help other people through grief--after the SIDS death, in 1991, of her two-month-old son, Christian Gabriel. In 1994, she founded SIDS Educational Services Inc. (SIDS-ES), a nonprofit organization dedicated to providing empathetic support to bereaved families, and serves as its executive director. Through SIDS-ES, she co-authored and published in 1994 her first book, the SIDS & Infant Death Survival Guide (formerly entitled The SIDS Survival Guide), which has been called the most comprehensive book ever written on SIDS.

Hunt, Carl E.

Carl Hunt is a native of upstate New York. He received his A.B from the University of Rochester (New York) and his M.D. from Yale University, New Haven, Connecticut. He obtained his training in pediatrics and neonatology at the University of Minnesota, Minneapolis. Following his initial pediatric faculty appointment at the University of Minnesota, he was the Chief, Division of Neonatology at Children's Memorial Hospital in Chicago and Associate Professor of Pediatrics at Northwestern University Medical School. He served as Professor and Vice Chair of Pediatrics at Northwestern University from 1983 until his appointment as Professor and Chair of Pediatrics at the Medical College of Ohio (Toledo) in 1988.

Dr. Hunt was appointed as Director of the National Center on Sleep Disorders Research at the National Institutes of Health (Bethesda) in 2001. The National Center on Sleep Disorders Research (NCSDR) supports sleep-related research and training, and is an advocacy and coordinating center for sleep education programs for health care professionals and for the public.

Dr. Hunt has been active in basic and clinical research related to SIDS and other breathing-related sleep disorders in infants and children. He continues to be an active investigator for the Collaborative Home Infant Monitoring Evaluation (CHIME) project. He is the author of numerous peer review journal publications and book chapters.

Jenkins Family

The Jenkins/Morin Family has experienced multiple losses to SIDS. There have been two generations affected resulting in Theresa Whiskeyjack being both a SIDS Grandmother and Great Grandmother. Through the tragedy of loss, they have endured and supported one

another. They all continue to raise awareness and to promote health and healing for their community in a variety of ways. They hope, through sharing the impact SIDS has had on their family they can raise awareness and promote healing for other families coping with the loss of a baby.

Kahn, André

André Kahn is a pediatrician, and father of three children. With his family he lives in an old house, in the country-side near Brussels, where friends are always welcome. In 1978, while his daughter Caroline was sleeping in her cot, she abruptly suffered a severe ALTE event. He could, by chance, help her survive the acute event. This prompted him to study the causes of the event, and the potential mechanisms for SIDS. In October 1985, he organized an international meeting in Brussels, where scientists and SIDS parents met for the first time. The next year the initial meeting of SIDS families took place in Lac Como, in Italy. His research work on the characteristics and mechanisms of SIDS has developed since with the help of many friends around the globe.

Kinney, Hannah

Hannah C. Kinney, M.D., is a neuropathologist at the Children's Hospital in Boston (USA), and Professor of Pathology at Harvard Medical School. She received her M.D. degree from Case Western Reserve University School of Medicine, Cleveland, Ohio in 1974, followed by residencies in pediatrics and pathology, and fellowships in clinical and experimental neuropathology. She has participated in SIDS research for almost 20 years. Her focus is upon possible brain mechanisms underlying SIDS.

Klass, Dennis

Dennis Klass, Professor of Religious Studies, Webster University, St. Louis, Missouri, USA, earned his doctorate in the Psychology of Religion at the University of Chicago. He has written over 40 articles or book chapters on death and grief and on the psychology of religious leadership. His current scholarly work is on comparing grief and continuing bonds with the dead across several religious traditions.

Krous, Henry

Henry Krous was born in Denver, Colorado and raised in Nebraska, Dr. Krous is currently the Director of Pathology at the Children's Hospital-San Diego and Professor of Pathology & Pediatrics at the University of California, San Diego School of Medicine. He is a member of the Society for Pediatric Pathology, of which he is a Past President, as well as the European Society for the Prevention of Infant Death, American Professional Society on the Abuse of Children, National Association of Medical Examiners, and the United States/Canadian Association of Pathology. He has served on the Executive Committee and Chair of the Pathology Group of the SIDS Global Strategy Task Force, Vice Chair of the SIDS Advisory Council to the California Department of Health Services, and San Diego County Child Death Review Committee. He is a member of the Editorial Board of Pediatric and Developmental Pathology. He is the author of numerous research papers, abstracts, and chapters, and has co-edited with Dr. Roger Byard the recent book, Sudden Infant Death Syndrome. Problems, Progress & Possibilities. He has been invited to give numerous national and international lectures about Sudden Infant Death Syndrome and other topics in forensic pediatric pathology. Dr. Krous is a recipient of the Senator Daniel E. Boatwright Award for "extraordinary public service on behalf of Californians touched by SIDS."

Kukdookaa (Terri Brown)

Kukdookaa was born into the Tahltan Nation. Her strong matrilineal culture has shaped her philosophy and values as a Tahltan woman. Terri is a member of the Crow clan. Terri is the mother of three children, Aaron, Jasmine and Elliott.

Terri attended residential schools in Whitehorse from the age of 10. She acquired a BA in Economics, minor in Linguistics at Simon Fraser University. Her political activism spans 3 decades. Work history includes service to First Nation communities in Band offices, Tribal Councils and Corporations. During the past decade she has worked primarily with women's organizations including; Aboriginal Women's Council, Indian Homemaker's Association of BC, Vancouver Status of Women, and the Helping Spirit Lodge Society. Terri was elected the first Aboriginal President of the National Action Committee on the Status of Women. Participated in the following events:

- Represented Canadian women at the Beijing +5 UN Conference, New York City
- Visited war affected camps in Eritrea and met with officials and toured Oxfam projects in Ethiopia
- Speaks at international and national gatherings on aboriginal women's issues.
- Represented G8 countries and presented to the UN on poverty and violence
- Participated in the preparation of "Gathering the Voices" document and presented at the WCAR
- Attended the World Conference Against Racism in Durban, South Africa

- Working Group, Draft Declaration on the Rights of Indigenous Peoples, UNN Geneva
- World Intellectual Property Organization, UNN Geneva

Terri grew up on a remote reservation. Her strong cultural and spiritual identity has given her the motivation and strength to survive the many hardships that aboriginal women face. Terri was greatly influenced by her late mother, Mrs. Jean Brown, who encouraged her to get an education, and to leave her community for opportunities to advance her self and to be a proud Tahltan woman.

Terri is the President of the Native Women's Association of Canada and as such continues to provide a voice for Aboriginal women to ensure that they are included in the decision-making and governance of our Nation and effect real change.

Kuwaki, Tomoyuki

Tomoyuki Kuwaki was born in Japan and has been living in Japan since then. He apologize for his "broken" English since he has never studied abroad. He has been interested in how the brain controls the lung, heart, and blood vessels for adjusting their performance to our bodily demand. He is also interested in SIDS, because knowing disease is the best way to learn health, and vice versa.

Langille, Tina

Tina Langille is a practicing Registered Nurse at Henry Ford Hospital in the Neonatal Intensive Care Unit in Detroit, Michigan, USA, but resides in LaSalle, Ontario, Canada. With her 15 years in the NICU, Tina has taught 100's of nurses about Death and Dying in the NICU, developed and co-ordinates The Bereavement follow-up program where she is in contact with bereft families for about a year after the death of their baby. Tina has also served as a volunteer for The Canadian Foundation For The Study of Infant Deaths (CFSID) since the death of her daughter in 1995 from SIDS. Tina started a local Chapter in Windsor, Ontario, where she has facilitated a support group for bereft families since 1996, presented numerous educational sessions for nurses, police officers, expecting parents and students. She has been recognized as Volunteer of the year in 2000, wrote a public education manual on SIDS (1999) that was distributed to all CFSID Chapters and Affiliates, appeared as a SIDS educator in the new STEPS video (2003) for reducing the risk of SIDS in the teen population and sits on the CFSID Board of Directors(2002) as Vice-President. Tina gives a unique balance to her presentations with professional and personal experience with many different types of infant deaths, and grieving families.

Larsen, Denise

Dr. Denise Larsen (Ph.D, C. Psych) is Assistant Professor of Counseling Psychology at the University of Alberta and Director of Research at the Hope Foundation of Alberta. Denise is a former elementary school teacher. She was also a practicing psychologist at two major cancer hospitals for several years. Her research interests focus on the study of hope in education as well as in health care. She currently maintains an active hope-focused counseling practice with the Hope Foundation of Alberta, which is located in Edmonton

MacCormick, Mary

Mary MacCormick became a volunteer for the Canadian Foundation for the Study of Infant Deaths after her 17-year-old daughter's son, Jesse, died in 1990. The death of her grandson prompted her passion to educate all teens about SIDS and reducing the risk. In 2001, she was hired by CFSID to coordinate the Special Teen Education Program SIDS (STEPS), a new program designed for the teen audience. Mary is passionate about this program and has dedicated her work to the memory of her grandson.

Mary MacCormick and her husband reside in St. Catharines, Ontario (Canada). They are the proud parents of Sandra, 31, Jonathan, 23 and Tyler, 21. They have four grandchildren ranging in age from 2 months to 12 years old.

Matturi Luigi

Born in Italy. Since 1972 Full Professor of Pathology and Chairman of the Institute of Pathology University of Milano, Italy. Since 1980 Head of the First School of Specialization in Pathology, University of Milano. Since 1995 Coordinator of PhD Program in Pathological Anatomy, University of Milano. Since 2000, Coordinator of the Master of Pathology of Stillbirth and SIDS, University of Milano. Since 1999 Chief of the "Reference center of the Italian Republic's Lombardy Region for pathological study of Unexpected Fetal death and Sudden Infant death Syndrome (SIDS) victims," Institute of Pathology, University of Milano, Lombardy Region. Head of the E-Learning Center of the University of Milano 1989-2000. Since 1990 President of the Italian Society of Biomorphometry. Member of the Italian Committee on Medical Research and Public Health 1982-1983. Associated member of the Argentine Cardiology Society. Visiting Professor of the University of Buenos Aires, Argentine. Honorary Professor of the UNIBE University, San Josè, Costa Rica. Since 1998 Vice President of the "Italian Society of Stillbirth and SIDS" (S.I.M.I.P.). The scientific activity (over 250 publications) is mainly addressed to the study of cardio-vascular pathology (atherosclerosis and cardiac conduction system) and to the investigation of the neuropathology of

Stillbirth and SIDS. Recipient of the Gold Medal Award of the City Hall, Milano 1988. Appears in the Special Millenium Edition of Who's Who in Science and Engineering. Since 1996 member of the Editorial Board of "Cardiovascular Pathology", the official journal of the American Society for Cardiovascular Pathology.

Martin, Karen
Karen Martin (MA PhD candidate) is a sociologist in Edmonton. She is a university and community death educator, and author of When a Baby Dies of SIDS: The Parents' Grief and Search for Reason.

Melvin , Cathy
Dr. Cathy Melvin is a Senior Research Fellow and Director of Child Health Services Research at the Cecil G. Sheps Center for Health Services Research and a Research Associate Professor in the Department of Maternal and Child Health at the School of Public Health at the University of North Carolina at Chapel Hill. Dr. Melvin holds Bachelor of Arts with Honors (1974), Master of Public Health (1976) and Doctor of Philosophy (1985) degrees from the University of North Carolina at Chapel Hill. She serves as Principal Investigator and director of the Robert Wood Johnson Foundation-funded Smoke-Free Families National Dissemination Office.
Dr. Melvin's research interests include dissemination and dissemination research, translation of research into practice, health systems change and tobacco control and prevention, especially for pregnant and parenting smokers. Dr. Melvin has directed tobacco initiatives at the U.S. Centers for Disease Control and Prevention to reduce smoking during pregnancy, estimate the costs attributable to maternal smoking, provide training and other support for cessation programs, monitor smoking and other maternal behaviors affecting birth outcomes, and prepare guidelines for successful interventions with pregnant smokers. She has served as a temporary advisor to the World Health Organization on cessation issues.
She is co-founder and chair of the National Partnership to Help Pregnant Smokers Quit (National Partnership), the only national organization working to mobilize the health care system and local communities to help pregnant smokers get the help they want and the support they need to quit smoking and remain tobacco-free.
Dr. Melvin an editorial board member for the American College of Obstetricians and Gynecologists Educational Program for Smoking Cessation during Pregnancy; and a scientific advisory panel member of the Association of Women's Health Obstetric and Neonatal Nurses Research Based Practice Guidelines on Treating Tobacco Use during Pregnancy.
Over the last 20 years, Dr. Melvin has provided health program development, epidemiology, surveillance, evaluation and research consultations in public and private settings in both the US and other countries.

Miller, Sally
Dr. Sally Downham Miller is one of the country's leading authorities on grieving and overcoming loss. She is the Director of Mourning and Dancing – a non-profit grief support organization. She also has had a long career in public education as teacher, professor and school principal. She has spent much of the past two years in New York City and its suburbs working with schools, Families and communities affected by the terrorist attack of September 11th.
Sally is a frequent guest in the media and a regular on the *Montel Williams Show*. She is the author of two books, entitled *Mourning and Dancing: A Memoir of Grief and Recovery* and the award- winning *Mourning and Dancing* for Schools. She lives in Indiana and in New York City with her husband Dr. Will Miller.

Mitchell, Ed
Ed Mitchell is a paediatrician. He was born in Iran, had primary school education in South Yemen, and for secondary school education went to boarding school in England. He did his undergraduate medical degree at St George's Hospital Medical School in London. He has worked in the UK, Zambia and New Zealand where he has lived for the last 25 years. He is currently Professor of Child Health Research at the University of Auckland. His clinical work is as a developmental paediatrician
In 1984 he was asked to establish and chair a committee to review all deaths in infancy in the Auckland region. The number of deaths from SIDS was horrendous and the similarity of these deaths was striking. This triggered his quest to understand and prevent SIDS.
He is married with two grown-up children (grown-up at least some of the time!), and spends as much time as possible sailing.

Morin Family
The Jenkins/Morin Family has experienced multiple losses to SIDS. There have been two generations affected resulting in Theresa Whiskeyjack being both a SIDS Grandmother and Great Grandmother. Through the tragedy of loss, they have endured and supported one another. They all continue to raise awareness and to promote health and healing for their community in a variety of ways. They hope, through sharing the impact SIDS has had on their family they can raise awareness and promote healing for other families coping with the loss of a baby.

Morris, James A

James Morris is a consultant pathologist working in Lancaster, UK. He was born in the north of England, studied medicine at Cambridge and Manchester and then pathology at Bristol and Leeds. He first became interested in SIDS in the early 1980s. He performed a post mortem examination on a young women who literally dropped dead due to tampon associated staphylococcal infection leading to toxic shock syndrome; shortly afterwards he examined a case of SIDS in which the infant had a heavy growth of S. aureus in the nasopharynx. This led him to wonder if bacterial toxins could cause SIDS and to propose the common bacterial toxin hypothesis. Professor Morris is a hospital based pathologist but he has close links with Lancaster University and in 1997 he was made a honorary professor at the university in recognition of his research work, much of it concerned with SIDS. He is also interested in the application of mathematics in medicine and has bred two mathematicians to help him with his work. His daughter, Kate, now uses mathematical models to study another complex and unpredictable organism - the stockmarket. His son, Robert, is a PhD student of combinatorix in Memphis. Professor Morris' favourite publication, a product of his children's summer vacation when they were students at Cambridge, is given below. He experienced his 15 minutes of fame following the successful appeal of Sally Clark against her conviction for murdering her two infant sons. The appeal was based on new evidence of staphylococcal infection causing death in the second child.

Morris KL, Morris RD, MorrisJA. The mathematical basis of sexual attraction. Medical Hypotheses 2002; 52: 288 - 296.

Narita, Masaaki

Masaaki Narita was born in Kobe, Japan in 1961. He went to Medical school in Hiroshima, Japan and did his postgraduate studies at the Kobe University School of Medicine. He was research associate at Washington University School of Medicine in the USA from 1994 to 1998. He became assistant professor at the University of Tsukuba in 1998. He is a pediatrician and a pediatric Neurologist as well. He is presently with the Institute of Basic Medical Sciences at the University of Tsukuba.

O'Meara Christine

Christine O'Meara is a communications specialist with the North Carolina Healthy Start Foundation in the southern U.S.A. In this capacity she is the campaign coordinator for the N.C. Back To Sleep Campaign for SIDS Risk Reduction, a national award-winning state impact public awareness and education campaign (2001). She also serves as project director of the Infant/Toddler Safe Sleep and SIDS Risk Reduction in Child Care (ITS-SIDS) Project, a statewide train-the-trainer initiative she developed in 2002. Over 10,000 licensed childcare providers have participated in the ITS-SIDS training. Chris has worked with lawmakers and the N.C. child care regulatory agency to affect policy change requiring licensed caregivers to adhere to safe sleep and SIDS risk reduction standards and is the lead ITS-SIDS trainer. As a child health advocate, Chris weaves tobacco-use prevention, cessation and secondhand smoke issues into her SIDS risk reduction efforts. Maternal and child health nurses, newborn nursery and Neonatal Intensive Care Unit staff, SIDS counselors, social workers, health educators and child care health consultants have participated in her SIDS-related in-service and workshops at hospitals, county health departments, child service agencies and state-wide, regional and national conferences. Chris incorporates her Anthropology and Public Health Education background into a creative use of social marketing and enjoys the challenge of maximizing limited resources to develop multi-media strategies aimed at reducing SIDS in English and Spanish speaking communities.

Richerson, George B.

George Richerson is Associate Professor of Neurology, Cellular & Molecular Physiology, and Neuroscience at Yale University School of Medicine. He graduated from Iowa State University with a B.S. in Aerospace Engineering, and then received his MD & PhD in Physiology & Biophysics from the University of Iowa in 1987. He completed a residency in Neurology at Yale University in 1991. He practices adult neurology, in addition to performing research and teaching. He performs his research on animals (primarily rats and mice), and focuses on the role of serotonin neurons in the control of breathing. He began this work because of its relation to diseases of the adult nervous system, such as sleep apnea and ALS, as well as because of its inherent scientific fascination. However, in recent years it has become clear that this work is highly relevant to understanding the pathophysiology of the subset of SIDS that is associated with abnormalities of the serotonin system.

Rognum, Torleiv

Torleiv Ole Rognum, baby boomer after the second world war born April 9, 1948.

Married with Elsa, four children and 1 grandchild. Several of the brothers and sisters of his grandfarther emigrated to Canada 100 years ago, and he has a second degree cousin in Edmonton. He is looking forward to meeting her this summer.

Dr Rognum studied medicine in Austria and Germany, and graduated in 1974. He defended his doctoral theses on cancer research (PhD) at University of Oslo in 1982. After training in pathology and paediatrics, he has been working in forensic medicine, University of Oslo, as associate professor from 1984, and as full professor since 1991. Since 1984 SIDS has been one important focus of his research interest. At his institute he now has a SIDS research group consisting of 4 medical doctors, 1 PhD researcher, 1 forensic expert and 3 technicians. Since 1986 he has been member of the Medical Advisory Board of the Norwegian SIDS Society, and for 8 years he was the leader of the board. He was the co-ordinator of the scientific programme of the Third SIDS International Conference in Stavanger in 1994, and was also co-responsible for the ESPID Conference in Oslo, 2003. From 2001 he is executive chairman of European Society for the Study and Prevention of Infant Death (ESPID). His vision is that researchers and health personnel concerned with SIDS will join the new international society for the study and prevention of infant death (ISPID), and that this organisation will function as the scientific advisory board of SIDS International. Such an approach will facilitate further fights against sudden infant death syndrome.

Roper, Janice

Janice Roper is the author of the highly acclaimed Dancing on the Moon, a picture book to help children and their parents through grief. Janice, mother of three daughters, lost her only son, Daniel C. Roper IV to SIDS in 1995. Janice and her oldest daughter Selena, now age 9, posed for the artist who painted the 14 beautiful oil paintings that make up Dancing on the Moon. Selena will participate in the workshop on sibling grief with her mother.

Sandvik, Oddbjorn

Dr. Sandvik, a psychologist, runs a private clinic in Norway. For the past 13 years he has been working in the area of grief support, For several years he was the leader of a nationwide grief support program in Norway. He has offered numerous courses and seminars on grief and grief support for volunteers and health professionals and has supervised and developed eduational programs for grief support volunteers. He has also co-authored a book about grief and has written numerous articles about it as well.

Sawaguchi, Toshiko

Toshiko Sawaguchi is a Japanese forensic pathologist educated in Japan by Prof. Akiko Sawaguchi and in Wales by Prof.Bernard Knight. It was the first opportunity for her to be interested in SIDS when she was involved in the Japanese National SIDS Project supervised by Prof. Hiroshi Nishida of Pediatrics about 10 years ago. She was working for SIDS in Wales, in Japan and in Brussels from the integrated prospect.

She was one member of International Child Care Practice Survey (ICCPS) by Dr.Tony Nelson and invited Prof. Andre Kahn into the Japanese National SIDS Project for the pathological and physiological linkage study for SIDS.

Shatz, Anat

Anat Shatz was born and grew up in Israel. She completed her medical training at the Hadassah Medical School of the Hebrew University, Jerusalem, with an M.D. Degree and completed a pediatric otolaryngology fellowship at the Children's Hospital, Birmingham, U.K., in 1991. She currently serves as a senior Ear Nose and Throat Surgeon at the Shaare Zedek Medical Center, Jerusalem, where she established and leads the Pediatric Airway and Sleep Center, a multidisciplinary center treating infants and children suffering from various breathing and sleep disorders, especially obstructive sleep apnea, congenital lesions, and infants at risk for the Sudden Infant Death Syndrome (SIDS). She is active in all aspects of pediatric otolaryngology, with special interest in the upper airways, particularly the endoscopic treatment of the upper airways and endoscopic sinus surgery in Cystic Fibrosis patients. Since 1988, she has been conducting research into pathogenetic mechanisms that could explain apnea episodes, upper airways' obstruction and sudden infant death. In 1999, Anat Shatz established The Israeli Foundation for the Study and Prevention of Sudden Infant Death and has been serving as its chairperson since then. She has published numerous articles in prestigious professional journals on various topics within pediatric otolaryngology, focusing on understanding of sleep disordered breathing in young children and on SIDS.

In 1999, Anat Shatz was elected to serve as Israel's representative to ESPID (European Society for the Study and Prevention of Sudden Infant Death). She organized and chaired the eighth ESPID International Conference held in Jerusalem, Israel. The conference theme was dealing with Prevention of Infantile Apnea and Sudden Infant Death.

She also holds a position of Senior Lecturer at the Faculty of Health Sciences, Ben-Gurion University in the Negev, an affiliate of Shaare Zedek Medical Center, Jerusalem. She is a member of the American Academy of Otolaryngology/Head & Neck Surgery and the American Society of Pediatric Otolaryngology.

She has developed a special relationship with Canadian researchers devoted to SIDS, built over many years of mutual visits, meetings at various scientific gatherings, exchange of ideas and participation in common projects.

For example, she attended an exclusive seminar organized in Montreal in October 2000, and used this opportunity not only to outline areas for future cooperation, but also to get to know Aurore Côté and her colleagues on a more personal basis, with much appreciation to their friendly attitude and kind hospitality.

Shotton, Heather

Heather Shotton is the Vice President and co-founder of Mikela's Miracles SIDS Connection. She worked for eight years with non-profit organizations in the area of American Indian health prior to co-founding Mikela's Miracles SIDS Connection. Since 2000 she has worked in the area of Sudden Infant Death Syndrome (SIDS). Heather has been responsible for the development and implementation of a national American Indian SIDS outreach and education program entitled "Preserving Our Heritage…Saving Our Babies". She has presented extensively on the topic of SIDS among American Indians, and has served as a consultant for tribes throughout the United States in this area of maternal and child health.

Heather received her Bachelor of Arts in Native American Studies from the University of Oklahoma in 1999, where she focused on tribal law, policy and development. She will be receiving her Masters in Human Relations in the summer of 2003, from the University of Oklahoma. Heather worked with the Association of American Indian Physicians for two years, and later joined the Native American Marrow Recruitment Project in 1999. Her work in the area of American Indian health has enabled her to develop excellent communications skills, program development experience, and a strong relationship with tribes and tribal health agencies throughout the nation. Heather also serves as an adjunct professor of Native American Studies at Seminole State College, located in Seminole, OK. She has extensive knowledge and understanding of American Indian culture and traditions, as well as program development, implementation and evaluation.

Takashima, Sachio

Sachio Takahima worked as a research fellow with Drs. D Armstrong and LE Becker in 1977-1978 in the Department of Pathology, The Hospital for Sick Children, Toronto. He noticed the importance of SIDS, and worked the neuropathological research of SIDS.

In 1979, he became Associate professor, Department of Child Neurology, Tottori University, Japan. He visited Toronto several times and continued to work on the pathogenesis of SIDS with Dr. LE Becker

Dr Takashima was Director, Department of Mental Retardation and Birth Defect Research, National Institute of Neuroscience, National Center of Neurology and Psychiatry, Japan (1987-1999). He held the International Symposium of SIDS with Australia and Neural transmission development with Canada and USA.

He became Director General, National Institute of Neuroscience, National Center of Neurology and Psychiatry (2000-2002). He is presently working for the persons with developmental disabilities as a medical doctor and Professor of International University of Health and Welfare. He and Dr. Y Ozawa are still continuing the SIDS study as a member of SIDS research project group of The Ministry of Welfare, Health and Labor in Japan.

Thach, Bradley

Dr Thach graduated from Princeton University, New Jersey in Biology and from Washington University in St. Louis, in Medicine. In 1973-1974, he was a post doctoral fellow in Canada at McGill University in Montreal. He is presently professor of Pediatrics at Washington University, in St. Louis. He won several awards including, most recently, the Anaheim Award for Sleep and Breathing Research in Infancy-2000 and the Alumni Faculty Award, Washington University, 2003. He is a Board member First Candle/SIDS Alliance-2003

He mentions his favorite cities for Research as being : Montreal, Canada and Bethesda, Maryland. His favorite past time is outdoor activities- (canoeing, hunting, hiking, fishing) and his great hobbies are Bird-watching, blacksmithing, making useful (??) things out of junk. His favorite people are Barbara, Elizabeth, Mary and Susanna Thach. His favorite dog is Liza Thach.

He loves old songs and quote "The Rock'n Pneumonia and the Boogy Woogy Flu" – from Huey Smith and the clowns as his favorite.

Tonkin, Shirley

Shirley Tonkin is a 4 th generation New Zealander. She was brought up in a country town, Stratford, whose most notable Medical Practioner was Dr Doris Gordon, a close family friend.

She qualified at Otago University and went into General Practice in Napier after marriage. A sudden infant death in her practice, which was put down to a rapid pneumonia raised her curiosity- as she had seen that baby the previous day and he was perfectly healthy.

She moved to Auckland where research facilities were available and was helped by the late Sir William Liley.

She joined the Department of Health and in her own time home visited nearly 200 parents whose babies had died. She worked on the parents' stories and it was obvious that stomach sleeping was dangerous. She also wondered why stomach sleeping was safe for some babies -but not for others. Her work on the individuality of babies has carried on from that.

She has always felt an affinity with Canada. Her father was wounded on the Somme in the first World War. He was taken to a Canadian Hospital and always said that the doctors there saved his life. She has a nephew living in Vancouver whom she will shortly visit and she has made several visits to Canada.

Van Eyk, Jennifer

Jennifer Van Eyk has been a Canadian Heart and Stroke Foundation Scholar (1996-2001) and Heart and Stroke Career Investigator (received 2001), in addition to receiving a Canadian Institutes of Health Research Investigator Award. In the last two years, she was the recipient of the three awards for research excellence, two from Queen's (Chancellor and Basmajian) and one provincial (Premier's). Dr. Van Eyk is a Fellow of the American Heart Association, a member of the editorial board of Circulation Research and guest editor for a series on Proteomics for that journal; and, she has joined the editorial board of Proteomics specifically as editor for Technical Briefs. She is co-editing with Dr. Mike Dunn "Genomic and Proteomic Analysis of Cardiovascular Disease: Molecular Mechanisms, Therapeutic Targets and Diagnostics" published in 2002. Her research combines physiology and proteomics to provide an in-depth analysis of the molecular basis for a variety of cardiac and skeletal muscle diseases ranging from myocardial stunning and heart failure to respiratory muscle injury in obstructive lung diseases and sepsis, and using this information to develop new serum biomarkers and potential therapeutic targets. Dr. Van Eyk's lab is now located at Johns Hopkins in Baltimore, Maryland. Here she is Director of The Johns Hopkins National Heart Lung and Blood Institute (NHLBI) Proteomics Center and Director of Bayview Proteomics Group.

She developed her passion for science because she wanted to address health issues for the old and the young. She has two daughters who were born in Edmonton.

Vege, Åshild

Åshild Vege was born in a small town at the southern coastline of Norway, but is living in Oslo, working at the University there. She has been engaged in SIDS research since 1992 and the close co-operation with the Norwegian SIDS society and the bereaved parents have meant very much to her.

This will be her first visit to Canada and she really look forward to experiencing the country's beautiful nature and make new friends.

Waters, Karen

Karen Waters is a Pediatrician and researcher, who commenced her research work in Sydney. In her clinical work she manages children who have breathing abnormalities, including apnoea, during sleep. Her research focus is on the control of breathing and hypoxia. Her research into SIDS is also taken from this perspective.

During her Post-doctoral fellowship in Montreal (Canada), a study was undertaken to examine cellular findings in the brains of infants who had died from SIDS, and her research in Sydney has continued to investigate that problem. She currently heads the Respiratory Support Service at the Children's Hospital at Westmead. Her research group works with projects to do with Sleep Apnoea and SIDS research, and includes examination of the consequences of exposing animal models to intermittent hypoxia.

Saturday July 3 Oral presentations

Professional Education and Reduce the Risk

Salon 15-16 H	10:30	How to alter Midwives actions when their beliefs are so imbedded in their own history?	Tanner, Margaret	Australia
	10:50	Nursing and Midwifery Knowledge, Attitudes and Practices relating to SIDS and the Reduce the Risk Messages in Queensland.	Young, Jeanine	Australia
	11:10	Evidence Based Education Can Reduce Infant Mortality	Jeffery, Heather	Australia
	11:30	The importance of a national consensus for the definition of SIDS and autopsy approach to unexpected infant death for Australian SIDS organisations.	Carey, Janet	Australia

Expanding Bereavement Services beyond SIDS
Chair: Hazel Brooke, Scotland and Paul Rusinko, United States

Salon 13-14 H	10:30	Supporting Bereaved Parents in New South Wales, Australia: A merged bereavement support service 3 years down the track.	Richardson, Ros	Australia
	10:50	SIDS organisations embracing new research frontiers	Richardson, Ros	Australia

Epidemiology and Risk factors - Chair: Bradley Thach, United States

Salon 12 S	13:30	Immunisation and sudden infant death syndrome: Results from GESID	Vennemann, Mechtild	Germany
	13:45	SIDS and vaccination using hexavalent vaccines. Results of the German study on sudden infant death (GeSID).	Bajanowski, Thomas	Germany
	14:00	SIDS in child care settings: 2001	Moon, Rachel	United States
	14:15	Do risk factors for SIDS differ with age at time of death?	Arnestad, Marianne	Norway
	14:30	Social Inequalities in Sudden Infant Death Syndrome	Schlaud, Martin	Germany

Classification of Sudden Infant deaths - Chair: Roger Byard, Australia

Salon 3 S	13:30	Evaluation of the death scene - development of a scoring system to operationalise observations	Sveum, Lisbeth	Norway
	13:45	Sudden & Unexpected death in Infancy. Reflections of a Pediatric Pathologist	Franciosi, Ralph	United States
	14:00	Classical SIDS, do they exist?	Stray-Pedersen, Arne	Norway
	14:15	Reclassification of post neonatal infant mortality	Beal, Susan	Australia
	14:30	Continuous monitoring of cot death cases and controls in the Netherlands	Engelberts, Adele	The Netherlands

Addressing Risk Factors

Salon 13 -14 H	13:30	Perspectives on Infant Care Practices and Sudden Infant Death Syndrome (SIDS) in a selected population of women in Saskatchewan	Mpofu, Debbie	Canada
	13:50	Infant care practices: Are Queensland parents using the Reduce the Risk recommendations?	Young, Jeanine	Australia
	14:10	Reducing the Risk of SIDS by Educating Retailers	Weber, Maxine	Australia
	14:30	Spreading the Reduce the Risk message through innovative means	Epstein, Joyce	United Kingdom
Salon 6 I	13:30	Back to Reality - An Indigenous Model for a SIDS Response	De Joux, Raeleen	New Zealand
Salon 15 I	13:30	Publish or Perish: How to Get a SIDS Publication Profile	Tipene-Leach, David	New Zealand
Salon 16 I	13:30	One Foot in the Door - A Cultural Icon - A Relevant Practice today	Hopa, Pauline	New Zealand

Caring for our Volunteers; New Ideas, New Practices - Chair: Betty McEntire, United States

Salon 15-16 H	15:30	Quality assurance programme for the volunteer bereavement support of the Norwegian SIDS Society	Kalstad, Trine	Norway
	15:50	Arranging the Peer Supporter/Client Match - the challenge for the Counselling Manager	Taylor, Gregory	Australia
	16:10	Counselling service for volunteer bereavement support	Kalstad, Trine	Norway
Salon 6 I	3:30	Ending the Journey	Gage, Randi	Canada

Sunday July 4

Culturally based Efforts to Reduce the Risk I

Salon 3 H	10:30	Moving from Outreach to Partnership: Culturally Competent Risk Reduction Efforts	Bronheim, Suzanne	United States
	10:50	The process of developing and validating competencies of Pacific Community SIDS Educators	Finau, Eseta	New Zealand
	11:10	Djäka Yothu1: SIDS education in Northern Territory indigenous communities	Lambert, Margaret	Australia
	11:30	Reclaiming Traditional Values and Practices to Ensure Maori Babies Get the Best Start in Life	De Joux, Raeleen	New Zealand

Fathers, the Forgotten Bereaved Parent - Chair: Barbara Heather, Canada

Salon 13 -14 H	10:30	Love Your Woman, Because It Ain't Nobody's Fault: The Sudden Infant Death Syndrome (SIDS) Experiences of Maori Men	Edwards, Shane	New Zealand
	10:50	Moving into their Comfort Zone Support for Fathers following the Death of a young child	Carlin, Kevin	Australia
	11:10	Local support groups for men experiencing grief	Mathiesen, Trond	Norway

Salon 5 I	10:30	Innovative Strategies for Reducing SIDS Among American Indian/Alaska Native Populations	Rosenman, R. Mona	United States

Salon 6 I	10:30	SIDS & Kids Queensland: Working Together Towards A Better Tomorrow	Hebert, Sonia	Australia

Salon 15 I	10:30	Creating Communities of Care with Aboriginal Families: A relational and shared-values workshop	Hanson, P. Gaye	Canada

Salon 6 I	10:30	SIDS Risk Reduction – Tobacco as a Risk	Morris, Cynthia	Canada

Sleeping Environment - Chair: Anatz Shatz, Israel

Salon 3 S	13:30	Micro-environment and Sleep Characteristics in two-month-old Infants	Kelmanson, Igor	Russia
	13:45	Trends in Infants Bed-sharing with Parents in Oslo, Norway	Stray-Pedersen, Arne	Norway
	14:00	Infant-Parent Bed Sharing Practices: Cross-Cultural Comparisons	Hauck, Fern	U.S.A.
	14:15	Co-sleeping and Sudden Unexpected Death in Infancy. A Case-control study.	Tappin, David	United Kingdom
	14:30	Heavy Bedding is a Risk Factor for SIDS	Schlaud, Martin	Germany

Cardiorespiratory Physiology - Chair: Jean-Paul Praud

Salon 5-6 S	13:30	Prolongation of QT Intervals in Victims of Sudden Infant Death Syndrome : A Polysomnographic Study	Franco, Patricia	Belgium
	13:45	Home monitoring: evaluation of impedance and pulse oximetry alarms.	Piumelli, Raffaele	Italy
	14:00	Obstructed Breaths in the Terminal Event in Sudden Infant Death Syndrome (SIDS)	Pylipow, Mary	United States
	14:15	The impact of blood oxygen levels on submental and diaphragmatic EMG during obstructive apneas in infants	Wulbrand, Henning	Germany
	14:30	Comparison of hypoxic arousal responses between term and preterm infants.	Horne, Rosemary	Australia
	14:45	The efficacy of swaddling in infants who cry excessively; a randomized controlled trial	Sleuwen, Bregje	The Netherlands

Building a Caring Community for the Grieving family - Chair: Margaret Robinson, Canada

Salon 15 H	13:30	Responding when a baby dies - multi-agency protocols	Deri-Bowen, Ann	England
	13:45	Technology Aids in Grief Support Services	Griffin-Hilbert, Chelsea	United States
	14:00	Follow-up routines after traumatic events and deaths: Routines on the Web site	Kalstad, Trine	Norway
	14:15	The 'Parenting Dream': A Counselling Model for SIDS Families	Nolte, Judi	Australia

Culturally Based Efforts to Reduce the Risk II

Salon 16 H	13:30	Maori SIDS: The Life History Project	McManus, Verne	New Zealand
	13:50	Maori Collaborative Research With Maori Communities on Sensitive Issues	Edwards, Shane	New Zealand
	14:10	Sleeping With The Enemy - Working Smarter, Not Harder	Haretuku, Riripeti	New Zealand

Parent Session Chair: Deb Boyd, United States

Salon 2 PAR	13:30	Changing local activity of bereavement support: Regional family seminar	Kalstad, Trine	Norway
	13:45	Burial guide - an information booklet to parents who have lost a small child	Schrader, Line	Norway
	14:00	Conflicting Advice and Parental Guilt	Cormack, Helen	United Kingdom
	14:15	Adolescents in grief - Information booklet for adolescents in grief and their social network	Schrader, Line	Norway
	14:30	Support for extended families, especially Grandparents, after the death of a child. Alliance of Grandparents, A Support in Tragedy	Graben, Sandra	United States

Sensitive Issues, Sensitives Workers: The death Scene and Parental Grief
Chair: Dennis Caulfield, Canada

Salon 13-14 H	15:30	The SIDS Careworker Study	Tipene-Leach, David	New Zealand
	15:50	Training programme for professionals supporting bereaved parents: Explaining the need for post mortem examination and encouraging consent for the retention of tissue and organs following a child death	Deri-Bowen, Ann	England
	16:10	How to approach parents at the death scene using a police interview model	Sveum, Lisbeth	Norway

Tobacco Smoking Cessation in Pregnancy
Chair: Cathy Melvin, United States and Carol Sutherland-Brown, Canada

Salon 15-16 H	15:30	Upholding Traditional Beliefs While Incorporating Science-Based Research	Griego, Dolores	United States
	15:50	Patupuauahi	Hopa, Pauline	New Zealand
	16:10	Motivational Interviewing - A tool for addressing smoking cessation across cultures	Minett, Ingrid	New Zealand
	16:30	Better Practices in Smoking Cessation During Pregnancy & Postpartum	Jategaonkar, Natasha	Canada

What Do the Numbers Mean? What We Did That Worked?				
Salon 6 I	15:40	Tamariki Maori Coordination	Pompallier, Tania	New Zealand
	16:00	A decade of Maori SIDS: What Do The Numbers Mean?	Tipene-Leach, David	New Zealand
	16:20	Maori SIDS: A New Zealand Success Story	Tipene-Leach, David	New Zealand
	16:40	The Maori SIDS Prevention Programme: What Did We Do That Worked?	Haretuku, Riripeti	New Zealand

Tuesday July 6

Salon 15 I	10:30	Soul Stories and Wise Ways	Miller, Denise	Canada

Salon 16 I	10:30	Ensuring Good Business with Aboriginal	Dahlstrom, Josie	Australia

Posters

All poster can be viewed in the Foyer, on the Assembly Level

****The number, to the left of the poster title, indicate the poster board****

Saturday, July 3

Respiratory Physiology and Pathophysiology - Chair: Tracy Carbone

S 13 :30 – 15 :00	1	Bedsharing and the infant's thermal environment in the home setting	Baddock, Sally	New Zealand
	2	Cardiac Autonomic Responses To Auditory Challenges In Swaddled Infants	Franco, Patricia	Belgium
	3	Autonomic activity in SGA infants sleeping prone and supine	Galland, Barbara	New Zealand
	4	Prenatal cigarette smoke exposure leads to aberrant breathing patterns during hypoxic and hyperthermic challenges in newborn rats	Hasan, Shabih	Canada
	5	Comparison of heart rate responses to nasal air-jet stimulation between term and preterm infants	Horne, Rosemary	Australia
	6	Effects of sleep state and sleeping position on blood pressure in infants in the first 6 months of life	Horne, Rosemary	Australia
	7	Surveillance study Apparent Life Threatening Events (ALTE) in the Netherlands	L'Hoir, Monique	The Netherlands
	8	Grandparents and other senior family members and Back to Sleep: Knowledge and Behavior	Moon, Rachel	United States
	9	Web network against SIDS and ALTE	Parmigiani, Stefano	Italy

Risk campaign - Chair: Manisha Witman

S 13 :30 – 15 :00	10	Risk factors for sudden infant death syndrome, ten years after the introduction of the campaign to reduce the risk.	Alm, Bernt	Sweden
	11	Unusual and unsafe sleeping environment in sudden infant death victims in Quebec	Côté, Aurore	Canada
	12	The influence of swaddling on sleep characteristics and arousability in healthy infants	Franco, Patricia	Belgium
	13	Expectations and Organisations of Parents' Associations	Gabbi, Giampaolo	Italy
	14	Cross Sectional, Population Study of Sleeping Positions for Term or Near Term, Well Babies In Hospitals in The State of NSW, Australia	Jeffery, Heather.	Australia
	15	Cot Mattresses as Reservoirs of Potentially Harmful Bacteria and the Sudden Infant Death Syndrome	Jenkins, Richard	United Kingdom
	16	Eight years after:an evaluation of SIDS awareness in the Tuscany Region	Piumelli, Raffaele	Italy
	17	Sudden Infant Death Syndrome (SIDS) And Infant Care Practices	Sankaran, Koravangattu	Canada
	18	Saliva: does it provide protection against Sudden Infant Death Syndrome?	Weiss, Peter	United Kingdom

Monday, July 5

Bereavement, Support and Counseling - Chair: Oe'livia Chasse

H 10:00 - 11:00	19	Maori Grieving After the Loss of a Baby to SIDS	Clark, Eileen	New Zealand
	20	Survey of Bereaved Families in Japan	Fukui, Stephanie	Japan
	21	The SIDS Manual 2004	Kalstad, Trine	Norway
	22	Taking care of brieving parents at Naitre et Vivre France	Morinay, Myriam	France
	23	A support programme for anxious parents	Waite, Alison	United Kingdom
	34	Treasured Babies Program. Supporting Best Perinatal Crisis Bereavement care in Hospital	Passey, karen	Australia

Tobacco and Smoking

H 10:00 - 11:00	49	Tobacco Talk for Two: A Prenatal/Postnatal Group Tobacco Reduction Toolkit	Foreman, Gail	Canada
	50	Smoking Cessation During Pregnancy: A Coordinated Regional Approach	Jickling, Sheri	Canada
	51	Hard Cases - changing smoking in challenged families	Metzger, Barbara	New Zealand

Pathology - Chair: Åshild Vege

S 13:30 – 15:00	24	Analysis of villous vasculature in placentae from sudden infant death syndrome (SIDS) infants with and without intrauterine growth restrction (iugr).	Ansari, Tahera	United Kingdom
	25	Investigation of sudden infant death syndrome and intra uterine growth restricted placentae using ho-1 & ho-2, vegf and cd68.	Ansari, Tahera	United Kingdom
	26	Identification of villous membrane apoptosis in sudden infant death syndrome (SIDS) and intra uterine growth restricted (iugr) placentae using m30 and bcl2.	Ansari, Tahera	United Kingdom
	27	Real-time quantitative PCR assay for the detection of Helicobacter pylori - no association to sudden infant death syndrome	Bajanowski, Thomas	Germany
	28	Detection of specific antibodies in cord blood and infant saliva to staphylococcal toxins implicated in sudden infant death syndrome (SIDS)	Harrison, Linda	England
	29	Epidemiology of Apparent Life-Tthreatening Events (ALTE) - is there a relation to SIDS risk factors?	Kiechl-Kohlendorfer, Ursula	Austria
	32	Crib Death: Cardiac Sampling and Study of the Conduction System	Matturri, Luigi	Italy
	35	Helicobacter Pylori and SIDS	Stray-Pedersen, Arne	Norway
	36	IL-6 and risk factors in SIDS	Vege, Åshild	Norway

Neuropathology - Chair: Hannah Kinney

S 13:30 – 15:00	37	Postnatal metabolic and neurochemical development of brain stem respiratory nuclei in rats: Potential implications for SIDS	Wong-Riley, Margaret	United States
	31	Sampling Techniques of the Brainstem in Sudden Infant Death Syndrome (SIDS) and Sudden Unexpected Perinatal Death	Matturri, Luigi	Italy
	30	Correlation between the NMDA receptor and neuronal death in the SIDS medulla.	Machaalani, Rita	Australia
	33	Experimental Simulation of the Geomagnetic Activity Correlated	Persinger, M.	Canada

Sudden Unexpected Deaths - Chair: Marianne Arnestad

S 13:30 – 15:00	38	SIDS is not associated with a temperature dependant Brugada syndrome mutation.	Arnestad, Marianne	Norway
	39	Sudden infant deaths in car seats	Deschenes, Marianne	Canada
	40	Sudden unexpected death in children 2 to 18 years	Kobulnik, Jeremy	Canada
	41	Sudden Infant Death While Being Breastfed	Krous, Henry	United States
	42	Sudden Infant Death After Being Found Awake	Krous, Henry	United States
	43	Sudden Unexplained Death in Childhood (SUDC). A Report of 34 Cases.	Krous, Henry	United States
	44	Changes of concepts and diagnostic shifts over time	L'Hoir, Monique	The Netherlands
	45	Respiratory malacia: possible cause of sudden death in infancy and early childhood?	Rohde, Marianne	Denmark
	52	Increase pulmonary MMP's in hypoxic Piglets during Ressuscitation with 100% Oxygen	Munkeby, berit	Norway

10:30 - 12:00 **Salon 15-16**

Health and Allied Professional stream **Professional Education and Reduce the Risk**

1. How to alter Midwives actions when their beliefs are so imbedded in their own history?

Margaret E Tanner, Royal North Shore Hospital, Lane Cove., NSW

Introduction: On entering employment in a well-established Maternity unit in 2001, it was observed that staff were placing infants on their sides and occasionally prone. The latest Reduce The Risk Factors (RTR's) of SIDS were introduced in 1996 and endorsed by the Australian Government. The recommended positioning of the infants was supine only, unless medically indicated. Therefore, the question was, why then were these recommendations not being followed within this unit? The Early Discharge program (EDP) Midwife had also noticed this enigma and facilitated the SIDS association to conduct an in-service for the unit. The staff that attended this in-service were from various areas, such as the Post Natal unit, Neonatal Intensive Care Unit (NICU), Paediatric ward and the Community. The Midwives within the Maternity unit were surveyed about their understanding of the RTR's, their own beliefs of the best position for infants to sleep and why they had those beliefs. The initial response demonstrated that there was an awareness of the RTRs although there were reservations regarding the fear of choking if a baby was placed in the supine position.
Action: The undertaking was to carry out in-services in hope to cover as many staff as possible. Attendees were from NICU, the Post Natal ward, Midwifery students and Labour ward. In-services were even conducted for evening and night duty staff. Other media was also introduced to support the lectures.
Outcome: A change of practice occurred with the majority of midwives 93% placing infants in the supine sleep position. This change has taken the whole 8 months to achieve a positive change.
Discussion: Change is not an easy thing to achieve. But with consistent education and support, the use of easy to understand tools and ongoing education I believe this has been achieved. Change is a slow process.

2. Nursing and Midwifery Knowledge, Attitudes and Practices relating to SIDS and the Reduce the Risk Messages in Queensland.

Jeanine Young, Research, Royal Children's Hospital; School of Nursing, Queensland University of Technology; School of Population Health; Peter O'Rourke, School of Population Health, University of Queensland, Brisbane, Queensland

Background: Queensland has one of the highest rates of Sudden Infant Death Syndrome (SIDS) in Australia. Evidence suggests that many nurses have knowledge deficits about SIDS risk factors; some disagree with current recommendations, and these deficits impact upon nursing practice and information provided to parents. Despite the potential impact of nurses in educating parents about optimal infant care practices, the understanding, acceptance and implementation of 'Reduce the Risk' (RTR) messages by nurses in Queensland had not been previously examined.
Aim: This study investigated nursing and midwifery knowledge, attitudes and practices relating to SIDS, the RTR messages and parent education in Queensland.
Methods: A cross-sectional survey design was used to determine nursing and midwifery knowledge, attitudes and practices relating to SIDS risk factors and RTR messages in three groups of nurses and midwives from 1) acute neonatal and paediatric care; 2) antenatal, perinatal and postnatal care; 3) community child health throughout Queensland.
Results: Completed surveys were returned from 934 (76%) nurses and midwives. Most nurses were aware babies should be: placed on their backs to sleep, 664 (72%); feet to foot if in a cot, 736 (80%); and kept smokefree, 890 (98%); however only 563 (61%) correctly identified current RTR messages. Considerably few respondents, (320, 35%), correctly identified the recommended sleep position for babies with mild gastro-oesophageal reflux. Only 548 (59%) nurses agreed with the recommended sleep position for babies with reflux, and 175 (19%) indicated supine positioning increases the risk of aspiration. Of the respondents, 669 (74%) provided parent education about SIDS in daily care and discharge planning. Only 563 (62%) nurses advised parents to use recommended supine positioning for healthy babies, and 306 (36%) advised parents to place babies with reflux in the recommended sleep position.
Conclusions: Many nurses and midwives have knowledge and attitude deficits relating to SIDS risk factors and RTR messages, and these deficits impact upon advice provided to parents. This state-wide study has made recommendations to state health agencies for the development of effective education programs to impact positively on nursing and midwifery knowledge and ultimately parental practices relating to SIDS and the RTR messages.

3. Evidence Based Education Can Reduce Infant Mortality

H. Jeffery, RPA Newborn Care, Royal Prince Alfred Hospital & University of Sydney, Australia, Sydney, NSW
M. Kocova, F. Tozija, D. Gjorgiev, International Project Unit, Ministry of Health, Skopje, Republic of Macedonia, Skopje,
K. Foster, J. Polverino, RPA Newborn Care, Royal Prince Alfred Hospital, Sydney, Australia, Sydney,
D. A. Hill, Consultant in Medical Education, Sydney,

Background: The Perinatal Mortality Rate (PMR) in The Republic of Macedonia is one of the highest in Europe. The World Bank supported the appointment of a consultant (HEJ) to head a local team to address the problem. Education was given priority in a strategic plan to build the capacity of the Macedonians to develop systems to implement best evidence practice into the perinatal health system .
Objectives: Description of the development, implementation and evaluation of the education intervention.
Methods: Curriculum development was based on a dynamic needs assessment. Learning objectives were defined and interactive, small group teaching methods were introduced with regular formative assessment. Twenty-five Macedonian doctors and nurses participated in a four to six month training program at a tertiary perinatal unit in Sydney (Phase 1). Australian teams conducted four, two week education modules for a further thirty-six doctors and nurses in a purpose built continuing education centre in Macedonia (Phase 2). The Phase 1 trainees conducted 8 modules for their colleagues in Skopje (Phase 3). The intervention was evaluated by trainee reaction, assessment of competence, changes in hospital practice and a before (1997-99) and after (2000-01) comparison of PMR.
Results: Positive responses to the program exceeded 80 per cent, 139 doctors and nurses were certified as competent by performance based assessment methods. Introduction of evidence based practice in the 15 participating hospitals (93% all births) was verified in six key areas of neonatology. The PMR fell significantly from 27.5 to 21.5 per 1000 births (RR 0.79, CI 0.74,0.86). The Early Neonatal Death rate in babies > 1000 grams fell by 36 per cent.
Conclusions: The educational intervention has built on the capacity of Macedonians to introduce best evidence practice and improve outcomes in the area of Perinatology. Sustainability is envisaged by the "train the teachers" approach and concurrent strengthening of the infrastructure and organizational framework. The model has potential applicability to health provider education and reducing SUDI.
Supported by the International Project Unit, Ministry of Health, Macedonia with IDA Credit, World Bank.

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4. The importance of a national consensus for the definition of SIDS and autopsy approach to unexpected infant death for Australian SIDS organisations.

Janet Marie Carey, SIDS and Kids, Weston, ACT

The lack of a standard definition of SIDS in Australian forensic pathology and the variation of infant autopsy protocols across the nation has concerned the Australian SIDS organisations for a number of years and led SIDS and Kids to host a national Pathology Workshop aimed at creating consistency nationally, and ultimately on an international level.
Uniformity in definitions would allow bereaved families to have their child's death defined consistently in any state of Australia, rather than being determined by their geographical location.
SIDS organisations would have access to dependable and credible statistical data upon which to base the SIDS research that they fund, removing any corruption of information through the definitional process.
This presentation will outline the impact a standardised approach would have on SIDS organisations, coronial inquiries, and the validity of research conducted using Australian data. It will also discuss the effect that a varied approach has on bereaved families.

10:30 - 11:00 **Salon 13-14**

Health and Allied Professional stream **Expanding Bereavement Services Beyond SIDS**

1. Supporting Bereaved Parents in New South Wales, Australia: A merged bereavement support service 3 years down the track.

Ros RICHARDSON, SIDS AND KIDS NSW, CAMPERDOWN, New South Wales

The merger of the SIDS(Sudden Infant Death Association) and SANDS (Stillbirth and Neonatal Death Support Inc) in New South Wales in 2001 saw both the end and beginning of era's in the support of bereaved families. Both organizations recognized some 2 years before the merger that the continuity of their services lie in pooling their considerable resources and developing services for a wide range of families. Know known as SIDS and Kids NSW, the organisation provides support for families who experience the death of their baby or child during pregnancy, birth and infancy. This includes the experiences of miscarriage and early pregnancy loss, stillbirth, neonatal and infant death, Sudden Infant Death Syndrome, and the death of a child up to 6 years of age.
New South Wales is Australia's highest populated state, with approximately 88,000 births per year. SIDS and Kids NSW has the potential to provide support services for over 1000 families who experience the death of their baby or young child within our support brief per year.
The role of the SIDS and Kids NSW Health Promotion Department is to create and initiate core services of the organisation in the activity areas of Bereavement Support, Education, Training, Advocacy and Special Projects. This paper will provide an overview of our services.

2. SIDS organisations embracing new research frontiers

Ros RICHARDSON, SIDS AND KIDS NSW, CAMPERDOWN, New South Wales

In November 2001, following the merger of SIDS (Sudden Infant Death Association) and SANDS (Stillbirth and Neonatal death Support Inc) in NSW, the first SOS Forum was held. The SOS Forum, (SIDS and Kids Australia focusing on Stillbirth), sought to focus on the unsolved problem of stillbirth, to explore what is known, and what more we need to know to improve investigation and prevention mechanisms. The merged organisation of SIDS and Kids NSW now provides support for families who experience miscarriage and early pregnancy loss, stillbirth, neonatal and infant death, SIDS and death of a child up to age 6.
Because of it's great success, the SOS Forum sought to replicate the SIDS community-based approach to facilitating research. SIDS organisations have historically been very successful in bringing together relevant scientists, researchers and clinicians and challenging them to unravel the mystery of Sudden Infant Death Syndrome.
With the reduction in SIDS deaths, we are proud and grateful that one of the smallest groups of families we now support are those who have experienced the death of their baby through SIDS. This is due at least in part to the work of SIDS organisations in provoking and facilitating research in the area. Sadly however many babies are stillborn, and many of these deaths remaining unexplained. To be as successful in this new research frontier is now the challenge.
The SOS Forum met for the second time in November 2002, with a focus on the role of Pathology in the clinical investigations of stillbirth. This paper will provide outcomes from the two SOS Forums conducted, and on current plans and proposed activities for the future.

13:30 - 15:00

Scientific stream **Epidemiology and Risk factors** **Salon 12**

1. Immunisation and sudden infant death syndrome: Results from GESID

Mechtild Vennemann, M. Findeisen, T. Bajanowski, B. Brinkmann, G. Jorch, E. A. Mitchell. Institut für Epidemiologie und Sozialmedzin, Universitätsklinikum Münster, Münster,

OBJECTIVE: To investigate whether the immunisation increases the risk of Sudden Infant Death Syndrome (SIDS), after adjusting for potential confounders. DESIGN: A three year, population based, case-control study was conducted in half of Germany, consisting of 333 cases and 998 controls. Immunisation status was collected from the parental interview and the paediatric records. RESULTS: Immunisation was associated with a lower risk of SIDS (odds ratio 0.41, 95% confidence interval 0.28 to 0.59). After adjusting for potential confounders and factors including the sleep environment the risk of SIDS was still lower for immunised children (0.54 (0.30 to 0.99)). 41 children died in the 14 day period after immunisation compared with 183 controls immunised in the 14 days prior to the interview. (adj. OR 0.71, 04 to 1.3) CONCLUSIONS: Immunisations are not a risk factor for SIDS in Germany and the direction of the association is rather towards protection than risk.

2. SIDS and vaccination using hexavalent vaccines. Results of the German study on sudden infant death (GeSID).
Thomas Bajanowski, University Essen, Essen; Mechtild Vennemann, Trude Butterfaß-Bahloul and Bernd Brinkmann, University of Münster, Münster; Gerhard Jorch, University of Magdeburg, Magdeburg,
In October 2000 two new hexavalent vaccines - Hexavac (Aventis Pasteur) and Infanrix Hexa (GlaxoSmithkline) were introduced in Germany. Both vaccines protect infants from poliomyelitis, diphtheria, tetanus, pertussis, haemophilus influenza type B, and hepatitis B. 8.7 million doses were sold and about 3 million children were vaccinated until April 2003 in the European Union. In the last year of the GeSID (November 2000 - October 2001) 130 cases of infant death were investigated using an extended autopsy protocol including histology, toxicology, microbiology, virology. As a part of the parental interview data on vaccination dates and antigens were collected. In addition data on symptoms of illness during the last 7 days and 24 hours before death and medications were collected. 12 infants died within 14 days (12 SUDI, 11 SIDS) and 4 deaths (all SIDS) were observed within 2 days after vaccination. Only in 6 of these infants the physicians confirmed that a hexavalent vaccine was used while in the remaining cases other types of vaccines had been used. The circumstances of death of the six cases who got hexavalent vaccinations prior to death are decribed in detail. The expected number of SIDS for the study region in Germany within 2 and 14 days after vaccination was calculated to be 2.1 respectively 14.9. These figures demonstrate that there is no association between SIDS and vaccination using hexavalent vaccines.

3. SIDS in child care settings: 2001
Rachel Y. Moon and Bruce M. Sprague, Children's National Medical Center, Washington, DC

BACKGROUND: 20% of SIDS in the 1990's occurred in child care settings. This is much higher than the 8% expected from Census Bureau data. Factors associated with child care SIDS included older age, Caucasian race; older, more educated mothers, and unaccustomed prone position. Since these findings, much emphasis has been placed on promoting safe sleep environment in child care. METHODS: We conducted a retrospective review of all SIDS deaths in 9 US states in 2001. Information regarding demographics, SIDS risk factors, and child care arrangements were collected and analyzed. Deaths occurring in child care were compared with deaths occurring during parental care. RESULTS: Of 572 deaths, 88 (15.4%) occurred in child care settings. Of these child care deaths, 32.9% occurred in family child care homes, 21.5% in child care centers, 21.5% in relative care, and 19% with a nanny/babysitter at home. Infants in child care were more likely to be older (137.4 vs. 95.3 days, $p<0.0001$), Caucasian (52.1% vs. 38.7%, $p<0.0001$), and with mothers who were more educated ($p<0.0001$). Infants in child care were more likely to be sleeping in a crib (37.1% vs 23.3%, $p<0.0001$) and less likely to be sleeping on a sofa (4.8% vs 11.2%, $p<0.0001$) or a bed (37.1% vs. 51.1%, $p<0.0001$). There was no difference in sleep position found, placed, or usual in child care or home deaths. Approximately 50% of SIDS deaths in both settings were found prone. 33% of child care deaths and 41.1% of home deaths were associated with unaccustomed prone. CONCLUSIONS: While the proportion of SIDS deaths in child care has declined, it still remains high at 15.4%. Demographic factors associated with child care deaths have not changed. However, infants in child care are more likely to be sleeping on a safe sleep surface and no more likely to be placed or found prone. Educational efforts with child care providers should continue. In addition, there may be other, yet unidentified, factors in child care that place infants in those settings at higher risk for SIDS.

4. Do risk factors for SIDS differ with age at time of death?
Marianne Arnestad, Åshild Vege, and Torleiv Ole Rognum, University of Oslo, Oslo

The SIDS diagnosis is currently under discussion; an approach suggested with different categories of SIDS defined among others by age at death. SIDS is currently defined as the sudden and unexpected death of an infant less than 12 months of age, it is however recognised that such deaths also occur in small children. The objective was to study whether risk factors for SIDS differ with the age at time of death in infants and small children. Subjects and method: 203 cases of sudden unexpected and unexplained deaths in infants and small children were divided into 3 age groups: 1-6 months (age peak of SIDS), 6-12 months and 12-36 months. Data on epidemiological risk factors were obtained from questionnaires to parents in southeast Norway 1984-1998. As the epidemiological pattern for SIDS changed after the "back to sleep" campaign in 1990, risk factors were also compared before and after this time. Results: 147 cases <6 months, 33 6-12 months and 21 over 12 months were identified. Before1990 80% of cases were under 6 months of age, while this proportion fell to 62% afterwards. There was no difference between the age groups in being found prone; this was seen for ~90% of all cases prior to 1990 and ~70% thereafter. Co-sleeping at time of death appeared after 1990; only seen in infants <6 months of age. Being found with the face covered or body wrapped in bedclothing was significantly more frequent in the 6-12 months age group ($p<0.01$). Fever in the days before death on the other hand, was significantly more frequent in the age group 12-36 months ($p=0.02$). The well known sex distribution in SIDS with 2/3 males was less pronounced after 6 months of age, and the frequency of maternal smoking during pregnancy less pronounced after 12 months of age. Other known risk factors for SIDS, such as low birthweight, young maternal age, multiparity and signs of slight illness (except fever) prior to death showed no differences between the age groups. CONCLUSION: Co-sleeping is only a risk factor in the first 6 months after birth, whereas prone position is a risk factor in all age groups.

5. Social Inequalities in Sudden Infant Death Syndrome
Maren Dreier, Hannover Medical School, Hannover; Martin Schlaud, Robert Koch Institute, Dept. of Epidemiology and Health Reporting, Berlin; Mechtild Vennemann, University of Muenster, Dept. of Epidemiology, Muenster,

Background: The reduction in the incidence of Sudden Infant Death Syndrome (SIDS) since the identification of prone sleeping as a main risk factor has been followed by an increase in the relative importance of poor socio-economic status as a risk factor. Aim: To study the role of social strata and their indicators qualification, income and occupa-tional status as risk factors for SIDS and to analyse associations between common risk factors for SIDS and social strata. Methods: Data from the German study "Sudden Infant Death Syndrome", conducted 1998-2001 as a 1:3 matched case-control-study. Cases: n=328, controls: n=983. Social status was determined with the Scheuch-Winkler-Index, derived from three indicators: qualification, income and occupational status. Crude and adjusted odds ratios (ORs) were obtained by conditional logistic regression. Results: 50.9% of cases belonged to the lowest social class.The adjusted OR for SIDS of the lowest social stratum was 9.1 (95% CI 3.1,27.0). While the adjusted ORs of the social indicators poor qualification (OR 7.5, 95% CI 2.6,21.7) and low occupational status (OR 2.0, 95% CI 1.0,4.2) were significantly lower than the crude ORs, the OR of low income remained almost the same level after adjustment for SIDS- risk factors (OR 21.6, 95% CI 6.3,73.9). The risk of low qualification was mainly reduced by the following factors: low maternal age, maternal smoking, use of prone sleeping position and not breast feeding. Conclusions: Low socio-economic status is an important risk factor for SIDS. The association between qualification and SIDS can mainly be explained by known risk factors, not only by younger aged mothers but especially by health-relevant behaviour. In contrast, low income seems to represent a more independent risk factor.
References: Winkler J. (1998). Die Messung des sozialen Status mit Hilfe eines Index in den Gesundheitssurveys der DHP. Schriftenreihe des Robert-Koch-Institut 1/98. MMV Medizin Verlag, München, 69-74.

13:30 - 15:00

Scientific Stream Classification of sudden infant deaths Salon 3

1. Evaluation of the death scene - development of a scoring system to operationalise observations
Lisbeth Sveum, Marianne Arnestad, Åshild Vege, Torleiv Ole Rognum, Institute of Forensic Medicine, Univ. of Oslo, Oslo

Voluntary death scene investigations in cases of sudden unexpected death in infants and small children (SUDI) (0-3 years old) have been performed in southeast Norway since September 2001. The project ends March 2004. The aim of this preliminary study was to test a scoring system developed to evaluate the information gathered form the death scene. Material and method: The study includes death scene investigation with video recording of all cases, as well as a questionnaire to all families and 3 controls matched for age, sex and place of birth. During the last year of the study sleep scene investigations for control infants have also been included. In this preliminary study 33 SUDI cases were compared with 33 controls based on questionnaire data and analysis of video recordings for variables connected with sleeping environment for the last sleep in the SUDI cases and reference sleep in the controls; sleeping position when found, firmness of mattress, number of layers of clothing, type of duvet used, room temperature and occurrence of co-sleeping. Each variable was then scored according to presumed increased risk, a score of one giving no presumed risk and a maximum score of four indicating high risk; 20 being the combined worst case score. Results: As expected the scoring system disclosed higher risk scores for the SUDI group than for the controls. Significant differences were found for prone sleeping, co-sleeping, sleeping on a soft mattress, sleeping in two ore more layers of clothing, and sleeping with a thick duvet. The total score was significantly higher than in the SUDI groups (mean score 10.1) than in the controls (mean score 6.5) ($p<0.01$). Conclusion: Application of a risk score system helps us operationalise observations from the death scene. The approach may aid comparison of the results from different studies.

2. Sudden &Unexpected death in Infancy. Reflections of a Pediatric Pathologist
Ralph Anthony Franciosi, Children's Hospital and Medical College of Wisconsin, Milwaukee, Wisconsin

The exposure of pediatric pathologists to infants that die suddenly and unexpectedly is limited because the death usually occurs out of hospital. The responsibility for determining the cause and manner of these deaths lies with the medical-legal system i.e. Coroners/medical examiners. The training and work environment of coroners/medical examiners focuses on unnatural adult deaths, e.g. Homicide, accidents, suicides. Determining the cause and manner of infant death requires expertise in examining infant tissue. The cause of death relies upon interpretation of information obtained from the circumstances of death including the scene, medical history, gross and microscopic autopsy examination, microbiology cultures, skeletal x-rays, toxicology, biochemical and vitreous humor studies. Explained infant deaths have a specific "test" or finding [a lesion sufficient to cause death] that establishes the cause of death. Unexplained deaths are identified by diagnostic criteria accepted internationally e.g. SIDS. The latter do not have lesions sufficient to cause death, however, because the death is unexplained there is a tendency to interpret any postmortem lesion as ruling out SIDS. A misconception about the SIDS diagnosis is that the diagnosis is reached by exclusion. In reality it is one of inclusion based upon diagnostic criteria. Refining the criteria to include sleep related death is

needed. The coroner/medical examiner signs the cause of death statement on the death certificate. The statement has a two-part format. Part I indicates the immediate cause of death on line a. Subsequent lines list the underlying cause[s] with the initiating event on the last line. Part II lists other significant conditions contributing to death but not resulting in the underlying cause given in Part I. The following examples will illustrate the proper completion of a cause of death statement in SUDI.

PART I. LINE A [IMMEDIATE CAUSE] – CEREBRAL EDEMA WITH HERNIATION
LINE B [INTERMEDIATE CAUSE] – SUBDURAL HEMORRHAGE
LINE C [INTERMEDIATE CAUSE] – HEAD TRAUMA
LINE D [UNDERLYING CAUSE] – SHAKEN BABY SYNDROME
PART II. BILATERAL RETINAL HEMORRHAGES

PART I. LINE A [IMMEDIATE CAUSE] – SUDDEN INFANT DEATH SYNDROME
PART II. PRONE SLEEPING; CO-SLEEPING; UPPER RESPIRATORY INFECTION

PART I. LINE A [IMMEDIATE CAUSE] – PNEUMONIA
LINE B [UNDERLYING CAUSE] – CONGENITAL CENTRAL HYPOVENTILATION SYNDROME
PART II. HYPOVENTILATION DURING SLEEP

PART I. LINE A [IMMEDIATE CAUSE] – CEREBRAL EDEMA WITH HERNIATION
LINE B [UNDERLYING CAUSE] – FATTY LIVER
LINE C [IMMEDIATE CAUSE] – REYE SYNDROME
PART II. UPPER RESPIRATORY INFECTION

In summary the diagnostic approach to SUDI should include input from a pediatric pathologist. The collaboration between coroners/medical examiners and a pediatric pathologist will improve the accuracy of the cause of death statement on death certificates. Refinement of the diagnostic criteria for SIDS to include sleep related death is needed.

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3. Classical SIDS, do they exist?

Arne Stray-Pedersen, Marianne Arnestad, Aashild Vege, Torleiv O. Rognum, Institute of Forensic Medicine, Univ. of Oslo

The validity of the SIDS concept has been questioned by different researchers due to the imprecise and exclusionary nature of the definition. At the first SIDS Global Strategy Meeting in Sydney, Australia 1992, Prof. J. Bruce Beckwith proposed a new definition of SIDS, limiting the diagnose "classical SIDS" or category 1 SIDS to infants that died at an age between 3 weeks and 8 months. The age limit was maintained to include the 95 % confidence interval of the cases. During the years after Sydney, the back-to-sleep campaigns not only resulted in a dramatic drop in the numbers, but also changed the age-distribution of the SIDS cases in most western countries.

We have studied the age distribution of all infants and small children that succumbed to sudden unexpected and unexplained death examined between the years 1984 to 2003. During the period
1984-89, before the back-to-sleep campaign, the 90 % confidence interval (CI) of the 91 SIDS cases examined was between 29 and 257 days (4 weeks and 8,5 months). During the years 1990-2003, after the onset of the campaign, the 90 % CI of the 190 SIDS cases examined was between 16 and 570 days (2 weeks and 19 months).

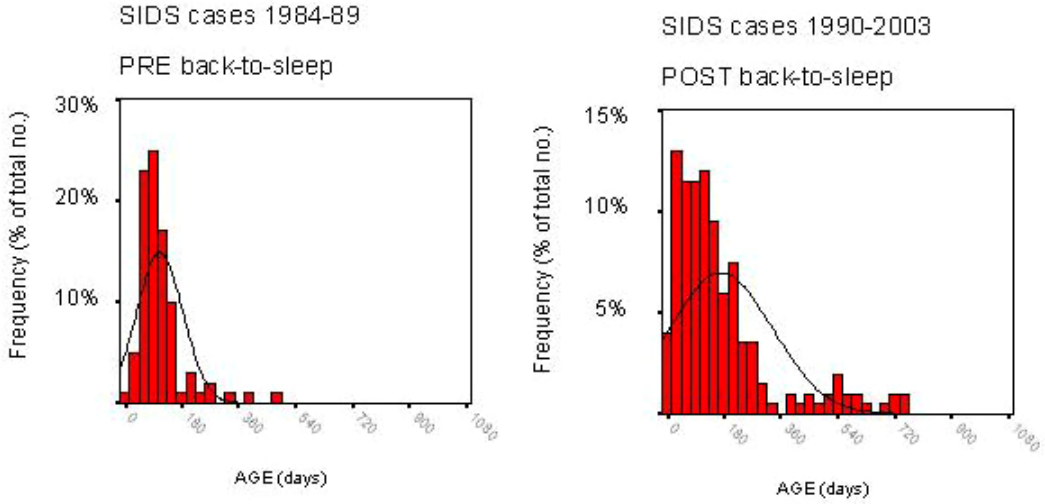

Figure. Age distribution of SIDS cases

The age distribution found in our pre back-to-sleep campaign cases fits fairly well with the definition of "classical SIDS" as proposed by Beckwith in 1992. It may well be that this population should be studied carefully against the classical risk factors, to generate hypothesis as to the mechanism of death.

4. Reclassification of post neonatal infant mortality

Susan M Beal, Post Neonatal Infant Mortality Committee, South Australian Department of Human Services, Adelaide, South Australia

1 Congenital Malformation
2 Conditions arising in perinatal period
3 Accidents including infants with face covered (previously often diagnosed as SIDS)
4 Inability to cope with prone (previously called SIDS)
5 Infection
6 Nonaccidental injury and abuse
7 Other (eg malignancy)
8 Undetermined (some of which would have previously been diagnosed as SIDS)

Reclassification of PNIM South Australia (SA) 2002

Cause of Death	No	Percentage	Rate per 1 000 live births
Congenital Malformation	8	30.8	0.46
Perinatal Conditions	5	19.2	0.29
Accident/Violence	4	15.4	0.23
Inability to cope with prone position	3	11.5	0.17
Infection	2	7.7	0.11
Undetermined	4	15.4	0.23
Total	**26**	**100**	**1.49**

This classification most correctly represents present knowledge about causes of infant death and the diagnosis of SIDS, previously useful, is now superseded and removed.

Inability to cope with prone

This used to be the largest cause of PNIM and the deaths were attributed to "SIDS". (In cities with a temperature in the place where the child sleeps of 0-12°C the incidence of SIDS in prone sleeping infants was approximately 5/1 000.)

If all infants are placed on the back if they are to be left unobserved then deaths of infants under three months and most infants under four months of age (who cannot roll to prone) would cease. If in addition infants are placed in a fitted sleeping bag, with arm holes, they experience difficulty turning to prone before six months of age and so these older infants would be protected. Thus inability to cope with prone is a preventable cause of death and so should be reduced to zero.

In SA (2002) the total PNIM of 1.49/1 000 LB is less than the SIDS rate of 2/1 000 in the 1970s - early 80s. The maximum infant mortality that could be attributed to SIDS in 2002 (inability to cope with prone + undetermined) is 0.4/1 000 LB which is one fifth of the earlier SIDS rate.

5. Continuous monitoring of cot death cases and controls in the Netherlands

Adele Engelberts, Diaconessenhuis Leiden, Leiden; Monique L'Hoir, Wilhelmina Children's Hospital, Utrecht, Utrecht; Guus de Jonge, retired, Amsterdam; Ben Semmekrot, Canisius Hospital Nijmegen, Nijmegen,

In the Netherlands continuous monitoring of cases and controls occurs. A paediatrician of the National Cot Death Working Group visits parents of cot death cases and about every other year prevalence data on infant care practices are gathered at TNO-Leiden.

Preliminary data about the infant care practices, compared to cot death cases are:

	1999 controls n=2534	2002 controls n=2909	1996-2001 cot death cases n=136
Breastfeeding			
Not breastfed	23%	19.7%	32%
Smoking after pregnancy			
Maternal smoking	20%	30.6%	38%
Paternal smoking	32%	28.3%	46%
Smoking in living room with child's presence	18%	7.1%	27%
Sleeping position			
Prone last night	8%	3%	21%
usually and sometimes prone		6.3%	
usually and sometimes side		19.7%	
pacifier use			
usually a pacifier at night, 0-1 months	29%	14.5%	27%
usually a pacifier at night, 2-4 months	36%	26.7%	37.5%
usually a pacifier at night, 5-9 months	46%	39.9%	48%
sleeping place			
infant's room		70.9%	
infnat's bed in parental room		22.8%	
bedsharing with parents	30%	4.9%	13%
bedding			
1 blanket		35.9%	
2 blankets or more		29.8%	
duvet	18%	7.2%	24%
blanket in sheetcover	22%	27.7%	8%
sleeping sack	44%	21.9% (thin) 24.6% (thick)	32%

Conclusion: Information on babycare habits in the general infant population and in cot death cases will be discussed and presented in more detail. Such information renders important information to focus the Dutch prevention campaigns.

1. Perspectives on Infant Care Practices and Sudden Infant Death Syndrome (SIDS) in a selected population of women in Saskatchewan

Debbie Jean Sihle Mpofu, Saskatchewan Institute on Prevention of Handicaps, Saskatoon, Saskatchewan; Koravangattu Sankaran, University of Saskatchewan, Department of Pediatrics, Saskatoon, Saskatchewan; Rosemary Bolaria, Saskatchewan Institute on Prevention of Handicaps, Victoria, British Columbia

BACKGROUND: SIDS continues to contribute substantially to the infant mortality rate. Saskatchewan has one of the highest infant mortality rates in Canada. Knowledge and awareness of SIDS and infant care practices has been identified as a modifiable risk factor that affects the SIDS rate. Information on these factors among Saskatchewan women is not available, particularly in the high-risk population. OBJECTIVES: The study sought to elucidate the level of knowledge of SIDS, and awareness of infant care practices that affect SIDS, in women attending a prenatal program, in five areas of the province. METHODS: A questionnaire developed and tested by the research team, was administered via face-to-face interviews. A descriptive analysis was used to convey the results and conclusion. RESULTS: Survey participants were118 women ranging in age from 16 to 40. One hundred and four (88%) women did not work outside the home; 101 (86%) indicated aboriginal ethnicity; and 64 (54%) had an education level of grade 10 or less. Bed sharing with parents and other members of the family was very common (78%). Knowledge of SIDS was related to education level of participants. Awareness of SIDS preventive measures was assessed in the following areas: awareness of what parents can do to keep their infant(s) safe from SIDS; knowledge of correct positioning of infant for the last sleep at night; awareness of safe sleeping place for infants. The study found that awareness of SIDS preventive measures was higher among those with higher education levels. Nearly all respondents had heard of SIDS, and 63% had knowledge of active practices aimed at preventing SIDS. However, ten respondents provided completely erroneous information about SIDS, for example, SIDS being caused by the actual crib. Twenty-nine (25%) participants indicated that they had used street drugs such as marijuana or cocaine within the past six months. Eighty-six (73%) respondents reported that they currently smoked cigarettes and 80 (68%) reported smoking while pregnant with this baby. Sixty-three (54%) reported alcohol consumption in the past six months, with 34 (29%) consuming five or more drinks at one sitting.CONCLUSION: The results are based on a selected population and therefore cannot be generalized. However, the awareness and knowledge about SIDS is suboptimal among the high-risk population in Saskatchewan. High-risk infant care practices are prevalent. Highly focused strategies are required to improve the awareness and knowledge of this group of women.

2. Infant care practices: Are Queensland parents using the Reduce the Risk recommendations?

Jeanine Young, Royal Children's Hospital, Adjunct Associate Professor, School of Nursing, Queensland University of Technology; Adjunct Senior Lecturer, School of Population Health, University of Queensland, Brisbane, Queensland; Peter O'Rourke, and Diana Battistutta, School of Population Health, Queensland University of Technology, Brisbane, Queensland

BACKGROUND: Sudden Infant Death Syndrome (SIDS) remains the leading cause of death in infants aged one week to one year in Australia. Since 1991, Queensland's SIDS mortality rate has been persistently higher than the national average. Infant care practices are the most important set of factors for reducing the risk of an infant from dying of SIDS, yet the prevalence of these practices and risk factors remain unknown in Queensland. A state-wide prevalence study was conducted to substantiate findings from a pilot study (n=36) that demonstrated serious deficiencies in parental practices related to infant care associated with a reduced risk of SIDS. AIM: This study benchmarked the prevalence of infant care practices associated with reduced SIDS risk in Queensland. METHODS: Using the Queensland Health's perinatal database, a cohort of 3952 mothers giving birth to infants within Queensland during April 2002 was identified. When infants were approximately three months of age, mothers were mailed a 10-15 minute questionnaire. Postal reminders were issued to non-responders 4 weeks after the first mail-out. RESULTS: Completed surveys were returned from 2531 (64%) primary caregivers, including 69 (3%) who indicated they were of Aboriginal or Torres Strait Islander descent. Routinely, 302 (12%) infants were placed prone and 558(22%) were placed on their side to sleep. Maternal smoking was reported by 1248 (51%) respondents, although only 507(20%) reported smoking during pregnancy and 546(22%) post-partum. Of 2126 (84%) infants sleeping in a cot, 1153 (54%) were not placed with feet to foot of the cot. Bed-sharing was common practice for 1136 (45%) infants and 278 (25%) of these infants bed-shared with a mother who smoked. A considerable number of infants used a pillow for their head or whole body (354,14%); slept on a sheepskin (495,20%); or slept with a cot bumper (376,15%) or soft toys (193,8%) in the immediate bedding environment. CONCLUSIONS: Results indicate that caregivers in Queensland are employing suboptimal infant care practices in greater numbers than their non-Queensland counterparts that may increase their infant's risk of SIDS. These findings have important implications for parental education by all health professionals midwives caring for families of young infants.

3. Reducing the Risk of SIDS by Educating Retailers
Maxine Weber, SIDS and Kids, SA

Since the Reducing the Risk of SIDS education program began in Australia in 1991 there has been concern that retailers continue to sell nursery care products that do not conform with SIDS and Kids safe sleeping recommendations.
This issue has been successfully addressed in an Australia retail store by introducing a staff training program and a series of lectures to the stores clients.
It has resulted in the store retailing products that meet SIDS and Kids guidelines and building customer loyalty by offering them the opportunity to attend lectures given by SIDS and Kids educators. The clients are informed about SIDS and Kids recommendations and given general safe sleeping advice for their babies.
Lectures are provided quarterly and customers of the store are invited to book a place. Following the SIDS and Kids presentation representatives of nursery products then have time to demonstrate their range to the invited guests.
This very simple program is making a difference

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4. Spreading the Reduce the Risk message through innovative means
Joyce Epstein, Foundation for the Study of Infant Deaths, London,

The Foundation for the Study of Infant Deaths (FSID) has been using a range of innovative means to keep the Reduce the Risk message in the mass media, acknowledged as an important source of information on infant care for people from all socioeconomic groups. For example a study published in 1993, two years after the RTR message was launched in the UK, found that among 16-24-year-old women 88% had heard of the RTR message from TV, 41% from newspapers or magazines, and only 18% from health professionals. (1)
However journalists in the print and broadcast media willingly gave space to the Reduce the Risk message when it was new in the early 1990s, but now they ask what else is new. The challenge is to make the proven, enduring, largely-unchanged Reduce the Risk message a fresh news story.
FSID has turned to corporate sponsors, such as toy companies, children's clothing manufacturers and baby food companies, for help. The marketing divisions of such companies are interested in raising their profile and in promoting sales by associating with a good cause. They are aware of the research suggesting that consumers are increasingly more likely to buy a product that has a charity link. For example in 1997, 24% of the British public believed that when buying a product it was very important that the company showed a high degree of social responsibility; by 2002 this had risen to 44%. (2) Establishing a corporate/charity link and getting it publicised is thus something that companies are willing to pay for.
Using funds received from companies, FSID has created news stories out of old stories. We have commissioned opinion surveys identifying gaps in public knowledge of particular aspects of safe infant care, eg that 63% of parents of young babies did not know that 18ºC was the recommended temperature of the room where their baby sleeps; or that 35% of smokers thought it was safe for them to bedshare. Such results create effective news "hooks" picked up by the press. The press story is further enhanced by announcing new initiatives to fill the knowledge gap, eg the availability of a new resource like a free cartoon sticker being given to all maternity units to place on infant cots illustrating the RTR message, or a free room thermometer give-away for parents, or a colourful postcard to be distributed by midwives showing the bedsharing risks, which are in turn funded by the sponsoring company. Press coverage also inevitably provides an opportunity to remind the public of the whole range of the Reduce the Risk message, not just the aspect on which the story focuses.
In addition to disseminating the information to an exceptionally wide and diverse audience numbering millions, including the difficult-to-reach lower income groups that read publications in Britain such as TV Quick, such projects also generate funds for FSID's research, support and education work.
Detailed case studies will be presented showing the benefits, and the drawbacks, of this approach.
1 Report of the Chief Medical Officer's Expert Group on the sleeping position of infants and cot death, Department of Health, HMSO, 1993.
2. Annual CSR study, MORI, 2002

13:30 - 15:00 **Indigenous Stream**

Back to Reality - An Indigenous Model for a SIDS Response **Salon 6**

Raeleen de Joux, Winifred Rata, Vanessa Savage, University of Auckland, Auckland,

The reality for Maori SIDS team frontline workers is a community whose social and economic marginalisation have made them the minority culture with a major percentage living in third world conditions. Although Maori have made some headway over 160 years of colonisation, we still lead the way with lack of education, high rates of unemployment, poverty, crime, suicide, loss of cultural identity and poor health.
The typically Western culture features of first response services impact negatively on Maori. The first contact following the SIDS event is usually by ambulance officers closely followed by uniformed Police officers who have little or no experience with grieving parents or of Maori customs. This is compounded by the complex situation involving coronial investiation with legal and medical requirements that bypassing any cultural imperatives around death rituals or religious practice. Although inroads have been made and there are now Iwi (tribal) Liaison Police Officers, Maori families are still being blamed, persecuted and traumatised by the SIDS experience.
This paper will explore the 'Maori workers experience of SIDS'. The three authors have personally supported 25 Maori SIDS families within the last six months and will explore the issues of being Maori and working within the Westernised first response support services. Their coping skills are born of their indigeneity and their experience leads them to propose an indigenous model for a SIDS response.

Publish or Perish: How to Get a SIDS Publication Profile **Salon 15**

David C Tipene-Leach, University of Auckland, Auckland,

Indigenous programs for the prevention of SIDS in our own communities are mostly about whether our people hear our message and respond positively to the things we have to offer. There are many levels of success of which a decrease in SIDS is the most important. But process outcomes, for example: the development of health promotion strategies, the funding of programs, the training and mobilization of workers and the responsiveness of institutions to indigenous needs are also vital. One of the core parts of gaining credibility among our SIDS peers, rightly or wrongly, is about the publication of the above strategies, activities, outcomes and the movement into original research.
When the Maori SIDS team began a decade ago we were all too busy to write. Looking back however, writing has been an important part of our development and a major feature of our ability to share our experiences with our communities, our national colleagues and the world.
This paper takes a lighthearted approach to the sixteen publications that the Maori SIDS team has participated in and draws up some simple ideas for getting your own indigenous SIDS publishing efforts up and running.

One Foot in the Door - A Cultural Icon - A Relevant Practice today **Salon 16**

Pauline Hopa, University of Auckland, Auckland,

The art of engagement between peoples is often learned in a haphazard milieu of trial and error. This presentation will outline the powhiri (the Maori ritual of encounter) so that it illustrates a time tested template of encounter as necessary in health work today as it ever was in days gone by. Traditionally, the process served to discover whether a visiting party was friend or foe, culminating in the formal welcome of the manuhiri (guests) by the tangata whenua (home people).
It begins with the karanga, the high-pitched voices of women from both sides, calling to each other in an exchange of information to establish intent and the purpose of the visit. A haka powhiri ensues, a chant and dance of welcome, during which the manuhiri are symbolically drawn onto the marae (sacred courtyard).
Next is the mihi or exchange of greetings by the orators from both sides. The kaupapa or purpose of the occasion will be discussed, and general present day issues and concerns aired. Each speech is followed by the performance of a waiata (song), or sometimes a haka (dance), by the orator's support group. At the completion of their speeches the visiting party will present a koha (gift) and then move across the marae to greet their hosts with a hongi (pressing of noses). The ceeremony concludes with a sharing of kai (food), called a hakari (feast). The two groups become as one.
Maori SIDS continues to use this traditional practice in their work, indeed would be crippled without it. Maori tradition is a tenet of the Maori SIDS program. National Coordinator for Maori SIDS, Pauline Hopa is also a tutor and exponent of Maori performing arts. With the group, Toitu Te Whenua, she will bring you the sights, sounds and sensations of the Maori - that inculcate trust and ensure an exchange of equals.

1. Quality assurance programme for the volunteer bereavement support of the Norwegian SIDS Society

Trine Giving Kalstad, the Norwegian SIDS Society, Oslo, Norway

BACKGROUND: The parental bereavement support of the Norwegian SIDS Society is based on a peer-to-peer principle: volunteers who have lost their child due to SIDS provide bereavement support to other bereaved SIDS families. In order to prepare the volunteers, they attend a two weekend process-oriented seminar on how to support bereaved families. To constantly assure the volunteer bereavement support, the Norwegian SIDS Society has established a quality assurance programme consisting of four initiatives: 1) process-oriented seminars, 2) seminar compendium describing how to provide bereavement support, 3) counselling service offered to volunteers and 4) establishing quality assurance routines at the secretariat for follow-up of bereaved families and volunteers. In this presentation I will focus on the fourth initiatives. GOAL: To continously assure the quality of the volunteer bereavement support by establishing routines of contact between the secretariat and the bereaved parents and the volunteers. This will provide parental feedback of the volunteer bereavement support and better knowledge of local activity. Another benefit will be increased consciousness regarding the tasks and expectations of the role as a volunteer in the organisation. We also expect that the Norwegian SIDS Society as such will be more visible as a provider of bereavement support and information. HOW: To prevent accidental contact with both bereaved families and volunteers, the secretariat must actively and by routine approach the bereaved families and the volunteers giving bereavement support: When the Norwegian SIDS Society is informed, the secretariat takes contact with the new SIDS family offering parental bereavement support. The secretariat assigns a volunteer. Only the first contact between the bereaved family and the volunteer is arranged by the secretariat. From there, the secretariat contacts the family and their volunteer by routine up to two years. A manual for these routine calls is developed. The presentation will describe the preliminary results.

2. Arranging the Peer Supporter/Client Match - the challenge for the Counselling Manager

Gregory C Taylor, SIDS and Kids, Canberra, ACT

The therapeutic approach undertaken by SIDS and Kids organisations in Australia to bereaved families whose children have died suddenly and unexpectedly, is to offer counselling, volunteer peer support or counselling and peer support in partnership. Counselling is provided by professional counsellors on either a face-to face basis or by telephone. Peer support by trained volunteer parent supporters who have suffered the death of an infant or young child, is provided either face-to face, by telephone or in peer group settings. The challenge faced by the counselling manager is to provide the most appropriate means of support available to clients to engender the most positive therapeutic outcomes. The benefits of providing peer support to bereaved families as a therapeutic intervention are well known, but many issues may need to be considered before referring a peer support worker to a family or individual. This paper will focus on some of the issues to be considered about the peer supporter/client match and include: --variables concerned with timing of support, that is, crisis support versus non-crisis support; --the effect of socio-economic and multicultural differences between supporter and client's background; --gender differences and sexual orientation between supporter and client; --common characteristics of supporter and supportee that may transcend cross-cultural boundaries; --the importance of boundary issues; --the notion of the continuing bond; and --the importance of peer support worker debriefing and supervision.

3. Counselling service for volunteer bereavement support

Trine Giving Kalstad and Oddbjørn Sandvik, the Norwegian SIDS Society, Oslo.

BACKGROUND: The Norwegian SIDS Society has 40-50 volunteers who themselves having experienced the loss of a child, ready to provide bereavement support to other bereaved SIDS families. Even though the volunteers have attended a seminar on how to give local bereavement support, many volunteers from time to time experience voluntary bereavement support as demanding and exhausting; the feelings of loneliness and insufficiency demotivate them. As part of the quality assurance programme the Norwegian SIDS Society has established a counselling service available for volunteers. GOAL: To strengthen the volunteers in their local bereavement support work by offering them an interlocutor. The organisation hope that the counselling service composed of trained counsellors will contribute to prevent burned-out and worn-out volunteers, and strengthen and motivate them to continue bereavement support work. HOW: There are two important conditions for being a counsellor in The Norwegian SIDS Society: the counsellors have to be familiar with the role as a volunteer, and he/she needs to be a trained counsellor. Five volunteer counsellors were challenged to work out the counselling service in co-operation with a staff member of the Norwegian SIDS Society and a psychologist, who also does the preparation seminars in bereavement support for volunteers. The conditions and challenges, professional secrecy and how to activate this counselling service were discussed at a planning seminar. Premises for the counselling service are voluntary effort and using telephone as the medium for counselling. This offer of counselling is not outreaching, but depends on an initiative from the volunteers themselves when experiencing their bereavement support situation as difficult and stressing. The planning process and experiences from the first 6 months will be described in the presentation.

15:30 - 17:00	Indigenous stream	Salon 6

Ending the Journey
Randi Gage, Loon's Cry Consultants, Winnipeg, Manitoba, Canada

This presentation is delivered in a workshop style using overheads. The presentation discusses death, dying and loss from each perspective. We will be discussing Death, Your Death, Your Loss, Our Loss and the Future. We will Laugh, We will Cry, We will learn to say Good-Bye.Topics covered are:
When Dying You Have To Face A Number Of Changes; -- The Seven Tasks of Dying; -- Fears of the Dying;
Definition Of "Family" -- The Five Stages of Grief; -- The 4 Tasks of Grief; -- Grief Flashback; -- The Family Guilt
Three Phases Following a Death; -- Treat Me Nicely, As I Die, Please!
Workshop Debriefing (when requested or needed). Workshop is approximately two (2) hours with three (3) breaks. All materials are developed for the community level with handouts for participants. The workshop can be edited to fit a shorted time frame. As well, it can be targeted to any age group from childhood to senior citizens.

July 4, Morning session

10:30 - 12:00	Salon 3
Health and Allied Professional Stream	**Culturally Based Efforts to Reduce the Risk I**

1.Moving from Outreach to Partnership: Culturally Competent Risk Reduction Efforts
Suzanne M. Bronheim, National Center for Cultural Competence, Washington, DC

In the United States, and throughout the world, the implementation of campaigns to promote safe sleep, smoking cessation and other approaches to reducing the risk of SIDS have had a dramatic impact on the death rates due to SIDS in the broad population. Unfortunately, those risk reduction strategies have not been uniformly adopted and SIDS rates for diverse cultural, racial and ethnic populations remain unacceptably high. In the United States, SIDS rates for African Americans and Native Americans are two to three times higher than the general population. Public health promotion is most successful when it embraces principles of cultural and linguistic competence. This presentation will provide a framework that is not culture/ethnicity specific to guide the efforts to provide effective risk reduction campaigns to diverse populations. The approach will emphasize key principles, values and components of engaging and partnering with culturally, ethnically, racially and linguistically diverse populations. It is based on the value that communities have the inherent ability to recognize their own problems and intervene on their own behalf in partnership with the medical/scientific community. Health promotion efforts that are developed in partnership with community, rather than brought to the community are more likely to reflect the heath beliefs of the intended audience and be respectful of the culture. The presentation will present a series of concrete steps that public health educators and others can utilize to create culturally and linguistically competent risk reduction efforts. Included will be: 1) how to identify key credible voices in the community; 2) how to create co-leadership with the community; 3) how to identify key health beliefs and practices; 4) how to address issues of literacy and linguistic access; 5) how to identify contextual issues beyond the control of individuals in the target audience; and 6) how to ensure economic reciprocity in partnerships.

2. The process of developing and validating competencies of Pacific Community SIDS Educators
Eseta Fifita Finau, University of Auckland, Auckland,; Sitaleki 'Ata'ata Finau, School of Public Health, Fiji School of Medicine, Suva

In 1995, SIDS was identified as an increasing problem among Pacific migrants in New Zealand. This trend was in contrast to the declining incidence of SIDS among Pakeha (Europeans). The incidence rate among the indigenous Maori was unaffected by the national SIDS prevention campaign during the same period. A Pacific SIDS Prevention Programme was established in 1997 at the Pacific Health Programme, Department of Maori & Pacific Health, University of Auckland, New Zealand, to address the above dilema. The training of ethnic specific Community SIDS Educators was identified as the culturally appropriate vehicle for SIDS prevention among Pacificans. The relevant competencies for these Pacific specific Community SIDS Educators were identified, validated and maintained through processes to be detailed in this presentation.
This paper also reviews the development of the Community SIDS Educators and their competencies including the rationale for undertaking the project and some of the lessons learned during the process. These lessons should be of universal applicability to SIDS prevention globally.

3. Djäka Yothu[1]: SIDS education in Northern Territory indigenous communities

Margaret T Lambert, SIDS & Kids Northern Territory, Darwin, NT; Christopher J Brocklebank, Ngaanyatjarra Health, Alice Springs, NT

The delivery of educational programs based on research into SIDS has gone a long way in reducing occurrences of sudden infant death in Australian babies. However an opposite trend is seen with the indigenous population. SIDS rates of indigenous babies have increased over the past two decades and are six times higher than for non-indigenous babies. Apart from lifestyle being a possible contributor, research has identified young maternal age, high parity, and small-for-gestational age as factors common with indigenous SIDS when compared with control indigenous infant groups[2]. The Northern Territory, with its high indigenous population (the majority of which live in traditional settings) has the highest rate of SIDS / "undetermined" child deaths in Australia. Most of these deaths occur in the northern region (Top End) of the Northern Territory.

What is being done to address this problem?

1. At the national level, a Memorandum of Understanding between SIDS & Kids and the National Aboriginal Community Controlled Health Organisation (NACCHO) has been signed, to ensure that educational programs on SIDS reach Aboriginal Medical Services and indigenous communities around Australia. The Northern Territory has a representative on the SIDS Aboriginal and Torres Strait Islander Committee.
2. At the local NT level:

(a) Culturally appropriate materials (video, poster, pamphlet) have been developed. In addition, *isee-ilearn.com* is developing "spoken" documents that can be understood in English and indigenous languages. The user does not have to be able to read or write or understand English to be able to understand the information (examples of spoken documents will be provided at the conference).

(b) The problem of distributing information across the vast area of the Northern Territory had led to the proposal of a partnership agreement with the Northern Territory Department of Health that will result in SIDS education being delivered by Aboriginal Health Workers in communities. This move will not only extend on the current limitations of reaching remote communities but will also place the delivery of the message in a more culturally appropriate context.

(c) New partnerships are being formed with the Department of Education to incorporate SIDS education into NT school curriculum.

[1] Title translation is "Look after baby", from Gupapungu, one of the Yolngu Matha languages of North-Eastern Arnhem Land.

[2] Alessandri, L.M. et al. (1996). An analysis of sudden infant death syndrome in Aboriginal infants. *Early Human Development*, 45, 235-244.

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4. Reclaiming Traditional Values and Practices to Ensure Maori Babies Get the Best Start in Life

Raeleen de Joux, University of Auckland, Auckland,

Maori are the first nation people of Aotearoa, New Zealand. Our ancestors lived in tribal communities where the health and wellbeing of mother and baby was the responsibility of the whole tribe. Traditional pregnancy, birthing and child rearing practices were integral in ensuring the survival of the Maori race and preservation of whakapapa (genealogy) lines.

The purpose of this paper is to outline the influence of colonisation and Western practices on traditional childrearing practices and the impact on the health and wellbeing of Maori babies and their whanau (extended family). It identifies the experience of Tamariki Maori (Maori child health) Regional Co-ordinators in mapping a strategic pathway to reduce the impact of SIDS on Maori communities. The paper will take you on a journey from a time when Maori, as the indigenous people of Aotearoa, had their own health infrastructure and practices, the tragic outcomes of interaction with a dominant culture, the impact of colonisation, through to the struggle to reclaim traditional practices as a means to improve the health status of Maori babies and their whanau.

The basis for the learning comes from the Maori whatauki (proverb):

'Ma te titiro whakamuri, ka kite i te huarahi haere whakamua'.

You need to look back to see the pathway to go forward.

Love Your Woman, Because It Ain't Nobody's Fault: The Sudden Infant Death Syndrome (SIDS) Experiences of Maori Men

Shane Edwards, University of Auckland, Auckland.

The accumulated experience of the Maori SIDS Prevention Team in Aotearoa/New Zealand, is that fathers often miss out on the support services provided after the loss of a baby. This paper is part of a broader qualitative investigation undertaken by our Maori research team, into the contexts and processes within which Maori SIDS occurs and presents an investigation of the life stories of Maori fathers. The paper privileges the voices of Maori men, treating their narratives as data that can build our understanding of the meaning and impact of their experience of SIDS. Insights on the highly gender-specific of the style of their stories, particularly in terms of their reticence in talking about themselves, and their perceptions of the roles available to them in families, as fathers and in grieving, are reported. We argue that our findings present challenges to those involved in grief counselling around SIDS and offer suggestions for development in this area and in prevention and health promotion approaches.

"Moving into their Comfort Zone" Support for Fathers following the Death of a young child

Kevin Carlin, SIDS and Kids Victoria, MALVERN, VIC

As a Social Worker doing bereavement counselling, I am in my comfort zone in the one to one counselling and in facilitating groups. However, there have been some challenges thrown up by my realisation that these conventional methods don't appeal to a high proportion of bereaved fathers. As the only male Social Worker at SIDS and Kids, I was expected to do something to address this imbalance in the supports we had to offer families following the death of a child.
In this paper I will discuss our efforts to provide regular opportunities for bereaved fathers to meet on 'their own terms'. 'Their own terms' were while fishing, golfing or playing snooker. Since November 2000, I have facilitated seven activities that have been consistently well attended. The participants have been predominantly blue-collared workers – a group difficult to engage with around bereavement. Our approach has been to create a social activity where the participants have the opportunity to enjoy the recreation and, as a consequence, meet other bereaved fathers. The activities are natural activities for these fathers to enjoy. They are relaxed in these settings. These are social events and fathers very often 'bring a mate along with them', grandfathers, brothers and older boys in the family often attend as well. In an evaluation fathers reported some interesting outcomes; after the activity they had 'good conversations' with their mate on the way home in the car. They reported talking about their experience of the infant's life and death and compared their experiences with other men on the trip; it stimulated conversation with their partner when they got home; the ' fishing (golf) broke the ice'. The focus was on the activity and afterwards, they report enjoying the opportunity to have a beer. 'It stirred things up for me and I had a good talk to my mate.' The activity seemed to give legitimacy to talk about their child with others. While these activities put the bereaved fathers into their comfort-zone, they took me right out of mine. I will elaborate on this in my paper.

Local support groups for men experiencing grief

Trond Mathiesen, Norwegian SIDS Society, Oslo; Arild Aanestad, Norwegian SIDS Society, Rogaland branch, Sola.

In 2001 the Norwegian SIDS Society organized a seminar for men having experienced the loss of a child. The seminar addressed the individual experiences and needs after the loss. One of the main purposes of the seminar was to inspire men to suggest alternative, supplementary bereavement support services. One suggestion was the establishment of local support groups for men experiencing grief. To this day, two local groups for bereaved men have been established. The presentation will refer to the experiences of one of these groups – a group that has been active since 2002. The idea is to create an all-male arena for mutual bereavement support. Not as a replacement, but as a supplement to the bereavement support services they make use of together with their wives. The group meets on the average once every month. The purpose of the meetings is to facilitate social intercourse and the mutual exchange of experiences. These meetings most often have a different approach than the services they make use of together with their wives. The most obvious difference is that the men get together and do specific activities together, for example hiking, go-cart racing or weekend fishing trips. With an initial focus on joint activities, the threshold for participation is lower. One is not expected to share one's feelings and thoughts. Presence alone is accepted. The effect of this bereavement support approach, however, is that a safe environment for conversations with mutual exchange of thoughts and feelings is created. The men report that both the content and form of these conversations are different from what they experience in the bereavement support groups they attend with their wives. The group discuss issues that would not be addressed if their wives had been present. The men also report that they use different words, have more intense conversations in an all-male environment and that the volume is higher. In conclusion, the men agree that the all-male group must continue to meet. The good turnout clearly indicates that men need this supplementary arena for mutual bereavement support.

Innovative Strategies for Reducing SIDS Among American Indian/Alaska Native Populations **Salon 5**

R. Mona Rosenman, CJ Foundation for SIDS, Hackensac, NJ

Every day in the United States, nearly eight babies fall victim to SIDS. A recent Aberdeen Area Indian Health Service Infant Mortality Study identified protective and risk factors associated with SIDS. Several conclusions from the study suggest courses of action to reduce the incidence of SIDS among American Indian and other high-incidence populations. The study noted that alcohol consumption by women of childbearing age (especially during pregnancy), maternal and environmental tobacco exposure during pregnancy, and pregnancy by women under the age of 20 increase the risk for SIDS. Though the cause of SIDS is not known at this time, we know that risk reduction education reduces the occurrence by nearly 50 percent. A new initiative that addresses all three risk factors mentioned above, while enhancing the Back to Sleep Campaign, can reduce a devastating health disparity among American Indian/Alaska Native populations
Learning Objectives: A demonstration of a unique partnership between Tribal Authorities and the CJ Foundation for SIDS that resulted in culturally appropriate programs which will "*allow participants to walk away with a stronger knowledge and deeper understanding* " of Sudden Infant Death Syndrome, and will allow participants to gain a strong motivation to change behaviors and save babies' lives through: 1) reducing alcohol use by pregnant women; 2) increasing smoking cessation (maternal and secondhand); and 3) educating teenagers on the risk factors for SIDS associated with teenage pregnancy within American Indian/Alaska Native communities

SIDS & Kids Queensland: Working Together Towards A Better Tomorrow **Salon 6**

Sonia Hebert, and Heather Lee, SIDS and Kids Queensland Inc., Brisbane, Queensland

The State of Queensland is located in Eastern Australia. It is better known throughout the world for it's beautiful beaches, abundant wildlife and The Great Barrier Reef. Tourists flock to this beautiful State to enjoy its warm weather and laid back lifestyle. However, all is not perfect in paradise. We are aware of various serious health issues, and a high infant mortality rate in the Aboriginal and Torres Strait Island population.
Australia's Aboriginal and Torres Strait Island families are 6 times more likely to suffer the tragedy of a SIDS death as compared to the rest of the Australian population (ABS, 2000). In addition it would seem reasonable to say, that Indigenous Australians are more at risk of sleeping accidents (such as accidental asphyxia, suffocation, and aspiration) compared to the rest of the population. In the year 2000, eleven of the fifty notifications (22%) that SIDS and Kids Queensland received were notifications of Indigenous babies' deaths. Our organisation is not a legally notifiable body; therefore, there may have been many more deaths than we are aware of at this time. In early 2001, the Human Services Unit of SIDS and Kids Queensland began organising an information workshop. The aim of the Aboriginal and Torres Strait Island Workshop was to gather people from various Queensland communities in the hope that together we may be able to find a way to help save Aboriginal and Torres Strait Island babies' lives.
This workshop lead to the formation of the Queensland Aboriginal and Torres Strait Island Advisory Group. This group represents various Indigenous communities throughout Queensland, and has lead to the discussion of various issues within these communities that impact on High Infant Mortality Rates. This presentation will share some of these issues that have impacted on the Aboriginal, Torres Strait Island, and South Sea Island people that in turn may be risk factors for high infant mortality.

Creating Communities of Care with Aboriginal Families: A relational and shared-values workshop **Salon 15**

P. Gaye Hanson and Michael Aherne, The Pallium Project, Edmonton, AB

This workshop will expose participants to the essence of in depth teaching-learning experience for those providing grief and loss support with Aboriginal clients in community or institutional settings. This workshop uses an approach which focuses on building and keeping functional care relationships with Aboriginal clients and families and is sensible about the dynamics of the historical and social context for many Aboriginal clients facing grief and loss in their families. This workshop is designed to be practical. It promotes understanding, respect, and accommodation as a pathway for enabling Aboriginal clients to maintain dignity and reduce undue pain and suffering during bereavement. Providing insights into the diversity of spiritual beliefs and values around children and family, the workshop will assist in care providers becoming increasingly responsive to unique requirements in individual families. The workshop will use experiential and participatory methods, some of which have a foundation in Aboriginal clture. Case studies and media will be used to provide opportunities for participants to carefully reflect upon, and develop useable, take away strategies for working with Aboriginal clients so that they might become more effective in everyday practice. Key areas of subject matter that will be touched upon include: the importance of cross-cultural relationship building; situating care of Aboriginal clients in historical and current context; the importance of establishing functional care relationship in a climate of diversity; and an overview on strategies for building and keeping relationships with Aboriginal clients.

The Journey of Tobacco

Jo-Ann Daniels, Alberta Community Crime Prevention Association, Edmonton, AB

The Journey of Tobacco is a workshop about tobacco as a medicine and how we may use this medicine to find the process of healing, physically, mentally, emotionally, and spiritually. The workshop explores from the beginnings, how we ask for help from our medicine people, and how then tobacco makes the journey to help heal us. An historical and cultural perspective on health and healing will be presented. The workshop is interactive using the medicine wheel and people's experiences, so that we share our knowledge and learn together.

SIDS Risk Reduction – Tobacco as a Risk

Cynthia Morris and Francesca Vinson, Kahnawake, Québec, Canada

- tobacco and its physiological effects on the body.
- Stress management – tobacco as a coping mechanism
- Tobacco uses vs. misuse – participatory
- Traditional significance of tobacco – participatory
- Misuse education
- Provincial resources – program development

1.Micro-environment and Sleep Characteristics in two-month-old Infants

Igor A. Kelmanson, St. Petersburg State Paediatric Medical Academy, St. Petersburg,

Sudden unexpected infant death is known to occur with increased probability in the families with inadequate infant developmental stimulation and micro-environmental organization. At the same time, sleep-related troubles have repeatedly been implicated into potential mechanism(s) of sudden infant death. The study aimed at evaluation of possible relationship between the quality of infant micro-environment and maternal reported behavioral features during sleep in 2-month-old infants, age known for peak incidence of sudden infant deaths. It comprised 115 randomly selected, apparently healthy infants (50 boys, 65 girls) from the community setting who were singletons born at term with normal birth weight in St. Petersburg in 2001-2002. Quality of infant care was estimated using the "PROCESS" (Pediatric Review of Children's Environment Support and Stimulation) inventory enabling to measure infant's developmental stimulation and organization. Infant's behavior during sleep was assessed using an adapted version of the Children's Sleep Habits Questionnaire (CSHQ). The babies facing more developmental stimulation and from more organized environment less often fell asleep in parents bed ($P = 0.036$). Infants from more organized environment were more often put to sleep at the same time at night, more often were ready to go to sleep at bedtime and less often struggled at bed; it was more common with them to have right amount of sleep and to have about a same amount of sleep each day; less often they moved a lot during sleep and woke up in sleep. These associations remained significant after adjustment has been made for each of such potential confounders as infant's gender, weight at birth and at study, gestational age, Apgar score at 1 and 5 minutes, birth order, maternal age and education, maternal marital status, infant's feeding at birth and at study, as well as to their simultaneous effects. Lower micro-environmental organization and developmental stimulation may be associated with certain disadvantageous infant behavioral features during sleep, related in part to the increased risk of sudden infant death. Infants with parentally reported sleep problems should be carefully considered for possible flaws in the quality of micro-environment.

2. Trends in Infants Bed-sharing with Parents in Oslo, Norway

Arne Stray-Pedersen, Marianne Arnestad, Torleiv Ole Rognum, Institute of Forensic Medicine, University of Oslo, Oslo,

Background: In several studies bed-sharing during the first months of life is found to be a significant risk-factor for SIDS. The frequency of bed-sharing seems to alter in different communities. Large variations are also seen with time, by the end of the nineties approaching 25 % in south east Norway. Following a drop in the number of SIDS cases in our area in 2003, we performed a pilot-study to explore whether this reduction could be explained by a change in bed-sharing trends.
Method: 131 mothers attending maternity clinics in different parts of Oslo were asked questions about their bed-sharing habits.
Results: The preliminary results demonstrate that 29.8 % of infants below one year of age bed-shared with their parents the night before attending the maternity clinic, and 31.3 % bed-shared regularly. Interestingly, infants below 2 months seem to bed-share more often than infants between 2-6 months of age (table).

Table. Frequency of infants bed-sharing with parents

		Bed-sharing			
		Reference night		Routine night	
Age (months)	Total No.	No.	%	No.	%
0 – 2	44	15	34,1	18	40,9
2 – 4	42	10	23,8	10	23,8
4 – 6	33	9	27,3	9	27,3
6 – 12	11	5	45,5	4	36,4
SUM	131	39	29,8	41	31,3

Discussion: The proportion of bed-sharers had not changed from a previous study from southeast Norway in the late 1990s. Decrease in bed-sharing cannot explain the low number of SIDS cases in 2003. Previous studies report that bed-sharing predominantly act as a risk factor for SIDS in the age group between 0-2 months of age. Our findings suggest there still is a basis for further reduction in SIDS rates, given bed-sharing is abolished for the smallest infants.

3. Infant-Parent Bed Sharing Practices: Cross-Cultural Comparisons

Fern R. Hauck, University of Viginia Health System, Department of Family Medicine, Charlottesville, Virginia
Juleen L. Stephen and Mir S. Siadaty, University of Virginia Health System, Charlottesville, Virginia

Background: Although rates of infant-parent bed sharing may be increasing, it remains a controversial practice. Little is known about why families bed share, especially cross-culturally. Further, no studies have been published that describe the attitudes and advice of physicians regarding this practice.
Objectives: A pilot study to determine the frequency, reasons for and circumstances of bed sharing among families in Central Virginia, examine the attitudes and advice given by physicians, and describe cross-cultural comparisons.
Methods: Interviewer-administered 38-item survey. Families with children under one year were invited to participate while attending clinic at 5 practices: 2 family practices, 2 pediatric practices, and 1 Women, Infant and Children (WIC) clinic. The target sample was 100. Mothers who indicated that they bed share with their infant were invited to participate in a follow-up, in-depth semi-structured interview.
Results: 101 surveys were completed, with one-third each from family practices, pediatric practices, and WIC. The mean age of the infants was 5.2 months (S.D. 3.7 months); 45% were white, non-Hispanic, 39% were African American, 10% were white, Hispanic, and 7% were other. The infant's usual sleep location was as follows: 53% in a crib or bassinette in the mother's room; 24% in a crib in a separate room; 16% in bed with mother (with or without others); and 7% other location. Almost half of the mothers (49%) reported ever bringing the baby to bed at night, citing multiple reasons: to calm when fussy (53%), because it is safer (41%), to be close/bond (39%), to breastfeed (16%), and because it is commonly done in their family (10%). Only one-third of mothers reported that their doctor had talked to them about the practice. African Americans were most likely to bed share and non-Hispanic whites least likely (OR 3.70, 95% CI 1.66-8.25 and 0.42, 0.19-0.93, respectively, with the referent being the average rate for the entire sample).
Conclusions: Based on these data, bed sharing with infants is commonly practiced for various reasons. Further analyses will be presented, including results of 14 in-depth interviews. These findings will help inform the debate on bed sharing and formulate advice on this controversial issue.

4. Co-sleeping and Sudden Unexpected Death in Infancy. A Case-control study.
David Michael Tappin, Glasgow University; Hazel Brooke, Scottish Cot Death Trust; Russell Ecob, Statistical Consultant; Glasgow, Scotland.

PURPOSE. Current advice on bedsharing with infants suggests it is safe if the parents are non-smokers, have not taken alcohol or any medication causing them to sleep heavily and are not unusually tired. The aim was to establish if this is still the case?
METHODS. This was a national study in Scotland, UK, population 5•1 million, with 53,000 births per year. A 1:2, case:control, design included 123 infants who died of SIDS between 1/1/96 and 31/5/00 and 263 control infants.
RESULTS. Sharing a bed, couch or chair during last sleep was associated with an increased risk of SIDS, multivariate OR 2•86, 95%CI 1•38, 5•90. Sharing a couch or chair presented the greatest risk, OR 12•14 95%CI 1•33, 110•62. In parents' room not bedsharing was protective particularly for infants whose mother smoked OR 0•15 95%CI 0•03, 0•73. Young infants less than 11 weeks who shared a bed during last sleep OR 12•78, 95%CI 3•61, 45•22 were at greater risk p=0•01 than older infants OR 0•75, 95%CI 0•22, 2•56. This risk remained if mother did not smoke OR 8•51, 95%CI 1•18, 62•26 or if she breastfed OR 15•50, 95%CI 1•68, 142•86. All 5 (4%) young infants of non-smoking couples who died, had bedshared during their last sleep.
IMPLICATIONS. Bedsharing may cause an increased risk of SIDS for young infants. Sharing a couch or chair for sleep should be strongly discouraged at any age. Infants may be safer sleeping in the same room as parents rather than on their own.

5. Heavy Bedding is a Risk Factor for SIDS
Martin Schlaud, Robert Koch Institute, Dept. of Epidemiology and Health Reporting, Berlin,; Maren Dreier, Hannover Medical School, Dept. of Epidemiology and Social Medicine, Hannover; Armin Fieguth, Hannover Medical School, Institute for Legal Medicine, Hannover,; Christian F. Poets, University of Tuebingen, Dept. of Neonatology, Tuebingen; Jan Sperhake, University of Hamburg, Dept. of Legal Medicine, Hamburg; Werner J. Kleemann, University of Leipzig, Institute for Legal Medicine, Leipzig,

OBJECTIVE: To study risk factors of SIDS by comparing "objective" scene data from cases and controls.
Design and Methods: Case-control study in parts of Germany from April 1999 to October 2001, including 64 cases (sudden unexpected death on 8.-365. day of life, SIDS approved by autopsy) and 191 controls (matched by age, sex, region, season). Sleep environment and death/wake-up scene data were obtained by standardised observa-tions/measurements in parents' homes, in cases shortly after death. The study protocol included: temperatures; di-mensions of room and cot; type, dimensions and weight of all bedclothes; type, dimensions and softness of the mat-tress; infant's clothing; items found in the cot; information on doors and window. Additional data were obtained by standardised parent interviews. Odds ratios (OR) and their 95% confidence intervals (95%CI) were calculated by conditional logistic regression. RESULTS AND CONCLUSIONS: Mean age of cases was 155 (SD 97) days, 61% were boys. A pillow was used in reference sleep by 48% cases and 19% controls (crude OR 4.6; 95%CI 2.32-8.94). After adjusting for infant's age and parents' nationality and socio-economic status, pillow use was associated with a 3fold risk of SIDS (95%CI 1.02-8.71). Mean weight of the bedding ('duvet', 'quilt') was 1393 (SD 761) gramms in cases and 874 (496) g in controls, yielding a crude OR of 1.2 (1.11-1.29) for each additional 100 g of bedding. Categorised by terciles of weight and adjusted for infant's age, a bedding weight of > 1110 g showed an OR of 3.1 (1.02-9.19) and a bedding weight of 656-1110 g an OR of 2.2 (0.93-5.29) compared to the lowest tercile (< 656 g). This is the first case-control study with "objective" scene data on SIDS world-wide. Despite the limitation of low numbers, our data suggest that use of a pillow and heavy bedding may be risk factors for SIDS.

13:30 - 15:00

Scientific stream **Cardiorespiratory Physiology** **Salon 5 and 6**

1. Prolongation of QT Intervals in Victims of Sudden Infant Death Syndrome : A Polysomnographic Study
Patricia Franco, José Groswasser, Etienne Denayer, Avram Benatar, Yvan Vandenplas, André Kahn; Free University of Brussels, Brussels, Belgium.

OBJECTIVE: Recent data suggested that prolonged QTc intervals are associated with Sudden Infant death Syndrome (SIDS). We hypothesized that QTc intervals in future SIDS victims were longer than those of healthy control infants. METHODS: QTc intervals were measured in the sleep recordings of 18 infants who eventually died of SIDS and 18 control infants. The infants of the two groups were matched for sex, gestational age, postnatal age, weight at birth and sleep position during recording. RESULTS: Compared to the control infants, the future SIDS victims were characterized by longer QTc intervals during the night during total sleep (p=.019), REM (p=.045) and NREM sleep (p=.029). Dividing the night in three equal parts, this difference was always present, but most marked during the first part of the night (p=.030), especially in REM sleep (p=.041). There was a negative correlation between parasympathetic tonus and the maximum duration of QTc intervals (r=-0.35, p=.049) in NREM sleep. CONCLUSION: Based on the present findings, it can be postulated that QTc intervals were increased in some future SIDS victims. This difference could be related to the autonomic dysfunction already reported in these patients.

2. Home monitoring: evaluation of impedance and pulse oximetry alarms.

Raffaele Piumelli and Luca Landini, Regional SIDS Center and Enrico Lombardi, Centre-Meyer Children Hospital; Niccolò Nassi, and Gianpaolo Donzelli, Department of Pediatrics, University of Florence; Florence, Tuscany, Italy

The inability of impedance cardiorespiratory monitors (TTI/ECG) to detect obstructive apneas remains a disquieting technological limitation due to the fact that, in previously published studies, death traces of infants monitored have shown a life threatening delay in alarm triggering, probably depending on undetected hypoxaemic events. Pulseoximetry (SpO2) is the standard technology for detecting blood oxygenation and is therefore considered a potential substitute for conventional TTI/ECG monitoring; however the high number of false alarms has always been considered the main limitation for its continued home-use. We have assessed the recordings of 35 infants enrolled between 03/16/2001 and 12/29/2003 who were simultaneously monitored with TTI/ECG and SpO2. VitaGuard VG 3000 monitor detects TTI/ECG via two or three electrodes placed on the chest and SpO2 by Signal Extraction Technology (Masimo-SET). The infants evaluated retrospectively in our study belonged to the following categories; ALTE (18), Preterms (11) Miscellaneous(6).Their median age was 45 days(Range 8-150).The total duration of monitoring was 1927 days. We found 400 true alarms that were classified as apneas, bradycardias, desaturations and 2825 false alarms that were classified as low impedance signals (LI), cardiogenic artifacts (CA), loose lead events (LL) and low SpO2 motion artifacts (MA). True alarms included 63 apneas, 17 bradycardias and 320 desaturations. Overall impedance false alarms (LI+LL+CA) totalled 1712, (LI=1439;LL=192; CA=81)accounting for 53.8% more than MA alarms which resulted in being 1113. In short, our data shows that pulse oximetry performed with Signal Extraction Technology has reached a good standard in that the false alarms due to SpO2 are less than those provoked by TTI/ECG. TTI/ECG failed to detect clinically significant events relating to desaturation alarms attributable to obstructive apneas or periodic breathing. Moreover, true impedance alarms were related to events that are also common in healthy infants. IN CONCLUSION, we are able to affirm that home monitoring can be effectively performed either with pulse oximetry alone or coupled with impedance when a more complete detection is required.

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3. Obstructed Breaths in the Terminal Event in Sudden Infant Death Syndrome (SIDS)

Mary E Pylipow, Kathy Harris, Bradley T Thach , Washington University, St Louis, Mo; Judith Henslee and Dorothy Kelly, SW SIDS Research Institute, Lake Jackson, TX; Valya Visser, Carolinas Medical Center, NC. S

BACKGROUND: Transthoracic Impedance (TTI) is commonly used to monitor respiration. Since respiratory and cardiac oscillations continue in TTI during airway obstruction, it is generally believed that TTI can not be used to detect obstructive apnea. The characteristics of the TTI waveform however, have not been considered. Change in TTT during breathing is determined by the fluctuating ratio of air to blood in the thorax (air is a poor conductor and blood a good conductor of electric current). Accordingly, we would expect to see a decrease in TTI with each obstructed breath. This is due to the surge of venous return which occurs with the negative thoracic pressure generated during each obstructed inspiration. Studies of SIDS tracings (Poets et al) indicate 3 phases in the death of the infant (hypoxic hyperpnea, then apnea, and finally gasping). Also, fewer successful autoresuscitations (AR) prior to death occur in SIDS infants than infants dying of other causes (Sridhar et al). Since successful lung inflation during gasping is the primary mechanism for AR, an obstructed airway could prevent AR. HYPOTHESES: 1) TTI can be used to detect obstructed breaths. 2) Airway obstruction is a factor in the failure of AR in SIDS. METHODS: First, four pre-term babies with apnea of prematurity and one micrognathic infant with obstructive apnea were studied to compare TTI, respiratory inductance (RIP), and flow measured by nasal pneumotachography during obstruction. Infants were continuously observed for activity and mouth closure and, following documentation of apneic events during sleep, experimental airway occlusion maneuvers were performed. Breaths were identified as obstructed when respiratory efforts in the chest and/or abdominal RIP tracings occurred in the absence of airflow. After identifying a baseline during periods of central apnea, breaths were scored as obstructed when a respiratory waveform larger than the cardiac artifact appeared entirely below the baseline (negative deflection). Finally, using this methodology, home monitor terminal event recordings of the death of 6 infants with autopsy diagnoses of SIDS were analyzed. RESULTS: In the healthy pre-terms studied, a total of 201 obstructed breaths were documented during sleep with a mean of 39 breaths per infant (range 22-92). Of that, 121 (60%) occurred spontaneously and 80 (40%) during experimental airway occlusions. Excluding apneas with tracings obscured by gross movement artifacts, a clear TTI baseline, required to diagnose obstructed breaths, could be established in most episodes (87/90 or 97%). TTI identified 187/201 (93%) of obstructed breaths. Impedance tracings in 6 infants dying of SIDS revealed their final breaths beginning 20 seconds prior to the monitor alarm until death. All alarms were triggered for bradycardia. Forty-four (mean 7, range 0-29) obstructed breaths were found in 4/6 infants. Three infants had obstructed breaths clustered early in hyperpnea (n=41) as well as gasping phases (n=3). One infant had a single obstructed breath during gasping alone. Furthermore, a distinctive and progressive pattern of obstruction over 8-15 breaths was seen in 3/6 tracings. In one patient, 2 brief partial ARs occurred followed by more obstructed breaths, however, AR failed after the third cluster of obstructed breaths. CONCLUSIONS: There are 2 critical periods in the sudden death of infants. The first period includes the initial factors precipitating hypoxia and the second is the failure of gasping to alleviate hypoxia. We demonstrated that impedance diagnoses obstructed breaths. Subsequently, we found that half of the SIDS infants studied had evidence of airway obstruction contributing to the initial hypoxia and also to the failure of AR during hypoxic apnea and gasping. Airway obstruction contributes to the terminal events in some SIDS infants.

4. The impact of blood oxygen levels on submental and diaphragmatic EMG during obstructive apneas in infants
Henning Wulbrand and Georg von Zezschwitz, The Eppendorf Center For Child Neurology, Hamburg, Hamburg.

During the past years we have shown, that internal and external upper airway obstruction can be terminated by a biphasic augmented breath synchronized with submental EMG activation. Only little is known about the upper airway stabilizing submental EMG activity during obstructive events in infants related to tcpO2 levels before apnea. Twelve preterm infants were studied at the conceptional age of 36, 40, 44 and 52 weeks (gestational age 29.1 to 34.2 weeks) during polygraphic recordings including EEG, submental and diaphragmatic EMG, ECG, thoracal and abdominal breathing movements and nasal thermistor. A total of 69 REM and 33 NREM related mixed and obstructive apneas (> 10 sec) have been observed. Following an initial increase at apnea onset we found a significant submental EMG activity decrease during the course of NREM sleep related apneas in correlation with pre apnea tcpO2-levels (Spearman Rank Corr. $p<0.05$) which were ranging from 44 to 80 mmHg. Termination of apnea was characterized by a synchronized submental and diaphragmatic EMG activation in 79% of NREM related apneas and 64% of REM related events. The magnitude of both submental and diaphragmatic EMG activity correlated with the pre apnea tcpO2 levels, too. The lower the pre apnea tcpO2 level, the more pronounced was the following decrease of submental EMG activity during apnea and the more distinct was the apnea terminating augmented breath as well as submental EMG activity increase (Spearman Rank Corr. $p<0.05$) during NREM sleep. These findings demonstrate a remarkable vulner-ability of upper airway maintaining muscle activity during apnea to an even mild decline of tcpO2-level. Moreover the relevance of synchronized submental and diaphragmatic muscle activation for termination of obstructive apneas in infants is emphasized. An even milder sleep environment related pO2-decline might be associated with impaired upper airway maintenance which could progress to airway obstruction related life-threatening events / SIDS long before reaching levels of hypoxemia.

5. Comparison of hypoxic arousal responses between term and preterm infants.
Rosemary SC Horne, Peter Parslow, Sarah Scott and Richard Harding; Monash University, Melbourne, Australia

AIMS: A failure to arouse in response to a hypoxic event has been proposed as a mechanism for Sudden Infant Death Syndrome (SIDS). Preterm infants are at increased risk for SIDS, and in this study we aimed to compare arousal responses to mild hypoxia (15% O2) between healthy term and preterm infants. METHODS: 9 preterm infants born at 29-34 weeks gestational age were studied with daytime polysomnography during which nasal airflow was monitored using a purpose-built pneumotachograph at 2-4 weeks and 2-3 months corrected age. A control group of 15 term infants born at 38-41 were studied at matched ages. The probability of arousal (POA) in each sleep-state was determined using chi-square analysis. Arousal latency was compared between sleep states with 2-way ANOVA for repeated measures and between preterm and term groups with 2-way ANOVA. Significance was taken at $p < 0.05$. RESULTS: In preterm infants POA was greater in AS compared to QS at both 2-3 weeks (AS 96%; QS 71%) and 2-3 months (AS 100%; QS 80%). Similarly in term infants POA was greater in AS compared with QS at both 2-3 (AS 97%; QS 80%) and 2-3 months (AS 100%; QS 74%). In addition arousal latency was longer in QS compared to AS at both 2-3 weeks ($p<0.01$) and 2-3 months ($p<0.001$) in term infants. There was no difference in either POA or arousal latency between groups. CONCLUSION: Our study has demonstrated that both preterm and term infants arouse readily to hypoxia in AS. Any impairment in this arousal response may place an infant at increased risk for SIDS in this sleep state. This study was supported by the NHMRC and the Sudden Infant Death Research Foundation of South Australia.

6. The efficacy of swaddling in infants who cry excessively; a randomized controlled trial
Bregje van Sleuwen, Monique L'Hoir, Tom Schulpen and Wietse Kuis, Wilhelmina Children's Hospital, Utrecht, Utrecht; Adele Engelberts, Diaconessenhuis Leiden, Leiden, Zuid-Holland.

In the Netherlands excessive crying occurs in 5-15% of all infants. A variety of different interventions can be launched to reduce the crying, but in The Netherlands no fixed strategy is used. Sometimes parents may employ potentially dangerous interventions to reduce the crying, for example placing the infants in a prone sleeping position or co-sleeping with their infant. Swaddling might be an effective method to reduce excessive crying and to prevent parents from using dangerous interventions, but no research on the effect of swaddling has been carried out. In the previous 5 years swaddling has become increasingly popular while no research has been carried out as to efficacy and safety. Therefore, in a randomized trial a standardized approach which consists of offering regularity and stimulus reduction was compared with an experimental group which received the same approach, supplemented with an old-fashioned intervention: swaddling. In this trial, 428 infants to the age of 12 weeks were enrolled and specially trained health care nurses guided them for a period of three months. Participating parents were mainly referred by healthcare workers from well-baby clinics. Outcome measurements are the amount of crying as measured by the 24-hours diary of Barr (1), and parent's perception of the crying in a cry-perception scale (2). The advantages and disadvantages of swaddling will be described. Data on the efficacy of swaddling will be presented. Based on this trial a nationwide campaign will be launched next year through well-baby clinics, to effectuate an efficient and standardized approach to excessively crying infants.
1 Barr RG, et al. Parental diary of infant cry and fuss behaviour. Arch Dis Child 1988;63:380-7.
2 Lester BM, et al. Infantile colic: cry characteristics, maternal perception of cry, and temperament. Infant Behav Dev 1992;15:15-26.

1. Responding when a baby dies - multi-agency protocols. Ann Deri-Bowen, FSID, London.

Responding when a baby dies requires a multi-agency response to ensure a thorough investigation takes place and that bereaved families are supported. Working together is difficult but essential if we are to understand why babies die and effectively distinguish natural from unnatural deaths. The Foundation for the Study of Infant Deaths (FSID) has been campaigning since 2000 for multi-agency protocols in England and Wales. FSID recommendations are that:
(1). A paediatrician, working in conjunction with the police should visit each family within 24 hours of the death to take a complete medical history and offer support to the family. (2). Every baby should receive a thorough post mortem examination by a pathologist who has special training and follows paediatric protocol. (3). A discussion is held after each death with the professionals involved. FSID has produced suggested approaches for police and coroner's officers, paediatricians, and casualty staff. Regional staff have visited coroners, paediatricians, senior police staff, child protection teams, and casualty departments to promote the campaign and offer guidelines for multi agency protocols. They have also initiated meetings with the key professionals to discuss the possibility of working towards a protocol. Two well attended conferences have been held for the professionals involved with programmes that have included setting up and running a multi-agency protocol, training for staff and evaulation. By January 2004 twenty eight areas in England and Wales have drafted multi-agency protocols. This presentation will look at how these working groups have been established, the number of areas where the protocol has been launched and those that are still in draft format and identify some of the constraints and pitfalls encountered.

2. Technology Aids in Grief Support Services
Chelsea A Griffin-Hilbert, SUDC Program, CJ Foundation for SIDS, Hackensack, New Jersey.

As life becomes more hectic, people are less able to attend "live" support groups or are not able to access one in their area. Internet support groups and group conference calling can offer new methods of grief support services. Very few examples of these types of intervention exist in the literature. The SUDC (Sudden Unexplained Death in Childhood) Program hypothesized that Internet support groups and group conference calls, when combined, would develop an excellent means of support to a diverse and geographically distant population. In September 2001, The SUDC program began providing services to 10 families who had lost a child over the age of one to a sudden and apparently unexplained death. In January 2004, the program serves over 95 families around the globe. Forty-five members use the Internet support group with an average of 300 monthly posts. To better serve the SUDC population, Internet group subsets have been created which focus on grandparents, men, siblings and those who have lost one twin. The SUDC program also utilizes bi-monthly group conference calls to supplement its support services. This enables those without access to a computer to have contact with others in the SUDC community. It also personalizes the contact through the sound of others' voices. Group conference calling is also used as a training method for the program's peer contact volunteers. The SUDC support services model will be discussed. Samples of the consent to participate, member feedback, other support services will be presented. Potential pitfalls and drawbacks will also be addressed.

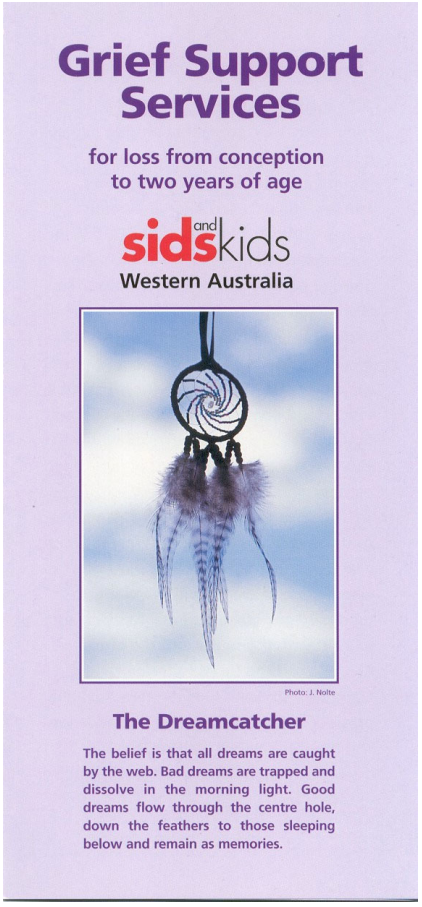
3. Follow-up routines after traumatic events and deaths: Routines on the Web site
Trine Giving Kalstad, the Norwegian SIDS Society, Oslo; Kari Dyregrov, Center for Crisis Psychology in Bergen, Bergen

BACKGROUND: A national study in Norway revealed accidentally and insufficient follow-up from the local health services after sudden deaths in children and youth. The study shows correlation between deficiencies in bereavement services and lack of organisational structures and plans. It also shows inadequacy of professional knowledge about crisis reactions and what constitutes adequate assistance. Community/Local health services providing assistance through formalisation of crisis teams and written plans for acute and long-term follow-up offer more and better help. The existence of written routines does reflect better psychosocial health in traumatic bereaved parents and siblings. A better quality of life is therefore possible to retrace by well-organised provisions of psychosocial help and care. Many institutions of local health services do not have formalising structures, or have a great potential for evolving their written routines and crisis team for psychosocial follow-up, which they also are required to do by law (1991). GOAL: The goal of the WEB-project is to work out detailed and differentiated routines for psychosocial follow-up after various traumatic events such as SIDS, suicide, accidents, murder, rape, fire accidents and the like. These routines will be elaborated and presented on a Web site. This project will in this matter give content to the instructions from the health authorities. HOW: The work out of these routines will be based on research, obtained knowledge of grief and crisis, and existing routines in local health services. The routines will be presented in a well-organised, user-friendly web-format, which can be printed out as a handbook of psychosocial follow-up. The routines will focus on the follow-up of the local health services and secondly draw attention to the cooperation between the latter and the specialist services and hospitals. This Web site will also have information from, and links to potential parental organisations and other volunteer- or peer organisations. The project is carried out at the Center for Crisis Psychology in Bergen, Norway. Norwegian SIDS Society is part of the reference group of the project, consisting of professionals from hospitals, local health services, the church, crisis psychology, police, data technique, survivor/parental organisations, and research.

4. The 'Parenting Dream': A Counselling Model for SIDS Families

Judi A Nolte, SIDS and Kids Western Australia, Perth, Kensington

A collaborative effort by the staff at SIDS and Kids Western Australia (WA), in 2003, identified the dreamcatcher as the symbol we wanted for our Grief Support Services Brochure (see Graphic 1). The intention of 'The Dreamcatcher' wording was to convey recognition of loss of a dream when there is a loss from conception to 2 years of age. Associated with the loss is the normal reactions of grief described in simplistic terms as a 'bad dream' to the 'good dreams' that are created through the memories that remain. Extending the dream concept further, the Family Services Counselling Team discussed our bereavement counselling process and believe that our goal, for effective client outcomes, is to counsel in respect to 'parenting dreams', thus the formation of a model that demonstrates the Parenting Dream Counselling Process at SIDS and Kids WA. That is, a process that examines the investment by the parent in the baby/child pre-loss ("dream-making") to, in collaboration with the client, the best bereavement process on an individual and/or group basis to continue their parenting dreams ("living the dream") albeit the final outcome is 'unascertainable'. This includes the client understanding self and what it means to be a parent., personal strengths of survival, enhancing partnerships, support through subsequent pregnancy/loss, maintaining/enhancing the relationship with surviving siblings and normalising what it means to be a bereaved SIDS parent within the family and community. For the counsellor, it means having an understanding of the concept of continuous grieving faced by parents, avoidance of applying grief and loss models that emphasise 'closure', and possessing the ability to sit with a lack of resolution - the "incredible or credible dream" of cause unknown.



1. Maori SIDS: The Life History Project
Verne McManus, University of Auckland, Auckland,

This paper reports a qualitative social science investigation of the contexts and processes within which SIDS occurs among Maori, the indigenous people of Aotearoa, New Zealand. The investigation carried out by the research unit of Maori SIDS team treats the knowledge and the life experiences of Maori SIDS parents, caregivers and whanau (family) as a valid database on which to build an understanding of the complexities that underlie Maori SIDS. We gathered life story narratives from a sample of families that had lost an infant to SIDS and then analysed them for broad themes which included family history and the context of the bereaved parents upbringing, their formal education and the life challenges in both the childhood and adulthood of these parents. From these analyses we are able to describe patterns in the lives of Maori that have experienced SIDS and, in line with a key aim of the project, suggest key domains in which health promotion and prevention activities could be deployed to help to reduce the incidence and sequelae of Maori SIDS.

2. Maori Collaborative Research With Maori Communities on Sensitive Issues
Shane Edwards and Verne McManus, University of Auckland, Auckland,

Maori in Aotearoa/New Zealand have been systematically dispossessed and marginalised through the processes of colonisation over the last 160 years. Among Maori the loss of a baby to SIDS impacts very heavily on families, often reverberating damagingly through communities long after the event. Research, particularly sensitive issues such as SIDS, among groups that are alienated from society is challenging and problematic. Suspicion and resistance to research interest often means access to participants is difficult, while data interpretation and accountability for findings are often contested. This paper draws on the experiences of indigenous Maori researchers working with Maori families who have experienced SIDS in a project to illuminate the conditions and processes within which Maori SIDS occurs, to detail practices employed to ensure the success of the research. The rationale and benefits of the approaches used are examined, remaining difficulties are acknowledged, and recommendations for other researchers working in similar contexts are offered.

3. Sleeping With The Enemy - Working Smarter, Not Harder
Riripeti Haretuku, University of Auckland, Auckland, New Zealand

The national Maori SIDS program was a foundation 'by Maori for Maori' health program established in 1994 after it became evident that mainstream solutions were having a minimal effect on improving SIDS outcomes for Maori. The deployment of a Maori approach with Maori workers was deemed to be the appropriate response to the prevailing situation. The long-term view however, was that the Maori SIDS program be eventually devolved into regional mainstream health promotion services. Ten years on however, the Maori SIDS prevention program has evolved into one seeking to develop further capacity in order to begin to address some of the wider issues of Maori infant health. There is a challenge for mainstream and by 'Maori for Maori' organisations to work collaboratively to achieve broader health gains but this approach is not well developed in New Zealand. The Immunisation Advisory Centre and the Maori SIDS program have forged such a relationship, an implementation of our Treaty partnership, to become a forum through which concerns for all children in New Zealand can be centralised. The mandate is use our energies and resources more efficiently and, focussing on the issues where need is great, to best utilise the knowledge generated from the University environment in which we are located and the support of actively involved and informed Maori communities.

13:30 - 15:00	Parent stream	Salon 2

1. Changing local activity of bereavement support: Regional family seminar
Trine Giving Kalstad, the Norwegian SIDS Society, Oslo, Norway

BACKGROUND: The Norwegian SIDS Society is a member organisation with local branches in all counties of Norway. One of our main tasks is to provide bereavement support to SIDS families. Meeting other bereaved parents is of great value, also documented in grief research. One consequence of relatively few SIDS deaths each year, is the fewer opportunities of meeting other bereaved families locally. Therefore some counties have started rethinking ways of organising local activity: With a regional approach to bereavement support work, families from two or more counties are invited to meet one weekend once a year. The Norwegian SIDS Society has adjusted the economical support programme, and supports up to 65% of the expenses based on a application with certain criteria. GOAL: Meeting the needs of both parents and siblings for meeting each other by arranging a regional weekend seminar of social and professional character. HOW: The idea of this seminar is to create a meeting place where the whole family attends; mothers and fathers as individuals and as a couple, and siblings. The seminar has both a social and a professional focus. The seminar took place in a zoo/amusement park. The seminar included separate seminars for men, women and children. The children were invited to do draw attention to their dead sibling by doing drawings and writings supervised by a grown up sibling – her self lost her brother due to SIDS. The programme also focused on parental challenges towards siblings after the loss of a child. We know a lot about grief and the differences between individuals, mothers and fathers. It is important to be aware of this and to make these differences a valuable resource for the parents' bereavement processes. This seminar illustrates the need of having arenas for both men and women where they are invited to discuss how to understand, accept and make use of the different individual grief strategies in terms of reactions and expressions.

2. Burial guide - an information booklet to parents who have lost a small child
Line Schrader, The Norwegian SIDS Association, Oslo, Norway

An information booklet on the burial of small children and babies is being developed to guide parents through the difficult days from the child's death to the funeral. The burial of their little child is one of the first things parents have to face after the child's death. For most parents this is a new and unexpected situation. They have to take many difficult decisions in the midst of the shock and pain, and they rarely know what to do or what kind of options they have. The information available from the undertaker is rarely well adapted to the special circumstance associated with loosing a child. The parents are often not able to express what they want or think in relation to the burial of their child.
A nation wide study of support and care for bereaved parents in Norway has pointed out that the parents need guidance on the many decisions to be taken before the child's funeral. They don't want anybody else to choose for them, but they want guidance, someone to call on them to be active and co-determinate and give qualified advice on what might serve them in the longer run. The burial guide aims to meet these needs. It will give information on what options are available to parents, what is recommended to do and what makes the choices difficult. It will contain practical information not only about the funeral arrangements, but also on issues that may come up before the funeral, for instance on taking photos, the death notice, undertakers, the obituary, etc. The booklet also aims to be an aid for both undertakers and hospital personnel in their difficult meeting with parents who have lost a child. The Norwegian SIDS Society is developing this information booklet using a Danish burial guide made by the Danish SIDS Parent Association as a basis, rewriting and adapting it to Norwegian conditions. Parents who have been through the experience of burying their own child, an undertaker and a hospital priest are involved in the making of the booklet through a reference group to assure the quality of the information.

3. Conflicting Advice and Parental Guilt
Helen Cormack, Scotish Cot Death Trust, Greenock, Scotland

In Scotland, as in much of the western world, parents are presented with a plethora of information on the birth of their child about current best practice in childcare. Among this information are the most recent strategies to reduce the risk of SIDS for their baby. Many of these strategies are the result of research initiatives and therefore carry substantial influence for parents of newborns. This presentation is designed as an informal and interactive workshop for parents, analysing the conflicting advice that has been offered to parents over the past thirty years from prone sleeping to bedsharing and discussing parents' reactions and feelings towards both the advice and the professionals who proffered it. Since the parents attending can be presumed to have followed the advice and still suffered the tragedy of cot death, discussion will centre on reaction to changes in advice, many of which are put in the public arena through media headlines as yet another study claims to have found a way to reduce morbidity. It is intended to use an animated Powerpoint presentation to set the scene and introduce the various advice topics and then to open the discussion up to the participating parents. A positive outcome is anticipated in that no matter what advice has been adopted, there are still no guaranteed preventions of SIDS.

4. Adolescents in grief - Information booklet for adolescents in grief and their social network

Line Schrader, The Norwegian SIDS Association, Oslo; Kari Bugge, Oslo University College and Ulleval University Hospital, Oslo; Eline Grelland, Ulleval University Hospital, Oslo, Norway

Grief after the loss of siblings, parents, friends or other beloved ones may have serious consequences for adolescents. They are in the midst of a vulnerable phase in life, and the loss can have a negative influence on their development. Grief reactions among adolescents are easily misunderstood, and they often meet little understanding from their surroundings. The youth's academic capabilities are often affected, reducing school achievements which may be decisive for their further education and career. Kari Bugge has in previous projects concerning bereavement support to children and youth in both hospitals and community health service, shown that both adolescents in grief and their families and friends need information about the grieving process and how to cope. Sometimes the adolescents in grief also need support and follow-up to be more able to work through and understand their own grieving process and to understand their family and friends. Unfortunately there are few options in terms of bereavement support groups or other help services for adolescents in Norway.
An information booklet about adolescents in grief is being developed to share important experiences from Bugge's projects and other existing knowledge of adolescents in grief. The booklet will provide information that can help adolescents understand and manage their own grief, at the same time it will give the youth's social network knowledge of how to give help and support. The target group is youth in grief (12-20 years) and their friends, family and school. As the booklet deals with adolescents in grief after the loss of beloved ones no matter the cause of death, several organisations are involved in the elaboration process to secure wide relevance: The Norwegian SIDS Society, the Norwegian Cancer Society, the Norwegian Association of Lost a Child, the Norwegian Organisation for Suicide Survivors and the Association for Traffic Injured. Adolescents who have experienced grief are also being involved. The booklet will be distributed through the mentioned organisations and in cooperation with Norwegian Health and Social Authorities.

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5. Support for extended families, especially Grandparents, after the death of a child.

Sandra Graben, and Lee Ward, Alliance of Grandparents, A Support in Tragedy, Clearwater, FL

"In the tree of life our roots are forever intertwined. With their last breath those we love do not say good-bye – for love is timeless. Instead, they leave us a solemn promise that when they are finally at rest in god they will continue to be present to us whenever they are called upon. Let us fear not, nor grieve beyond letting go the departure of those we have greatly loved for in the tree of life their roots and ours are forever intertwined." This is the base of the AGAST program. To facilitate their children's (the parents) well-being, grandparents need to be strong. In their grief for their grandchild, they need to remain cognizant of the grief of the grandchild's parents. Siblings, aunts and uncles of the deceased often need comfort. Grandparents, as the eldest, are looked upon as the strength in diversity. In 1993, a group of seven bereaved grandparents, understanding their own unique grief – not just for the grandchild, but also for the child that has suffered this unspeakable loss – decided to reach out to other grieving grandparents across the Nation and around the world. At the 1993 United States National SIDS Conference, The Alliance of Grandparents Against SIDS Tragedy (which became The Alliance of Grandparents, A Support in Tragedy in 1998) was organized, and in its eleven – year history has grown from that small group into an internationally recognized non-profit organization with peer contacts throughout the United States, and active grandparents in ten countries. At this time Norway and New Zealand have grandparent peer support, and Ireland, England and Australia reach out to grieving grandparents with pertinent information. Staffed entirely by volunteer grandparents, AGAST responds to "new" bereaves grandparents with information packets, personal contact, and remembrance cards. A quarterly newsletter has over 1300 recipients. AGAST gives grandparents an opportunity to release their own grief without adding to their children's grief, to talk with other grandparents that have "walked the walk", and to gain insight, through others' experiences, into what their children (the parents) are going through.

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6. SIDS in Twins and Higher Multiples

Kollantai, Jean, Anchorage, AK; Check, Alice, Beaverton, OR; united States

Introduction (Jean Kollantai): What are the realities, needs and challenges for families who lose a twin or higher multiple to SIDS? CLIMB (the Center for Loss in Multiple Birth, Inc.) has included several hundred such families in the U.S. and Canada over the years, along with several who have lost both twins, and a growing number who have lost a toddler twin or multiple to SUDC, Sudden Unexplained Death in Childhood. While these families have many needs and realities in common with families who lose a singleton to SIDS or SUDC, this presentation will explore their additional needs and challenges as multiple birth families, with the hope that it will not only be validating for parents, but assist caregivers, SIDS support groups, and others in the SIDS community in supporting them.
"SIDS in Twins" (Alice Check): The first question for all parents is: What is the risk to my other baby or babies? The realities faced by singleton parents of a subsequent baby are confronted most immediately with a "simultaneous baby" who was part of the same package, and one often identical to the baby who died; parents are confronted immediately with a monitoring

decision and other care issues, along with fresh fear. Alice Check (whose term twin son died of SIDS in 1987) has authored "SIDS in Twins" (1991, 2003) which compares and analyzes all the available published information (there is none known on triplets) to attempt to establish what is known about frequency and concordancy. She will summarize and comment on her findings—which include verification that as we suspected, the number of U.S. families of twins affected annually has remained the same, about 130.

Discussion of parents' needs and realities (Jean Kollantai, Alice Check, Paul Coache, Kim Coache): These include care of the other baby or babies while grieving; lack of specific information; disenfranchised grief ("you have another"); for some, loss of one of their first children after years of fertility technology; the needs of fathers, who are typically extra-involved with multiples; concern for the twin missing his twin, and raising a survivor not to feel "half of a broken set"; and the status, specialness and irreplaceability of multiples.

1. The SIDS Careworker Study

David C Tipene-Leach, University of Auckland, Auckland,

The SIDS Care-worker Study is a qualitative exploration of the experience of SIDS workers at the death scene and in the follow-up of SIDS families. The data are drawn from a single focus group interview of about three hours duration with six heavily experienced SIDS workers. They encompass a range of topics concerning the impact of SIDS and its sequelae on parents and families. This small study was done in order to help the Maori SIDS team pinpoint significant features for a wider study of the experience of a SIDS death. The outcome is a discussion of the six most important themes that arose as major issues for SIDS care-workers, themes that are likely to be found in any community of need. These themes include: the sensitivity of the death scene process, the cultural context of the death and it's impact on grieving, the actions of the police, the post-mortem process, the role female care-workers with grieving men and the role of the churches in recovery. In fact, this small study was hugely useful - confirming anecdotal experience, reminding us of the pre-eminent importance of factors outside our own control as care-workers and guiding the Maori SIDS team into the wider research arena.

2. Training porgramme for professionals supporting bereaved parents: Explaining the need for post mortem examination and encouraging consent for the retention of tissue and organs following a child death

Ann Deri-Bowen, FSID, London,

The Foundation for the Study of Infant Deaths (FSID) has designed a training package for professionals to explore how the sensitive issue of post mortem examinations and the need for consent for the retention of tissue and organs following a child death can be explored with bereaved parents. In England when a baby or child dies suddenly and unexpectedly the death will be referred to the coroner for investigation. A post mortem examination is part of the investigation for which parental consent is not required. Events in recent years have drawn particular attention to problems arising form the retention of tissue and organs of children, especially babies. The issues in obtaining informed consent are complex involving understanding of post mortem procedure, legal and ethical requirements, the nature of tissue samples and organs and the role of research. These must be viewed in the context of the support needs of bereaved parents. The Department of Health in England has funded FSID to hold sixteen half-day seminars for coroners' officers and seventy five one-hour seminars for paediatricians each year for three years. The focus for the training will be on child deaths referred to the coroner (age birth to16years). The training package covers: timetable for organising the seminar, setting up a meeting/seminar. resources for facilitators and for delegates, content of meeting/seminar, evaluation from delegates and evaluation from facilitators.
The half-day seminar content includes:
1. Understanding personal views of post mortems and tissue/organ retention
2. Information about sudden infant and child deaths
3. The need for paediatric pathology
4. The need for consent for organ/tissue retention for use in research, education and audit
5. Information parents need and what is helpful
6. A guide to the post mortem examination procedure involving a baby or child
7. Post mortem examination on a baby or child, ordered by coroner – consent form
8. Inquests
9. The disposal of tissue and organs
10. Support for professionals and families
The presentation will also include the evaluation of the first eight half-day training seminars and many one-hours sessions which will have taken place by the end of June 2004.

3. How to approach parents at the death scene using a police interview model

Lisbeth Sveum, Marianne Arnestad, Åshild Vege, Torleiv Ole Rognum, Institute of Forensic Medicine, University of Oslo, Oslo

In Norway the police use a basic interview model when investigating cases of abuse and homicide. The basic points included in this interview model are communication, legal protection, ethics and empathy, consciousness raising, thrust through frankness and information. The model is rooted in scientific studies of the psychology of communication.

In the project; "Better investigation in cases of sudden unexpected death in infants and small children" this interview model has been applied in approaching the parents during the death scene investigation. The model is based on 6 stages that guide you through preparing, performing and evaluating the interview. In every case there is a need for planning and preparation, such as gathering equipment needed, going through rapports and also get mentally ready. When we then meet the parents we strive to be engaged through giving information and possible explanations, as well as being ethical and show empathy. In the next stage we let the parents give a free account, after a few instructions. Clarification using question techniques and interview strategies is then necessary in order to gather all the information needed, and this can be a challenge in this model. To bring closure to the interview it is useful to summaries the information and be open for further contact. The last stage is evaluating your performance and the information you have obtained, and how to utilize this further.

We have experienced that the information gathered by using this interview model is more correct and verifiable. The model has also made it easier for us to structure the information we get from the parents, and in this way to better look after the infants legal protection.

15:30 - 17:30 **Salon 15-16**

Health and Allied Professional stream **Tobacco Smoking Cessation in Pregnancy**

1. Upholding Traditional Beliefs While Incoporating Science-Based Researcho

Dolores A. Griego, Sandoval County, and Jean Pino, Zia Pueblo, Bernalillo, NM
Christina Trujillo, Cochiti Pueblo, Della Tenorio, Santo Domingo Pueblo, Santo Domingo, Constance Anderson, Sandia Pueblo, Sandia Pueblo; Frances Vasquez, New Mexico Department of Health, Bernalillo, NM, United States

This project is the Collaborative efforts of the (7) Seven Central New Mexico Pueblos: Cochiti, Santo Domingo, Zia, Jemez, San Felipe, Sandia, and Santa Ana and the Sandoval County Community Health Alliance. The aim of the project is to create a templete brochure. The templete brochure incorporates traditional ways of caring for your infant with the science based approaches now known to help reduce sudden infant death. One focus is the re-introduction of the cultural and traditional cradle board. One key issue that the brochure addresses is the affect of second hand smoke and smoking. There exist spaces in the templet where identifying symbols and language for each of the seven different pueblos are inserted. Making the brochure specific for each pueblo. The following steps have occurred or will occur, and documentation is available at every step to show the progression. The end product is the creation of a culturally appropriate brochure created from within each pueblo.

• Forming the Collaborative: A historical overview for the call of material on the issue of Sudden Infant Death. Review of the meetings with the several pueblos to identify a need or interest for creating materials from within the pueblos
• Incorporating the professional community: Identifying individuals with a common interest to provide culturally appropriate materials to help establish a sustainable program that impacts the pueblo communities
• Identifying cultural barriers and proceeding without disturbing or disrespecting any traditional or sovereign boundaries. Creating a system of communication amongst western and native cultures.
• Plan of Action: Focus groups will be conducted at each of the pueblos, which consist of the elder women and other women of childbearing age. The facilitators, the recorder and the data collection person are members of the pueblos and trained by Leslie L. Randall, RN MPH.
• Focus group materials will be interpreted by facilitators and tribal epidemiologists and will be intertwined with the western science based information to fill the contents of educational material for the brochure.
• The brochures will validate traditional ways while incorporating science based research.

2. Patupuauahi
Pauline Hopa, University of Auckland, Auckland,

The high rates of cigarette smoking among Maori are a factor contributing to poor health status. Smoking rates for Maori women are particularly high. In 1996, the proportion of Maori women who smoked one or more cigarettes per day was more than double that for non-Maori women, and was consistent across all age groups. Maori women in all age groups except 65 years and over were more likely to smoke than Maori men. Approximately half of all sudden infant death syndrome deaths can be attributed to parental tobacco smoking. In 1999, Maori made up two thirds of SIDS nationally and the Maori SIDS rate was five times higher than that of non-Maori. Patupuauahi is a collective of community workers within Tai Tokerau (Northland) who actively promote Auahi Kore (Smokefree) in their work. They are drawn from social service providers, sport and recreation trusts, health providers, Iwi Runanga (tribal organisations) and education bodies. Their strategic intent is 'Auahi Kore' - literally 'no more smoke!' This presentation looks at their formation, the work they have done and what they continue to do.

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3. Motivational Interviewing - A tool for addressing smoking cessation across cultures
Ingrid R. Minett, Education for Change, Grafton, New Zealand

Motivational Interviewing: "a client-centered, directive method for enhancing intrinsic motivation to change by exploring and resolving ambivalence". Motivational Interviewing is a tool used by Smokechange Educators in New Zealand with pregnant women and their families and in families with young children where second hand smoke exposure has been recognized as contributing to the child's ill health and subsequent hospital admittance. When using motivational interviewing, the role of expert and student are reversed. The expert is the person on the programme (or the participant); the student is the interviewer, interviewing the expert through a process of open ended questions to gain a better understanding of the ambivalence to change. With a technique we call "bridges and barriers" the interviewer gains insight into things the participant enjoys about smoking (barriers) and the things they see good about becoming smokefree (bridges). In the process of understanding their ambivalence, the interviewer will often ignite a change process. Miller and Rollnick state that all people are motivated for something, but some can be trapped in a state of ambivalence. The "I want to but I don't want to" trap, or unresolved ambivalence. As a Smokechange Educator in Auckland I have had the opportunity to interview a number of mothers and fathers from different cultural backgrounds. Some changes I have seen include: less cigarettes/day; changed smoking habits (making rules around having a smokefree home or car); increased confidence for change; and becoming smokefree. While becoming smokefree is the most desirable outcome, fewer cigarettes smoked and more smokefree air lowers the risk of SIDS, miscarriage, stillbirth, and other complications. Increasing confidence lays a path for future changes.
This presentation focuses on three Smokechange participants from different cultural backgrounds. It tells their story on how the motivational interviewing process helped them make changes to their smoking.

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4. Better Practices in Smoking Cessation During Pregnancy & Postpartum
Natasha Jategaonkar, Lorraine Greaves, Renee Cormier, Karen Devries; British Columbia Centre of Excellence for Women's Health. David Aboussafy, WCB; Joan Bottorff and Joy Johnson,, UBC, Vancouver. Susan Kirkland, Dalhousie University, Halifax, NS, Canada

PURPOSE: This review examines overlapping literatures on aspects of women-centered health care and child-bearing and tobacco, and identifies elements of successful smoking cessation initiatives targeted at pregnant and postpartum smokers. Implications for tobacco policy are discussed. METHODOLOGY: This study is based on Health Canada's Better Practices Model, which involves several stages of systematic review and culminates in a set of evidence-based recommendations. Data were collected from over 65 published and unpublished smoking cessation programs and interventions aimed specifically toward pregnant and/or postpartum women. Findings have been reviewed to determine program suitability for pregnant smokers and considered in the broader context of woman-centered care. RESULTS: Twelve common components of interventions for pregnant smokers were identified. In general, existing programs are predominately concerned with the effects of tobacco on *fetal* health and relatively few emphasize maternal well-being. Additionally, programming designed specifically for sub groups of women and Aboriginal women is sparse. Seven better practice approaches were compiled from broader women's health and smoking literature for integration into future intervention efforts. CONCLUSIONS: While fetal health serves as a significant motivator to women who quit smoking during pregnancy, an equivalent focus on the woman's health is required to prevent relapse. Further research must be undertaken to develop effective interventions that facilitate sustained smoking cessation beyond pregnancy, both for women in general and for priority sub-groups of women, such as Aboriginal women, low income women and girls. RECOMMENDATIONS: Recommendations to service providers and policy-makers include implementation of several approaches which incorporate an increased focus on women's health, pre pregnancy and postpartum and the full social and cultural context of pregnant women's smoking.

15:30 - 17:00

Indigenous stream — Salon 6

1. A decade of Maori SIDS: What Do The Numbers Mean?
David C Tipene-Leach, University of Auckland, Auckland, New Zealand

New Zealand has had rising SIDS rates since the 1960's and in the late 1980's had the highest SIDS rates in the Western world. Maori SIDS rates were then over twice the non-Maori rate. The 1991 National Cot Death Prevention Programme led to a 50% reduction in SIDS from 1988 to 1992 and a consistent fall in SIDS has occurred since then. Official statistics suggest that there has been a much smaller decline in Maori SIDS. Subsequent investigation revealed that deaths of Maori babies had been systematically labelled non-Maori. The Maori SIDS Programme developed a SIDS Register that used a definition of ethnicity consistent with that in the Maori community. This Register identified a greater number of Maori SIDS deaths and indicated a significant fall in Maori SIDS over the years. Definitions of ethnicity are an important determinant of the apparent success or otherwise of prevention programs in indigenous communities.

2. Maori SIDS: A New Zealand Success Story
David C Tipene-Leach, University of Auckland, Auckland, New Zealand

The Maori SIDS program in New Zealand is a decade old. Looking back, it is clear that there is a combination of factors that has helped this small indigenous SIDS group to become an integral part of the diverse array of national child health promotion and health service providers, developing and maintaining credibility in both Maori communities and in health bureaucracies. New Zealand with its outrageously high rate of SIDS in Maori communities, nurtured and supported this program. This should be heralded at this SIDS 2004 Conference - where for the first time Indigenous people and their needs are being profiled.
The barriers to such development include the competition for the scare funding resource, the pressure towards a mainstream 'one size fits all' philiosophy, the institutional demand for a credible and professional profile and the need for a documentation and validation of a successful strategy alongside a strategic vision for health gain. Maori SIDS has met the above challenges. We have created an awareness of the SIDS issue in our community and been able to meet the subsequent service demand. We have provided a range of services that are not available in the mainstream. We have forged alliances with national and international SIDS organizations, health service providers and SIDS researchers and have developed our own research program that presently informs our service delivery. Indigenous peoples require their own programs to effect SIDS prevention and culturally appropriate care for indigenous SIDS families. Australian, American, Canadian and other dominant culture communitites need to provide for such development and could well model themselves on the New Zealand example.

3. Tamariki Maori Coordination
Tania A Pompallier, University of Auckland, Auckland, New Zealand

Fragmented service delivery is a major weakness in the current New Zealand system for provision of health care to infants and children. Poor intersectoral collaboration and the present competitive contracting environment demands an improved managed coordination between 'by Maori for Maori' providers and between those providers and mainstream services. The health of tamariki Maori (Maori infants and children) has special significance because of the Crown's obligations under the Treaty of Waitangi, the identified inequalities in health between Maori and non-Maori children and the Government's commitment to improve Maori health status so that Maori may enjoy the same level of health as non-Maori. In order for health services to address the above needs, more co-ordinated and complementary ways of working across the sector need to be established. This requires new management systems - a bottom up solution to the fragmentation problem. This paper addresses the management issues in the strategic development of a New Zealand infant focused, broad based child health care provider from a small, single issue Maori SIDS team.

4. The Maori SIDS Prevention Programme: What Did We Do That Worked?
Riripeti Haretuku, University of Auckland, Auckland, New Zealand

The Maori SIDS team has some success in the last decade and has participated in SIDS prevention programs that have seen a dramatic decrease in Maori and national SIDS rates. There are certain activities and certain ways in which we approached our work that we believe are responsible for this. Our work activities revolved around the development of a health promotion programme, the training of a myriad of health workers, the servicing of SIDS families, advocacy and policy work, dissemination and publication of work and participation in research. The major issues we encountered were family and community grief, the effect of the post mortems, the perversity of the 'modifiable/non-modifiable' risk factors, the bed-sharing issue and the pain of the death scene process. We survived as a team and saved babies by always maintaining a Maori world view, lobbying passionately for program resources, not believing the statistics that were put to us, having a 'complete' team from community workers through to academics and trusting only ourselves (and our grandmothers) to monitor our progress.
This paper summarises the activities of the Maori SIDS team for an indigenous audience.

Indigenous People Stream

Soul Stories and Wise Ways **Salon 15**

Denise Miller, Swan Song Storytelling, Edmonton, AB

In a story circle we are safe to set our spirits free. We travel effortlessly with the archetypal characters to places where we would fear to tread in our bodies. The stories resonate with us each in very individual ways. What is that feeling? Something is calling to you. Something is calling you to a place of healing, a place of transformation, a place of change. What is that something that is urging you on? That something that is urging you on really loves you. It is your higher self and it loves you unconditionally. It doesn't care what your story is. How scary it might have been. It only wants the best for you. Are you ready to answer the call and find out why you resonated with this story? This is a workshop for those open and on their path, those ready to Hear it, Feel it, and Heal it!

Ensuring Good Business with Aboriginal **Salon 16**

Josie Dahlstrom, Aboriginal & Torres Staite Islnder Advisory Committee for Sids & Kids Australia, Sydney, NSW

Although Aboriginal cultures have evolved and changed since the invasion in 1788 and colonisation by Europeans, there are still many traditional cultural beliefs, (i.e. socialisation, and rituals) observed and practiced. These cultural traditions and beliefs, even in the contemporary sphere, strongly clash with European (western) ideals, socialisation and models of management. This collision of the dominant and Aboriginal cultures is particularly pronounced in the health status of Aboriginal people. These problems, resulting from this cultural collision, in turn, impact heavily on the emotional, physical, spiritual, mental, well being of Aboriginal people. Watchdog agencies such as the New South Wales Health Care Complaints Commission are organisations set up as middle class white bureaucracies. Complaints about health services are a very important way for the health system to hear and respond to the problems confronting Aboriginal communities. We know making complaints can be difficult and we are working in partnership to reduce the difficulties Aboriginal people face when dealing with white organisations while using white rules. Often the people they need to serve most don't know about them or don't see them as relevant to their needs. Ways of doing business and the language undoubtedly alienates Aboriginal people and communities.
In the last three years the Commission recognise this reflection in the mirror and has started on its journey to do something about it. We will talk about what we have done

- To improve our understanding and make links with Aboriginal communities to help us become more accessible and provide better services
- Resolution Training for Aboriginal Health Worker
- Ensuring Cultural Sensitivity within the Commission

****The number, to the left of the poster title, indicate the poster board****

All posters can be viewed in the Fower, on the Assembly Level

13:30 - 15:00 Scientific stream Respiratory Physiology and Pathophysiology

001 Bedsharing and the infant's thermal environment in the home setting

Sally A Baddock, Barbara C Galland, Maartje G.S. Beckers, Barry J Taylor, David P.G. Bolton, University of Otago, Dunedin

Introduction: There remains considerable controversy regarding the risks and benefits of routine bedsharing between adults and young babies. To illuminate this discussion we have studied bedsharing and cot sleeping infants in the natural setting of their own home in order to identify differences in the thermal characteristics of the two sleep situations and their potential hazards.
Methods: Overnight video and physiological data was collected from 40 routine bedsharing infants and 40 routine cot-sleeping infants in the their own homes. Infants were aged 5-27 weeks and individually matched between groups for age and season. Physiological recordings included infant rectal and peripheral (shin) temperature. Room temperature was recorded from a sensor attached to equipment shelves 30cm from the ground. Overnight mean rectal, shin and room temperatures were calculated for each 30 minute epoch. Thickness of the bedding and clothing for each infant was estimated using the average measured thickness of typical New Zealand bedding and clothing of similar types. Effective thermal insulation was then calculated for the bedding and clothing of each infant. A log of infant sleep positions, the number of episodes of face covering, and awakenings, total awake time, number of feeding sessions and infant movements were also recorded.
Results: There was no significant difference in the overnight mean rectal temperature between the two groups however infants in the bedshare group had significantly higher mean shin temperature (1.2˚C) than the infants in the cot group. Bedsharing infants had more bedding than the cot-sleeping infants. Face covering events were significantly more common in the bedshare group and bedshare infants woke significantly more frequently than cot infants (mean times/night: 3.5 *vs* 1.4).
Conclusions: Bedshare infants in this study experienced warmer thermal conditions than those of cot sleeping infants but were able to maintain adequate thermoregulation to maintain a normal core temperature.

002 Cardiac Autonomic Responses To Auditory Challenges In Swaddled Infants

Patricia Franco, , Sonia Scaillet, , José Groswasser, André Kahn, Free University of Brussels, Brussels,

Objective: When infants have been swaddled and sleep supine, their risk to die from Sudden Infant Death syndrome (SIDS) is reduced with an odd ratio of 0.64-0.69. Alternatively, the risk for SIDS in swaddled infants shows a threefold increase in the prone position. The protective role of swaddling during supine sleep has remained unexplained. This study was designed to evaluate the effects of swaddling on cardiac reactivity to auditory stimuli during sleep in both the prone and the supine position.
Methods: Thirty healthy infants with a median age of 11 weeks (range 8 to 15 weeks) were studied polygraphically for one night, while sleeping successively prone and supine, or vice versa. The infants were studied while swaddled and non-swaddled in both positions. Heart rates (HR) were studied during REM sleep, before and after exposure to 90 dB(A) of white-noise.
Results: Ten infants were excluded from the study, because they woke up during the position change or the auditory challenge. Before the administration of the noise stimulus, swaddling decreased basal HR values only in supine position ($p=.049$). After swaddling, the basal HR values were significantly lower in supine than in prone position ($p=.003$). The auditory challenges were followed by a greater increase in heart rate when the infants sleeping supine were swaddled than when non swaddled ($p=.018$). When swaddled, beat-to-beat HR variability increased after auditory stimulation only in supine position ($p=.012$). No similar differences in heart rate changes were seen in the prone position.
Conclusion: When sleeping supine, swaddled infants had greater heart cardiac autonomic responses to noise challenges than when non-swaddled. In animals, movement restrictions have been associated to increases in parasympathetic activity, associated with greater changes in cardiovascular responses. If this condition represents a protective mechanism for SIDS, there is however, insufficient evidence to recommend swaddling as a routine procedure.

003 Autonomic activity in SGA infants sleeping prone and supine

Barbara C Galland, Barry J Taylor, David PG Bolton, Rachel M Sayers, University of Otago, Dunedin

Introduction: SIDS has been independently associated with both prone sleeping position and infants born small weight for gestational age (SGA). Earlier we studied infants born appropriate weight for gestational age (AGA) and found that measures of autonomic activity were reduced in the prone position compared to supine confirming several reports that the post-natal risk factor of prone sleeping damps some physiological responses. In the current study, we extended the risk factors to include the pre-natal risk factor of being SGA to study autonomic activity through measures of heart rate variability (HRV) and reflex HR and arousal responses to experimental tilting.
Methods: Two groups were studied at 1 and 3 months of age during a day nap: AGA (n=27) and SGA infants (n=27). AGA infants were >36 weeks gestation, >2500g and >25th percentile for gestational age. SGA infants were <10th percentile for gestational age. Mothers of all infants were non-smokers. HRV was measured during quiet and active sleep for 500 beats while sleeping horizontally. HR responses to a 60° head-up tilt were also measured and arousal responses observed and graded up to full awakening.
Results: HRV, assessed by point dispersion of Poincaré plots, was significantly reduced ($p<0.001$) in the prone position, was higher ($p<0.001$) in the active sleep state, and in older infants, but remained unchanged by gestational status. Baseline HR was similar in AGA and SGA infants but the HR reflex responses to the tilt were significantly reduced ($p<0.05$) in SGA infants (increase in HR: AGA 28 ± 1 bpm vs SGA 22 ± 0.8 bpm; decrease in HR: AGA -6 ± 1 bpm vs SGA -1 ± 1 bpm). There was no difference in the likelihood of arousal between groups. HR responses were greater in the active sleep state and in older infants but unchanged by position in both AGA and SGA infants.
Conclusions: The findings suggest that SGA infants have depressed autonomic reflexes that potentially could increase their vulnerability to SIDS. However sleeping SGA infants prone as opposed to supine did not add further to this vulnerability, confirming epidemiological data.

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004 Prenatal cigarette smoke exposure leads to aberrant breathing patterns during hypoxic and hyperthermic challenges in newborn rats

Shabih U. Hasan, University of Calgary, Calgary, Alberta

Background: Sudden Infant Death Syndrome (SIDS) is the leading cause of death in infants between one month and one year of age. Epidemiologic studies suggest a strong association between maternal cigarette smoking (CS) and SIDS. Other important risk factors include preterm birth, repeated hypoxic episodes and swaddling / over wrapping of infants. The mechanisms through which CS exert its detrimental effects on neonatal respiratory control remain unknown.
Objective: To investigate whether newborn rats, prenatally exposed to CS, will exhibit a depressed ventilatory response during thermal and hypoxic challenges.
Design/Methods: Pregnant Sprague-Dawley rats were exposed to either puffs of air or CS from d1 to 22 of pregnancy using the nose-only CS Exposure System. Rats received 1060 ml CS volume distributed evenly to eight animals; this quantity of smoke leads to plasma nicotine and carbon monoxide concentrations equivalent to those normally observed in heavy smokers. Breathing patterns were assessed in unanesthetized 7 days old pups using a continuous-flow, unrestrained, whole-body plethysmograph. The animals' body temperature was measured rectally throughout the experiment. After a settling time of 10 minutes, the rat pups were exposed to either 10% O2, balance nitrogen (hypoxia) or 8% CO2, balance air (hypercapnia) for five minutes followed by a 10 minutes washout period.
Results: During hyperthermic and hypoxic challenges, prenatally CS Exposed animals as compared to the Sham Group: 1) exhibited decreased frequency of breathing and increased gasping respiration and 2) were slower to recover after the termination of hypoxic challenges. The CS Exposed Group was more likely to gasp during hypoxia under thermoneutral and hyperthermic conditions and since both groups exhibited similar increases in body temperature, increased gasping and slower recovery in the CS Exposed Group was independent of body temperature. Hypercapnic challenges under thermoneutral and hyperthermic conditions had no significant effect on breathing patterns.
Conclusions: Prenatal CS exposure leads to frequent gasping and delayed recovery after hypoxic challenges. Further deterioration in respiratory control is observed under hyperthermic conditions. Our study demonstrates one of the possible mechanisms through which prenatal CS exposure under hypoxic and hyperthermic stresses increases the risk of SIDS.

005 Comparison of heart rate responses to nasal air-jet stimulation between term and preterm infants
Rita Tuladhar, MBBS, Richard Harding, PhD, Michael Adamson, Rosemary SC Horne, PhD, Monash University, Melbourne, Victoria

AIM: Prematurity is a risk factor for Sudden Infant Death Syndrome (SIDS) which may operate by altering the postnatal maturation of the autonomic nervous system. Our aim was to characterise the maturation of cardiovascular autonomic control during sleep in preterm infants by examining heart rate responses to arousing and non-arousing stimuli and contrasting these with data obtained previously in term infants (1).
METHODS: 15 preterm infants born at 26 to 32 wks GA with mean birth weights of 1225 ± 106g (mean ± SEM) (range 804 - 2406g) were studied with daytime polysomnography on 3 occasions: 36 wks GA (range 35 - 38 wks), 2-3 wk CPA (range 1 - 25 d CPA) and 2-3 mo CPA (range 64 - 98d CPA). Air-jet stimulation was delivered alternately to the nostrils in both active sleep (AS) and quiet sleep (QS) and HR changes recorded for both arousal and non-arousal responses.
DATA ANALYSIS: Baseline heart rate (BHR) data were collected over 20 beats prior to each stimulus presentation. For arousing stimuli maximum heart rate (MaxHR), was recorded and for non-arousing stimuli minimum heart rate (MinHR). The changes in heart rate (DHR%) were calculated as the difference between BHR and either MaxHR or MinHR, normalised for BHR. Comparisons between sleep states and postnatal ages were made with 2-way ANOVA for repeated measures and between groups with 2-way ANOVA. Results are expressed as mean ± SEM and significance was taken at $p<0.05$.
RESULTS: When preterm data were compared with term infants1 at matched ages, DHR% at arousal was greater in term infants ($p<0.05$) at 2-3 wks CPA. In addition, preterm infants demonstrated no significant sleep state differences in HR compared with term infants in whom HR was elevated in AS compared to QS at 2-3 wks and 2-3 mo of age.
CONCLUSION: Heart rate responses to stimuli inducing arousal were smaller in preterm infants compared to term infants at 2-3 weeks indicating that sympathetic activity in preterm infants may be lower than in term infants. This mechanism may account for the increased risk for SIDS of preterm infants.
1.Tuladhar R, Horne R, Harding R, Cranage S, Adamson TM: Early Hum. Dev. 2003:71;157-69
This project was supported by SIDS Australia, SIDRF (SA) and SIDassist

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006 Effects of sleep state and sleeping position on blood pressure in infants in the first 6 months of life
Stephanie Yiallourou, Andrew Ramsden, Adrian Walker, Rosemary SC Horne, Monash University, Melbourne, Victoria

BACKGROUND: Prone sleeping is a major risk factor for Sudden Infant Death Syndrome. It has been shown that autonomic control of heart rate is impaired in this position, however the effects on other cardiovascular variables are unknown. In this study we aimed to determine the effects of sleep position and sleep state on blood pressure control during the first six months of life. METHODS: Nine term infants (6F/3M) were studied at 2-3 wks, 2-3 mo and 5-6mo postnatal age with daytime polysomnography. Measurements of BP were recorded in 2 minute epochs using a Finometer TM with the cuff placed around the infant's wrist during both active sleep (AS) and quiet sleep (QS) in both the prone and supine positions.
DATA ANALYSIS: Movement artifacts were removed and data were averaged in 5s epochs for mean (MAP), systolic (SAP) and diastolic (DAP) arterial pressure. BP values were compared between sleep states, sleep positions, and across postnatal age using two way repeated measure ANOVA. Data are expressed as mean values ± sem, with $p<0.05$ considered as statistically significant. Results: Sleep position had no effect on BP at any of the ages studied. BP was affected by sleep state, with MAP and DAP being higher in AS than in QS in both the prone ($p<0.05$) and supine ($p<0.01$) positions at 2-3 mo. SAP increased with postnatal age in the supine position ($n=5$), being significantly higher ($p<0.05$) at 5-6 mo than at 2-3 wks in AS.
CONCLUSION: Our study has found that sleep state and postnatal age have an effect on blood pressure. The finding that sleep position had no effect on blood pressure was unexpected, but further study is needed to confirm this.
This study was supported by the NHMRC (284357) and the Sudden Infant Death Research Foundation of South Australia.

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007 Surveillance study Apparent Life Threatening Events (ALTE) in the Netherlands
Monique L'Hoir, Wilhelmina Children's Hospital, Utrecht, Utrecht; Adele Engelberts, Diaconessenhuis Leiden, Leiden; Jaap Mulder, Rijnstate Hospital Arnhem, Arnhem; Ben Semmekrot, Canisius Hospital Nijmegen, Nijmegen; Rob Bijlmer, AMC, Amsterdam; Koen Joosten, Sophia Children's Hospital, Rotterdam; K Liem, UMC Nijmegen, Nijmegen,

From January 2002 –January 2003, ALTE was subject of the Dutch Paediatric Surveillance Unit, which is part of the International Network of Paediatric Surveillance. All 14 members use the same active monthly surveillance method originally developed in the UK in 1986. The objectives of the surveillance were: 1. to estimate the incidence of ALTE presented to paediatricians in university and general hospitals; 2. to determine the national and regional patterns of presentation and referral; 3. to assess aetiology and to describe current practice regarding management investigations.
Any child from birth to the second year of age who had a severe episode characterised by a combination of apnoea, colour

change, marked change in muscle tone, choking, or gagging was included. PRELIMINARY ANALYSIS. Of 115 notifications 110 have been confirmed and questionnaires were completed, which is a response of 97%. It concerned 56% boys and 44% girls; 28.2% was < 2500 gram, 11.8% was born at < 32 weeks and 26,4% < 37 weeks. The percentage of first-born infants was 52%. Age of the mother at birth of this child was 4.5% < 20 years, 53.1% 20-30 years and in 46.9% > 30 years old. Symptoms before the incident were vomiting in 25.5%, infections in 24.5%, and feeding problems in 15.5%. Symptoms shortly after the incident were colour difference 43%, change of muscle tone 61%, breathing problems 11%. In 67% a medical problem explained the incident: 52% digestive problems, 13% apnoea, 11% infections, 5% cardiac conditions, 3% collapse after vaccination, 3% other conditions. In 33% of the cases no somatic explanation was found. In 10 cases polysomnography was conducted. In 6 children such incident had occurred repeatedly and in 10 children this had occurred once before. Fifty-one children were monitored in hospital and for 20 infants this was continued at home. In 17 cases a weighing program was started at home. Thirty-eight infants received medication after hospital discharge. In 6 cases the families were referred to a psychologist or social worker.

008 Grandparents and other senior family members and Back to Sleep: Knowledge and Behavior

Rachel Y. Moon, Rosalind P. Oden, Joana Iglesias, Children's National Medical Center, Washington, DC

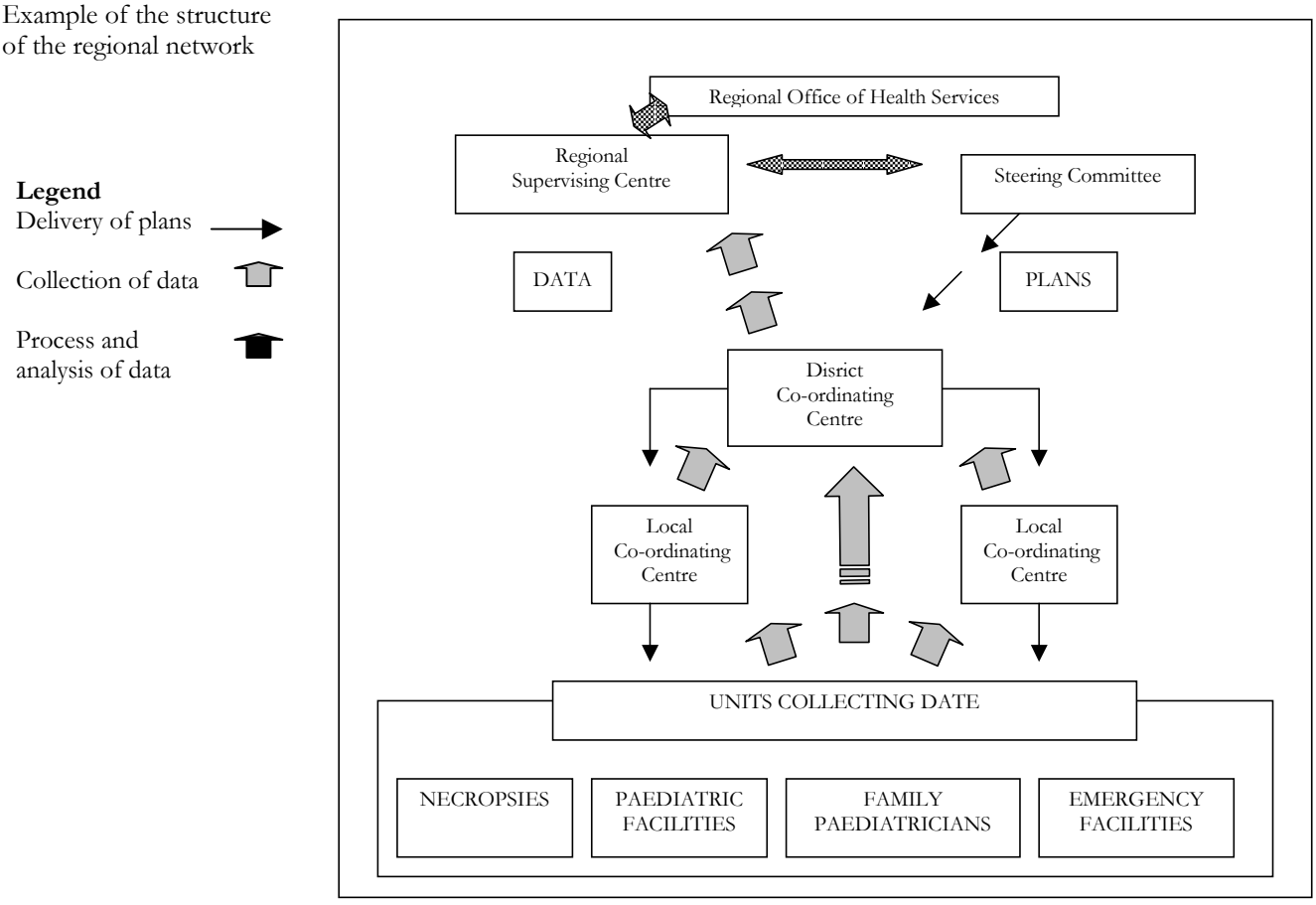
BACKGROUND: 20% of SIDS victims die while under the care of a nonparental caregiver, frequently a family member. 32% of infants with working parents are regularly cared for by relatives, and even more care for infants occasionally. Most relatives providing child care for infants raised their children before 1992, when the AAP first recommended that infants sleep nonprone. In order to reduce the racial disparity in SIDS, all relatives who care for an infant must be provided with the same information about safe sleep. Given that much child care is provided by relatives, it is important to determine what they know about Back to Sleep and whether they adhere to recommendations to place infants supine.
METHODS: This data was collected as part of a survey of current and prospective parents and other adult caregivers immediately preceding an educational intervention in a WIC clinic in Washington, DC. The clientele at this WIC site is largely African-American. Responses of nonparental caregivers were compared with those of parents. Outcome measures included reported infant sleep practices and knowledge of BTS recommendations.
RESULTS: Of the 731 respondents, 52 were nonparental caregivers (including 25 grandmothers and 13 aunts). Nonparental caregivers were older than parents (mean 38.1 vs. 25.2 yrs, $p<0.0001$) and had similar levels of education. Nonparental caregivers were more likely to have raised children born before 1992 ($p<0.0001$), and were more likely to have placed them prone (52.6% vs. 33.3%, $p<0.0001$). Nonparental caregivers were more likely to identify supine as the sleep position recommended by the AAP (52.2% vs 44.7%, $p<0.0001$), but less likely to believe that prone increased the risk of SIDS (45.9% vs 68.5%, $p<0.001$) and more likely to place infants prone (17.8% vs 13.3%, $p<0.0001$). Nonparental caregivers were more likely to place the infant in an adult bed than a crib (24% vs. 11.6%, $p<0.0001$).
CONCLUSIONS: Nonparental caregivers are more likely to place infants prone and less likely to use cribs than parents. More study is needed to ascertain why relatives do not use the supine position, and targeted educational interventions developed to improve use of supine and cribs with this population.

009 Web network against SIDS and ALTE

Stefano Parmigiani, Giulio Bevilacqua, Luisa Leali, Section of Child-Welfare and Neonatal Medicine, University of Parma, Parma; Emilia-Romagna Network Against SIDS, Emilia-Romagna Region, Emilia-Romagna,

Background: real frequency of both sudden infant death syndrome (SIDS) and apparent life threatening event (ALTE) are unknown. The cause of SIDS and ALTE as well as if ALTE is a precursor of SIDS are undefined.
Aims: to survey epidemiology of SIDS and ALTE in a well defined area, to collect pathological data from SIDS and clinical data from ALTE, to develop mass-information from the derived data, and to control application and effect on epidemiology of the preventive neasures.
Methods: a regional network has been constituted in the Emila-Romagna Region including all paediatric facilities. Pathologists, emergency facilities, family paediatricians have been involved too. Data on cases of SIDS and ALTE are collected and sent to the Regional Supervising Centre of Parma, were they are analyzed and stored to provide an on-line epidemiological register. Data collection is made
through a web form (C.S.A. med, Parma)that grants privacy of data. In this way results are available in real time. Pathological data on SIDS are centralized to the Institute of Pathology of the University of Milano to be analyzed following a specific protocol.
Discussion: the web network has just started to be operative and results will be available within months. Our network presents points of weakness as: 1.a great effort is necessary to collect data from few patients, and 2. funds are required to manage both the web and the organization. However it has points of strength as the possibility of having in real time incidence of SIDS and ALTE, as well as of collecting pathological and clinical data whose elaboration will drive to an increased knowledge of these diseases.
In the future it will be possible to enlarge this programme to other regions and to the whole nation. It is also foreseen to include sudden intrauterine unexpected deaths into the project.

Figure
Example of the structure of the regional network



010 Risk factors for sudden infant death syndrome, ten years after the introduction of the campaign to reduce the risk.

Bernt R Alm, Dept of Paediatrics, Göteborg University, The Queen Silvia Children's Hospital, Göteborg, Göran Wennergren, Gunnar Norvenius, Laslo Erdes, Per Möllborg, Rolf Pettersson, Nils Åberg

We have compared 430 healthy Swedish infants born between 1991 and 1995 with 599 healthy, six months old infants born in 2002, regarding the prevalence of risk factors for SIDS. Following the introduction of the campaign to reduce the risk of SIDS in Sweden in 1992, we could see a decrease in prone sleeping from 32 % to 7 % together with an increase in supine sleeping from 35 % to 44 %. Maternal smoking during pregnancy has gone down from 24 % to 10 %. The prevalence of breast feeding, already high in Sweden in the 90s, was largely unchanged, 69 % at six months of age in 2002.
This comparison shows that parents of small infants have accepted the advice on ways to reduce the risk of SIDS, and that information given at infant welfare clinics is still effective ten years later. Further improvements are possible by changing the side sleeping position to supine, and by decreasing tobacco smoking among pregnant mothers.

011 Unusual and unsafe sleeping environment in sudden infant death victims in Quebec

Aurore Côté, Stephanie Viau, McGill University, Montreal, Quebec

Our objective was to describe the prevalence of sleeping arrangements known to be unsafe in the population of infants dying suddenly and unexpectedly in recent years in Quebec. We also aimed at determining whether the last sleeping arrangement was unusual for the infant. This is a ten-year, population-based retrospective study. We reviewed sudden unexpected infant deaths (0 and 365 days of age) investigated by coroners between the years 1991 and 2000. Results. The exact sleeping arrangement was known for 443 of the 477 infant deaths reviewed (93%). Results: 96 infants (22%) where not cared for by their usual caregiver or were in an unusual sleeping environment (with their parents during travel or a stay with relatives or friends, for instance) at the time of death. Of the 443 infants whose sleeping arrangements were known, 81 (18%) slept in a recognized unsafe sleeping environment. In 93% of the instances of unsafe sleeping environment, the sleeping arrangement was new for the infant. The most common unsafe arrangements were unaccustomed prone sleeping (33 cases), beds where infants were found with faces buried in a pillow or cushions (26 cases), and sofa sharing (9 cases). The number of cases of unsafe sleeping arrangements did not change significantly over the ten years of our study.
Conclusions. Parents and caregivers still place their infants in a variety of unsafe sleeping arrangements. It does appear that this is done in response to the behaviour of the infant (crying or looking unwell) or because of unusual circumstances (travel). Our public campaigns to decrease the risk of sudden infant death should address those new issues.

012 The influence of swaddling on sleep characteristics and arousability in healthy infants

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Sonia Scaillet, José Groswasser and André Kahn, Free University of Brussels, Brussels.

Introduction: Swaddling is an old infant care practice. It favors sleep and reduces crying in irritable infants. As excessive crying is a reason not to put babies on their back to sleep, swaddling could be a method to promote supine sleep in an effort to prevent Sudden Infant Death Syndrome. The safety of swaddling must be controlled before being recommended for wider use. The purpose of this study is to evaluate the influence of swaddling on physiological sleep parameters and arousability in infants.
Methods: Sixteen healthy infants with a median age of 10 weeks (range 6 to 16 weeks) were recorded polygraphically during a night. They slept in their usual supine position. They were challenged successively during REM sleep in swaddled and non-swaddled conditions, and vice versa. The infants were exposed in both swaddled and non swaddled conditions to white noises of increasing intensities from 50 to 100 dB (A) to determine arousal thresholds, defined as the auditory stimuli that induced cortical arousals.
Results: Swaddling promoted a better efficiency of sleep (p=.030), more NREM sleep (p=.028) and less awakenings from sleep (p=.006). No differences were found in the frequency or duration of obstructive and central sleep apneas in both conditions. When sleeping swaddled, polygraphic cortical arousals occurred for significantly lower auditory stimuli that in the free condition (p=.005).
Conclusion: Swaddling promotes a better sleep with less awakenings. Motor restraints induced by swaddling could reduce proprioceptive stimulations to the reticular activating system and favor sleep. Swaddling induces increased arousability following auditory stimulations in REM sleep. In animals, movement restrictions have been associated to increases in parasympathetic activity, which reflects the individual's capacity to respond to stress.

013 Expectations and Organisations of Parents' Associations

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During the last decades, when the phenomenon of infant mortality has been object of interest by different health and care organizations, SIDS became, in many countries, the first cause of infant death in the age between two weeks and one year. Since then, especially in some Anglo-Saxons countries, parents decided to gather themselves in associations devoted to the intentions of curtailing the risk of SIDS and living more peaceful the role of close relatives. From the beginning, it was clear that not only the family support was the target of these associations, but also the research sustain, in view of the fact that SIDS drives few pharmaceutical studies. For that reason, associations tried to raise funds to exploit for fellowships and conferences organizations, with the intention to force the research interest in the field of SIDS. The last phase was originated by the requisite of a preventive system correlated with the evidence of some risk factors. Many efforts have been devoted to the health policy organizations in order to start up an effective process (through laws and regulations) reducing the impact of the mechanisms which usually follow the SIDS event. Considering the problem of anticipating parental responses and managing critical incident stresses associations of SIDS parents are involved in a support process aimed at:

a) easing a psycological support and an helping path;
b) dropping sense of guilt;
c) facing the eventual justice intervention during the first period of loss;
d) increasing the opportunity to have another child, making easier to get monitors from health structures;
e) reducing relation troubles between parents and siblings (Zerbi-Schwartz, 2002 and Bluglass, 2002).

Helping researchers means that parents expect to:

a) define useful protocols to update national and international databases, possibly to manage by the scientific community;
b) in SIDS case, scientific community is a broad term, since it involves experts in pediatrics, epidemilogy, physiology, pathology, psycology; to recognize the different causes of SIDS they should communicate each other: parents associations facilitate this process;
c) increase awareness of family pediatrics through risk factors campaigns;
d) put attention to the disadvantaged communities who experience disparity of health, privilege, income and opportunity in relation to the larger population (Randall, Cobb, Bryan, 2000);
e) offer more consideration to the autoptic exam, developing, at the same time, respect towards cultural values of single people, communities and the countries as well. This means that the test needs to be complete but ethical at the same time, if we accept the idea that "a poor autopsy is an unethical autopsy" (Berry, 2003).

Even though SIDS appear to be a phenomenon by definition withouth any actual explanation, literature and experience record a number of risk factors and general characteristics that could help to build a preventive system. The main features are:

a) prone position;
b) room temperature, generally higher than 20-22 degrees (Johnson, 2003);
c) period of event, usually colder months;
d) co-sleeping (Brooke, Tappin, Ecob, 2003);
e) young mothers;
f) babies whose mothers smoke during pregnancy and babies living in a smoking environment (Mitchell, 2000);
g) premature or low birth weight infants (Malloy, Hoffman, 1995);
h) infants with upper respiratory infections;
i) sub-sequent brothers and sisters.

Some researches show higher incidence in presence of some pathologies which can be diagnosticated in the early days. An effective preventive system should individuate these factors in order to apply a mechanism of monitoring and information able to reduce the incidence of the phenomenon and the ability to intervene in ALTE events. Finally, associations try to motive potential financial supporters. The life cycle of the fundraising activity may be define as follows:

a) creation of vision and mission: in case of SIDS the mission may be to live in a state of the world without any unexplained pathology;
b) identification of the good reason: this depends on the single donor and on its interests, but what is important is to make the project concrete and easy to evaluate (campaigns, purchase of monitors);
c) choice of markets to reach: from private, to corporates, from public to foundations;
d) selection of raising tools to employ: SIDS parents associations have experienced many occasions to get funds, from public events to direct contacts;
e) execution of decisions: all the people involved must acknowledge the project of the organization so to implement it;
f) evaluation of the performance: at last, the economics of fundraising needs the estimation of the optimal level of input-output cash-flows and the planning of the reporting for all the stakeholders.

The purpose of the paper is to define the attitude and to model the possible organisations of parents' associations which appear to be the principal stakeholders of SIDS, so to maximise their ability to reach all their targets.

014 Cross Sectional, Population Study of Sleeping Positions for Term or Near Term, Well Babies In Hospitals in The State of NSW, Australia

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Background: The SIDS rate in NSW for 2002 has decreased to 0.47 per 1000 births (total births 85 858) following the reduction in use of the prone position when sleeping, but remains more than twice as high as South Australia and European countries such as the Netherlands. The NSW Child Health Survey, a cross-sectional population survey of caregivers, indicates that overall one third of infants are placed on their side for sleep (urban areas 30%; rural areas 39%). This position has a two-fold increase risk of SIDS due to the risk of rolling to the prone position with increasing age. Literature from the United Kingdom and the United States suggests that health professionals are the most important sources of information for parents and hospital practitioners serve as a role model for what is practiced at home.
Aim: A self-report, cross-sectional, population survey of all nurse unit managers in postnatal wards and nurseries in NSW, and level of awareness of SIDS risk factors was designed to investigate current sleeping position policies and practices for well term and near term neonates.
Results: Of all 136 public hospitals that admitted babies in the early neonatal period, 134 surveys were returned (response rate 98.5%). Formal policies on sleeping positions for term or near term babies were reported in 25% of postnatal wards, and in 20% of nurseries. The selected sleeping position was almost always on the back for 61% of postnatal wards and 45% of nurseries. Side sleeping was used most or some of the time in 30% of postnatal wards and 32% of nurseries, with hospitals in rural areas using this position significantly more often than those in urban areas. The most common reason given for placing babies in the side position was to prevent aspiration of gastric contents.
The positive identification of SIDS risk factors by respondents ranged from 10% to 95%. The most important factors were identified as follows: sleeping in the prone position (73%), sleeping in the side position (39%), head covered while asleep (93%) and mother who smokes (95%). Social factors were the least well recognised.
Conclusion: The findings suggest that health professionals are not fully aware of the potentially lethal SIDS risk factors (prone and side sleeping) and practice side sleeping of well neonates in hospital to the same extent as parents in NSW (33%). Evidence based educational methods are needed to ensure that the strong evidence (epidemiological, anatomical and physiological) supporting the use of the back position for sleeping is known and practiced and that post intervention risk factors for unsafe sleeping are recognised and transmitted to parents.

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015 Cot Mattresses as Reservoirs of Potentially Harmful Bacteria and the Sudden Infant Death Syndrome

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Cot mattress materials were investigated as potential reservoirs of bacteria in relation to the sudden infant death syndrome (SIDS). The sleeping position of the infant significantly influenced bacterial population density of cot mattress polyurethane foams ($p<0.0000001$) and their covers ($p<0.004$). *Staphylococcus aureus* was isolated at significantly higher frequency ($p<0.03$) from the infant's head region of cot mattress materials. Significantly higher bacterial population densities ($p<0.001$) were associated with polyurethane foams from non-integral mattresses (exposed polyurethane foam), when compared to those from mattresses completely covered by polyvinyl chloride (integral type mattress). The frequency of isolation of *S. aureus* from polyurethane foams from non-integral mattresses was also significantly higher ($p=0.03$) than from foams from the integral type. The following factors were significantly associated with increased frequency of isolation of *S. aureus*: from the polyurethane foam, previous use of non-integral mattresses by another child ($p=0.03$ for all sample sites, $p=0.01$ for torso region); from the covers, sleeping in the prone position ($p=0.003$ head region, $p=0.001$ torso region). Prone sleeping was also significantly associated with increased bacterial population levels ($p=0.01$) and increased frequency of isolation of *Escherichia coli* ($p=0.02$) from the torso region of cot mattress covers. These findings could explain some recently identified risk factors for SIDS associated with type and previous use of cot mattresses. In particular, the finding could explain an increased risk of SIDS associated with sleeping on older mattresses not completely covered in polyvinyl chloride [1] and with routine use of an infant mattress previously used by another child [2]. *Clostridium perfringens* was isolated at very low frequency and *Streptococcus pyogenes* was not isolated from any cot mattress materials tested.

[1] Brooke *et al.* (1997) *Brit. Med. J.* 314, 1516-1520
[2] Tappin *et al.* (2002) *Brit. Med. J.* 325, 1007-1009

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016 Eight years after:an evaluation of SIDS awareness in the Tuscany Region

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In order to address the numerous social and scientific problems surrounding SIDS (Sudden Infant Death Syndrome), in 1996 a Regional SIDS Centre was founded at the Meyer Children's Hospital, the activities of which include:

- Home monitoring programs
- Clinical management of infants suffering from Apparent Life-Threatening Events (ALTEs)
- Support to bereaved families
- SIDS reduction campaigns.

In 1996 our first attempt to promote a"back to sleep campaign" involved the distribution of the booklet "Per loro è meglio/For them it's better" throughout the nurseries of our Region, however the effectiveness of this project has not yet been fully evaluated.
Moreover, in recent years the ethnic composition of our region has changed face and there is an increasing number of people of different communities who cannot be reached by the 'reduce the risk' message.
For these reasons in June 2003, the Regional Health Administration, the Regional SIDS Centre, the Regional Health Agency, and the Parents Association, Seeds for SIDS, agreed to promote an awareness campaign in Tuscany.
The strategic plan is based on the following steps:
1- Organization of a consensus conference with the directors of the three Vast Health Areas of our Region, the referees of family paediatricians, the referees of the nurses association, and the referees of the Parents Association Seeds for SIDS.
2-Organization of meetings with selected family paediatricians, district health managers, paediatric and obstetric hospital staff;
3-Data collection of SIDS awareness prior to beginning the campaign;
4-Distribution of printed material (booklets and posters) and diffusion of a short educational documentary by the regional network;
5- Data collection of SIDS awareness after the campaign.
Steps one and two have already been implemented and we are now completing data collection from a questionnaire on SIDS-risk-related infant care practices which was distributed between January 7 and February 28, 2004 to the parents of infants of approximately three months of age in 74 selected immunization centres. Immediately after filling out the questionnaire parents adopting unsafe practices are informed of the risk of SIDS. The data obtained will be evaluated in order to better define our educational efforts.

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017 Sudden Infant Death Syndrome (SIDS) And Infant Care Practices

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BACKGROUND: The rate of sudden infant death syndrome has been declining around the world and in Canada. However the rate of decrease of SIDS in Saskatchewan has been slower. The infant care practices and life style issues have been linked with the SIDS rate. There are very little information available on this in Saskatchewan. OBJECTIVE: We sought to elucidate some of these issues and practices surrounding Infant care and life style that are linked to SIDS in the Province of Saskatchewan Method: We analyzed demographic, infant care practice, and life style information that was available from the records of SIDS cases stored at Chief Coroners office in Regina and compared them with similar data collected from case comparison group that are matched for Gestation, Birth weight, Race, gender, place of birth and post natal age. RESULTS: Over a period of 13 years 258 SIDS cases and 235 case controls were reviewed. Unsafe infant care practices increased SIDS by three times.($p<0.01$) Unhealthy life styles also increased SIDS significantly. First nations infants showed 5.5 times greater risk of death than Non first nations peoples.($p<0.001$). CONCLUSION: The increased rate of SIDS in Saskatchewan was partly related to the increased death in First nations infants. Improvements in infant care practices and life styles are important elements that should be considered in the future strategies for SIDS reduction in Saskatchewan.

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018 Saliva: does it provide protection against Sudden Infant Death Syndrome?

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Historically it has been theorised that saliva, or at least the excessive production of saliva, may be a risk factor for SIDS. Also, until comparatively recently the main functions of saliva have been considered to be as an aid to digestion and protection against caries. However, more recent work has shown that saliva contains many extremely useful substances including antimicrobial agents and antifungals. Indeed, work on saliva assays, as an alternative to blood tests to detect and predict health problems, is an increasingly promising area of research. In addition various degrees of "dry mouth" have been shown to lower the body's defences against infection. Whilst not suggesting that xerostomia is a precursor of SIDS, it is clear that saliva production is at its lowest ebb during sleep. Therefore it is hypothesised that saliva could have a protective effect against SIDS either due to anti-infection agents or to the production of saliva inducing swallowing and possibly triggering brain activity and the ability to arouse from sleep. This theory is discussed in relation to some of the factors that have been suggested to provide protection against SIDS, for example pacifier use. Conversely, known risk factors for SIDS, such as sleeping, sleeping position, overheating, and tobacco smoke are considered in relation to saliva production. It is therefore considered that saliva, may be a useful and profitable area for future research.

019 Maori Grieving After the Loss of a Baby to SIDS

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Among Maori families the loss of an infant to SIDS is a terrible burden reverberating through their social networks for years after the event. The statutory services that investigate the death have a huge impact on such processes and have the ability to hugely magnify the trauma, or indeed to enhance the healing of the family. This qualitative investigation studied the experiences of Maori whanau following a SIDS death by reporting on narrative data gathered from the main players in the SIDS death scene and its sequel events. We interviewed the families, the investigating police officers, pathologists involved in the post-mortem and the Coroners responsible for the final reporting. Thematic analyses revealed multiple domains of immense significance to the determination of the impact of the death on the family. This paper covers a selection of these and considers their implications for appropriate service provision, enhanced grieving and social justice.

020 Survey of Bereaved Families in Japan

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SUBJECTS AND METHODS: In January of 2003 the SIDS Family Association Japan sent 480 surveys to all of our bereaved family members, 233 of them were returned to us by March 2003 (a return rate of 49%).
In our group of 233 the year of the baby's death ranged from 1980 to 2002. Most of the participants' babies died in 1997 to 1998. The cause of death was divided as follows: 6% miscarriage, 27% stillbirth, 22% perinatal death, 45% infant death.
RESULTS: At the time of death, bereaved families come into contact with hospital staff (79%) and medical doctors (88%) more than any other group. Around 30% of the hospital staff and medical doctors were rated to have Bad or Very Bad conduct to parents at the time of death. The data reveals that the police as a group rated lower than the other groups when it came to the treatment of bereaved families. For the police 40-50% were rated Bad to Very Bad as opposed to all other groups that were rated 20-30% Bad to Very Bad. Under 50% of our participants received adequate information and explanation at the time of death. Being given adequate information and explanation concerning the autopsy was even lower, under 40%.
The time with the baby who had died was not enough according to 57% of the participants. 35% received mementos and only 8% were encouraged to take photos. According to our data, follow-up care seems to be especially lacking. The data illuminates the fact that psychological follow-up care for families is not readily available or is not given in Japan. A full 93% were not able to consult a counselor, psychiatrist of social worker after the death and 90% were not given an introduction to any such professional. Also 67% were not invited to come back with questions after discharge in the months following the tragedy.

021 The SIDS Manual 2004

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Background: The value of acute care and follow-up regarding the family's bereavement process is widely acknowledged. We know that written routines contribute to better follow-up. Experiencing relatively low SIDS occurrence in Norway each year, there is an even larger need for a manual offering guidelines for appropriate admission and follow-up routines of families having lost their child suddenly and unexpectivly. "The SIDS Manual – admission routines and follow-up practices. Guidelines for health professionals dealing with SIDS or other sudden death in childhood" (from 1991) has been revised according to existing laws and regulations. The author is the Medical Advisory Board of the Norwegian SIDS Society. We have more than 900 subscribers, mainly hospitals and maternal and child health centres.
Goal: Contribute to improved routine bereavement services in hospitals and local health services, including acute and long-term follow-up of bereaved families due to the sudden deaths of children.
How: The SIDS Manual underlines the importance of actively offering respectful assistance through routine bereavement services. The need of (written) information both regarding cause of death, autopsy, practical issues, grief reactions and sibling needs is essential. The Manual also highlights the importance of professionals encouraging parents to take a personal and fulfilling farewell with the dead child. Further, the Manual underlines that professionals are responsible for contacting parental organisations for supplementary bereavement support. The Manual also consists up-to-date SIDS research information.
The Manual also includes: ·A consent note for notifying the Norwegian SIDS Society for parental support, if wanted.
·Ten short guidelines for public health nurses (in addition to the doctors and nurses)
·Background information concerning siblings and grief, gender and individual differences in grief reactions and expressions, and bereaved parents and their social network.
·Poems about the death of a child for distribution to parents
We experience big variations regarding admission and long-term follow-up of bereaved families. We hope the distribution by the Directorate for Health and Social Affairs will motivate subscribers to start using the Manual regularly.

022 Taking care of grieving parents at Naitre et Vivre France

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Since its creation, Naître et Vivre has had various different orientations depending both on the stage of its development and according to the region, where each orientation has been favoured to a greater or lesser extent. In the beginning, support was given mainly from the medical point of view and action undertaken to lead to the introduction of legislation. The next stage was providing information to parents on the degree of knowledge available on SIDS. And, now, support is given through offering a wide range of services, either providing information or accompanying bereaved parents in their psychological suffering.

On the subject of this evolution, it should be noted that the issue of offering support during bereavement in France has only very recently been put into practice and the association Naître et Vivre figures as a pioneer in the wider support given to grieving families. Naître et Vivre is the only association in France that offers support to parents grieving for a baby and as a result parents that have lost a baby through another cause than "Sudden Infant Death Syndrome" also turn to it. An evolution that can be found in the different services it provides, from the individual discussion, to the 24 hour telephone line open. It also organizes monthly thematic meetings, with specific contributors, bringing together parents from all horizons, in greater and greater numbers and very soon after the death has occurred. There are also "Support Groups" (accompanying the parents during 1 year in a closed group of 12 people + 2 trained counselors) with an ever-increasing demand on the part of the parents and an involvement and a desire to participate actively in the setting up of these groups.

France's efforts to prevent the fall of sudden infant death syndrome through information campaigns carried out in partnership (sometimes with the Ministry of Health) have dramatically reduced infant death syndrome from 2 per 1000 in the 90's to now only 0.44 per 1000. To give confidence back to grieving parents for their future while knowing how to be tuned in to their present suffering constitutes today the motivation of parents who have been trained as counselors (belonging to the Association Naître et Vivre).

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023 A support programme for anxious parents

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Robert Carpenter, London School of Hygiene and Tropical Medicine, London,
Robert Coombs, Royal Hallamshire Hospital, Jessop Wing, Sheffield,
Charlotte Daman-Willems, Children's Hospital, Lewisham, Lewisham,
Angela McKenzie, London School of Hygiene and Tropical Medicine, London,

Introduction: A support scheme for parents with children born following a cot death (CONI) has been extended to a wider range of families. The programme includes regular contact at home with a health visitor, movement monitor, symptom diary and weight chart. 147 of 193 districts in the UK offering CONI enrolled infants on CONI Plus.

Aims: To analyse the first 2000 infants enrolled on this programme between 1995 and 2003. Reasons for enrolment: ALTE (apparent life threatening event) at home 695 infants (35%); SIDS (sudden infant death syndrome) in close relative 559 infants (28%); previous sibling death (known cause) 164 infants (8%); mixed group including apnoea before discharge or more than one reason for enrolment 582 infants (29%). 96% of families found the programme helpful, 84% citing having a monitor and 63% regular HV contact as the most valuable elements. On average families saw their HV weekly, family doctor once in 5 weeks and hospital paediatrician once in 11 weeks

Results: Analysis of the two largest groups: ALTE group: 62% presented by age 2 months; 90% had been admitted to hospital following episode. An underlying diagnosis was identified in 331 (48%). 51% of parents reported a "true alarm" compared with 28% in other groups. Four infants in this group died, two suddenly and unexpectedly. SIDS in close relative: 89 families (16%) had experienced more than 1 previous unexplained death (range 2-7). Birth weights were greater (3179g vs 2831g; $p<0.0001$), gestational ages higher (38.8 weeks vs 35.6 weeks; $p<0.0001$) and a larger proportion of infants were born by normal delivery (80% vs 68%, $p<0.0001$) compared to other groups. There were four deaths in this group all attributable to SIDS (7.2/1000), an incidence 12 times the national rate.

Conclusions: The CONI plus programme provides valued support to a group of concerned families. There is a greater incidence of SIDS for infants born to families with a history of SIDS, although these families appear to exhibit fewer recognised risk factors for SIDS.

034 Treasured Babies' Program Supporting "Best Practice" in Perinatal Crisis Bereavement Care in Hospitals

Anne Giljohann, Nerida Mulvey, Karen Passey, SIDS & Kids Victoria, Australia

Outline: When a newborn child dies parents grieve, but our Community tends to 'disenfranchise' or dismiss their grief. Following miscarriage and stillbirth two frequent sources of additional distress for parents are the lack of acknowledgement of the existence of their baby, and the lack of recognition of their grief. Support and understanding are often not readily offered as the significance of the loss is not understood or acknowledged.

The Treasured Babies' Program (TBP), which was an initiative of an advisory group of bereaved parents at SIDS and Kids Victoria, aims to acknowledge and honor the life and death of newborn babies who have died, regardless of their gestational age, and to support bereaved parents in a practical and tangible way. This is done by providing sets of beautiful tiny hand-made clothes to health professionals, such as midwives, counselors, and funeral directors, so that the babies can be dressed and held by their parents, and dressed for burial.

Memories are created and mementoes made, which can help parents maintain a continuing link with their child. The meetings of volunteers who produce the TBP items function as an activity-based support group, and bereaved parents and others can be greatly supported by this program. The program also includes regular meetings of health professionals involved in perinatal bereavement, where ideas are exchanged in a supportive environment, and the feedback and experience of bereaved parents is welcomed.

This program began in July 2002 and was piloted with five Victorian hospitals from both metropolitan and rural areas. These hospitals were supplied with a range of 'TBP gifts' sets of clothes packed according to gestational age. SIDS and Kids recently commissioned an evaluation of TBP and the findings clearly indicate that families benefit from being given appropriate tiny clothes at the time of the death, and also staff who are caring for the families benefit from having readily available sets of clothing that are beautiful and suitable.

This presentation will use the literature relating to loss and grief to outline a clear theoretical basis for understanding how and why the TBP is helpful and effective. So both the experience of parents and staff, as well as theory, confirms its value.

Outcomes: SIDS and Kids has a goal of ensuring that good bereavement care is provided to families by hospitals. TBP can facilitate a process of education and information sharing regarding the hospital's role in crisis bereavement support.

The TBP 'gifts' enhance the bereavement support provided to parents by hospital staff following a perinatal death. Appropriate clothing supports and encourages parents to hold their deceased babies 'saying hello and goodbye'. The fact that the clothes are beautiful acknowledges and honors the life and death of the babies, and acknowledges and respects the validity and depth of the parents' grief. TBP has the potential to become a significant strategic tool in developing a link with hospitals throughout Victoria to develop 'best practice' guidelines for crisis bereavement support.

049 Tobacco Talk for Two: A Prenatal/Postnatal Group Tobacco Reduction Toolkit

Gail L Foreman, Heidi M Olstad, Sylvia Baran, David Thompson Health Region, Red Deer, Alberta

Canada Prenatal Nutrition Projects work with pregnant women to reduce their risk of delivering low birth weight babies through a variety of methods (e.g. tobacco reduction). The current smoking rate of CPNP participants in the David Thompson Health Region is above 50%, which is significantly higher than the provincial average of 32% during pregnancy. To increase the tobacco reduction component of Canada Prenatal Nutrition Projects (CPNP) in DTHR, Tobacco Talk for Two, a tobacco reduction toolkit was developed and evaluated.

A coordinated approach to address tobacco reduction with CPNP in DTHR was taken due to consistently high smoking rates across the region. Project coordinators, outreach workers and participants discussed possible approaches to address tobacco use. Participants stated they would not attend a class solely focused on tobacco, however, would be interested in tobacco reduction information that was integrated into regular programming. It was decided that 15-minute modules, to be used during group and individual sessions would meet their needs. A draft manual was developed for CPNP staff to review and evaluate. Changes to the manual were made based on input received. Tobacco Talk for Two manuals and training were provided to the CPNP projects for implementation.

Tobacco Talk for Two was designed to facilitate constructive conversation about tobacco with CPNP participants through the implementation of brief interactive modules that are non-judgmental, appropriate for both smokers and non-smokers, and based on the stages of change model. The interactive modules include: Getting the Facts on Smoking and Pregnancy, Benefits of Quitting, Secondhand Smoke, Understanding Why I Smoke, Support for Quitting, Aids to Stop Smoking, What Will Happen When I Quit, Coping with Situations that Make you Want to Smoke and Staying Quit. Each module includes specific goals and objectives, background information for the facilitator, facilitator's notes, participant activity sheets and handouts, and evaluation forms.

The evaluation included pre-post provider capacity surveys, participant evaluation of interactive sessions, facilitator evaluation of interactive sessions, and a comparison of the facilitators' plans to use the manual with actual manual use. Data is currently being analyzed and preliminary results will be available in April 2004.

050 Smoking Cessation During Pregnancy: A Coordinated Regional Approach

Sheri Jickling, Alberta Alcohol and Drug Abuse Commission, Grande Prairie, Alberta
Saara Bain, Peace Country Health, Grande Prairie, Alberta

The goal of this project was to implement a coordinated approach to decrease smoking during pregnancy in a rural northern Alberta region. This region was comprised of a population of approximately 90 000 (2001) in an area covering 23 000 square miles. Access to and provision of services was challenging due to the large, sparsely populated geographical region.

The project goals were to:

· Increase comfort and capacity of various providers (health professionals, Canadian Prenatal Nutrition Program (CPNP) workers, addictions counselors, and pharmacists) to address smoking during pregnancy
· Adapt and implement a brief intervention approach
· Adapt and distribute pregnancy specific cessation resources
· Identify and implement social supports for high risk women.

The project was conducted over three phases. The project is now pursuing a next stage; broadening the brief intervention training, integration, and evaluation in a much larger northern region.

PHASE I: Focus groups. This included a baseline survey of 33 pregnant or parenting women and 110 service providers. Non-identifying results will be displayed that summarize the findings. Regionally smoking rates were at 32.5% vs 28% provincially (source: Alberta Health Surveillance, 1999). A CPNP funded program had reported an exceptionally high rate with 63 % of clients responding "yes client smokes" in 1999-2000. Regionally 1379 new births were reported in 2002. According to these percentages 448-869, children were born to mothers who smoke in this area.

PHASE II: Training. Trainers from the Ontario Tobacco Strategy, were brought to the region and provided training over a 2 day period to a total of 53 service providers and "trainers". Training focused on implementation of 5 A's brief intervention approach. Distribution of over 500 quit kits, including Need Help Putting Out That Cigarette? print resource to participants for use in their practice.

PHASE III: Social support and community based groups were launched in four sites, conducted by a Health Promotion Facilitator and Peer Educator. Groups included monthly incentives, snacks, meals and childcare. Additionally this phase included weekly support calls, mail outs and adaptation of a Smoke Free Homes resource. Photos', experiences, successes and barriers will be presented.

051 Hard Cases - changing smoking in challenged families

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In New Zealand, one in three babies is born to a woman who smokes, two in three for Maori. If interventions are to make a difference, they need to make a difference for women like Lara. In her own words: "I am a twenty-one year old Maori woman, the solo mother of two children and on the DPB (Domestic Purposes Benefit). My Dad's never been in the picture. My Mum kicked me out at fourteen so I lived on the street. At seventeen I got pregnant and my partner took me in. It was a violent relationship lasting four years."

Lara was a participant of Smokechange (a programme of free, personalized support to change smoking, especially in pregnancy). She was one of the programme's many "hard cases" where the intervention time invested seemed to achieve very little change to smoking itself. Yet Lara used what she learned from Smokechange to change other more urgent problems in her life and in her own time also became smokefree.

This presentation will draw on traditional learning from my Ngai Tahu (Southern New Zealand Maori) childhood and apply it to a smoking intervention. Through the analogy of building boats, where the most battered timber is the most strong, most resilient and most suitable for the bow, the presentation will demonstrate the application of strength-based practice to working with "hard cases". It is our experience that when supported to recognise the strengths they have gained from their struggle, young women like Lara build confidence in their own ability to make changes in their lives, even to smoking.

For 339 participants in our Smokechange evaluation group, 92 were Maori. Participants with high readiness scores increased from 13% to 52% over three months participation, for both Maori and non-Maori. Participants smokefree increased from 2% for both groups at enrolment, to 28% for Maori and 33% for non-Maori at second assessment three months later.

We have found that a strength-based philosophy is effective in achieving changes to smoking in pregnancy for all our participants, including those more challenged.

024 Analysis of villous vasculature in placentae from sudden infant death syndrome (SIDS) infants with and without intrauterine growth restrction (IUGR).

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Background: SIDS is still a leading cause of infant death in industrialized countries and shows a marked correlation with IUGR. Our main objectives were to investigate placental vascular development and to identify changes in morphometry associated with SIDS and/or IUGR.
Method: All placentae were delivered at term via spontaneous vaginal delivery and were categorized, based on foetal birth weight, as control (n=10) and IUGR (n=10). SIDS placentae were also categorized based on foetal birth weight as either SIDS normal birth weight (NBW n=10) or SIDS IUGR (n=9), [above (NBW) or below (IUGR) the 10th centile for gestational age]. Each placenta was uniform randomly sampled; samples were processed to wax and immuno-histochemically stained using a CD34 antibody. Design based stereological techniques were used to estimate a number of volumetric and surface area parameters. Results were analysed using one way ANOVA. Control cases were compared with SIDS NBW cases and IUGR cases compared with SIDS IUGR.
Results: No significant differences were noted in placental weight and volume, terminal villous capillary volume or surface area (SA). However, a significant difference was noted in the SA of arterioles and venules in the intermediate villi ($p=0.016$) between control and SIDS NBW cases. Additionally a trend towards a difference in the intermediate villous arteriole/venule shape factor (a measure of branching complexity) was noted between control and SIDS NBW cases.
Conclusion: The trends towards reduction of capillary volume observed in both the IUGR and SIDS IUGR groups is probably due to factors associated with IUGR rather than SIDS. Neither SIDS nor IUGR associated factors have an effect on capillary surface area. However, surface area development of the arterioles/venules may have been subjected to SIDS factors rather than IUGR factors. The outcome of the arterioles/venule shape factor (if confirmed by extra cases) suggests that this vasculature may be less branched and more dilated in SIDS cases than in non SIDS cases.

025 Investigation of sudden infant death syndrome and intra uterine growth restricted placentae using HO-1 & HO-2, VEGF and CD68.

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Background: A previous study showed no significant difference in oxygen diffusive conductance (Dp) in placentae from SIDS infants when compared with age matched controls, suggesting that, although morphological differences were present in SIDS placentae, magnitudes were not severe enough to impinge on oxygen transfer.
Aim: We hypothesize that vascular remodelling or some form of compensatory mechanism is initiated in SIDS placentae to ensure optimum transfer. This was investigated using a number of growth factors that play a role in angiogenesis and vascular resistance.
Method: All placentae were delivered at term via spontaneous vaginal delivery and were categorized, based on foetal birth weight, as control (n=10) and IUGR (n=10). SIDS placentae were also categorized based on foetal birth weight as either SIDS normal birth weight (NBW n=10) or SIDS IUGR (n=9), [above (NBW) or below (IUGR) the 10th centile for gestational age]. Each placenta was uniform randomly sampled; samples were processed to wax and immuno-histochemically stained using the following antibodies: CD68, VEGF, and HO-1 & HO-2.
Results: CD68: There was a significant increase in the density of positively stained CD68 cells within the villous stroma in the IUGR group when compared with SIDS IUGR group ($p=0.013$) and control cases ($p=0.001$). No difference was noted between control and SIDS cases. HO-1 and HO-2: There was no statistically significant difference in the density of positively stained cells located within the trophoblast or the stroma for either HO-1 or HO-2 between any of the groups analysed. VEGF: There was a significant increase in the density of cells stained positively for VEGF within the villous membrane for IUGR, SIDS NBW and SIDS IUGR when compared to controls ($p<0.001$).
Conclusion: Macrophages play a crucial role in angiogenesis due to their expression of VEGF, a potent angiogenic growth factor. Increased levels of CD68 (IUGR) and VEGF (IUGR, SIDS NBW and SIDS IUGR) at term may represent altered patterns of vasculogenesis since "normally" VEGF expression decreases towards term. This may result in limited oxygen transfer since defective vasculature may not have been compensated for by increased levels of HO and its' by product, carbon monoxide a powerful vasodilator.

026 Identification of villous membrane apoptosis in sudden infant death syndrome (SIDS) and intra uterine growth restricted (IUGR) placentae using M30 and BCL2.

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Background: The villous membrane represents the greatest resistance to the transfer of oxygen between the developing foetus and the maternal inter-villous space. There is a trend towards a decrease in the harmonic thickness in both SIDS and IUGR placentae when compared with controls. Based on this observation we hypothesized that there is an increase in the rate of apoptotic turnover within the villous membrane which may be contributing towards the reduced villous membrane thickness.
Method: All placentae were delivered at term via spontaneous vaginal delivery and were categorized, based on foetal birth weight, as control (n=10) and IUGR (n=10). SIDS placentae were also categorized based on foetal birth weight as either SIDS normal birth weight (NBW n=10) or SIDS IUGR (n=9), [above (NBW) or below (IUGR) the 10th centile for gestational age]. Each placenta was uniform randomly sampled; samples were processed to wax and immuno-histochemically stained using M30 and Bcl2 antibodies. The density of positively stained syncytiotrophoblast (syn) nuclei and cytotrophoblast (cyto) cells was estimated. Results were analysed used one way ANOVA. Control cases were compared with SIDS NBW cases and IUGR cases compared with SIDS IUGR.
Results: M30: results show a trend towards an increase in the density of both positively stained syn nuclei and cyto cells in both SIDS NBW and SIDS IUGR cases when compared with control cases. However, these results did not reach statistical significance. Bcl2, these cases are currently being analysed.
Discussion: An increase in the number of syn nuclei and cyto cells experiencing cell death in the SIDS cases only suggests a SIDS specific feature. If confirmed on the addition of further cases, this suggests a defective villous membrane in SIDS cases which is not seen in IUGR cases.

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028 Detection of specific antibodies in cord blood and infant saliva to staphylococcal toxins implicated in sudden infant death syndrome (SIDS)

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Introduction: The common bacterial toxins hypothesis of sudden infant death syndrome (SIDS) is that nasopharyngeal bacterial toxins can trigger events leading to death in infants with absent/low levels of antibody that can neutralise the toxins [1].
Aim: To investigate nasopharyngeal carriage of *Staphylococcus aureus* (*S. aureus*) in the first year of life and determine levels of specific antibody to toxic shock syndrome toxin (TSST-1) and staphylococcal enterotoxin C (SEC_1) in samples of cord blood and infant saliva.
Subjects and methods
Seventy three mothers and their infants (39 males and 34 females) were enrolled in the study. Cord blood samples (n=66) were collected by midwives. Saliva samples and nasopharyngeal swabs were obtained from infants monthly for the first year of life. Nasopharyngeal swabs were cultured on blood agar and *S. aureus* isolates were identified using standard methods. The isolates were tested for production of TSST-1 and SEC_1 using a dot blot assay. Quantitative ELISA methods were used to detect IgG to TSST-1 and SEC_1in cord blood samples and IgA toTSST-1 and SEC_1 in infant saliva.
Results: Analysis of cord blood samples detected IgG bound to TSST-1 and SEC_1 in 95.5% and 91.8% of cases respectively. There was a marked variation in levels of maternal IgG to both TSST-1 and SEC_1 among cord blood samples. Risk factors for SIDS, maternal age, birth weight, and seasonality, significantly affected the levels of IgG binding to TSST-1 or SEC_1. Between 40 and 50% of infants were colonised with *S. aureus* in the first three months of life and 49% of the isolates produced one or both of the staphylococcal toxins. Analysis of infant saliva samples detected IgA to TSST-1 and SEC_1 in the first month after birth. Eleven percent of samples tested positive for salivary IgA to TSST-1 and five percent for salivary IgA to SEC_1. By the age of two months these proportions had increased to 36% and 33%.
Conclusion: Passive immunity to toxins implicated in SIDS cases varies greatly among infants. Infants are able to mount an active mucosal immune response to TSST-1 and SEC_1 in the first month of life.

[1] Morris, J.A., Haran, D. and Smith, A. (1987) Hypothesis: common bacterial toxins are a possible cause of the sudden infant death syndrome. Med. Hypotheses 22, 211-222

029 Epidemiology of Apparent Life-Tthreatening Events (ALTE) - is there a relation to SIDS risk factors?

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Objective - To investigate potential risk factors and demographic characteristics of ALTE and to examine an association between SIDS and ALTE in terms of shared risk predictors.
Methods - A prospective cohort study carried out in Tyrol, Austria, enrolled all live-born infants between 1993 and 2001 (n = 44,184; participation rate 86%). Data on pregnancy, delivery, sociodemographic characteristics, child care practices and infant's behaviour in the first four to six weeks of life were collected with a standardised questionnaire. ALTE was identified from hospital admission records.
Results - During the study period the mean annual incidence of ALTE was 2.46/1000 live births. The peak age-at-onset was nine weeks. Of the 164 ALTE cases clinical comorbidity was detected in 91 (55%). Vice versa, in 73 infants no cause and no concomitant disease for the event could be identified; these events were called "idiopathic ALTE".
The prone sleeping position, as the most important risk factor for SIDS, showed a modest relation to the risk of overall and idiopathic ALTE, which was not statistically significant when controlling for the multiple comparisons performed. The SIDS prevention programme in Tyrol did not considerably lower the frequency of ALTE and none of the ALTE infants experienced SIDS in later life.
In a multivariate risk model, family history of infant death (odds ratio (OR), 1.7; 95% confidence interval (CI), 0.9 to 3.0) and behavioural abnormalities like feeding difficulties (OR, 2.2; 95% CI, 1.4 to 3.4), episodes of pallor (OR, 2.3; 95% CI 1.3 to 4.1), cyanotic episodes (OR, 4.1; 95% CI, 2.3 to 7.2) and repeated apnoea episodes (OR, 7.0; 95% CI, 3.9 to 12.3) all were associated with an increased risk of ALTE.
Conclusions - Although there are some similarities in the clinical presentation and epidemiology of SIDS and ALTE, differences clearly predominate. Accordingly, ALTE and SIDS should not be considered different manifestations of the same disease process.

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032 Crib Death: Cardiac Sampling and Study of the Conduction System

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Crib death represents a great enigma, one of the main open issues in the social-medical and scientific setting of modern medicine. The aim of the present work is to describe the anatomo-pathological techniques to study the heart and particularly the conducting tissue in every case of crib death. At autopsy, the heart is removed taking care in severing the great vessels close to the pericardial reflections. The heart size and weight should be compared with the normal values. After the presence of gross cardiac malformations are excluded, the origin of the coronary arteries should be carefully inspected. The major epicardial coronary arteries and branches are excised transversely to their longitudinal axis. To examine the cardiac conduction system, two blocks for paraffin embedding are prepared. The first block contains the junction of superior vena cava and right atrium encompassing the entire area of the sinus node. The main visual reference for removal is the Sulcus-*Crista Terminalis*. Two longitudinal cuts are driven, parallely to the sulcus-crista line, through the atrial wall, with a medial prolongation on the right side to encompass the anterior aspect of inlet of the superior vena cava; on the left side, one has to section very medially the sinus intercavarum and prolong the cut on the superior vena cava wall. Of the two transverse cuts, the superior one is oriented to the removal, as much as possible of the cava funnel. The inferior cut removes, more or less distally, the fan of the pectinate muscles that radiate from the Crista Terminalis. The second block contains the atrio-ventricular node, His bundle down to bifurcation and bundle branches, with two centimeters of attached septum above and below. Holding the already opened heart so as to expose the interventricular septum against a fairly intense light source, one clearly spots the transparent area of the *pars membranacea* and pins it between thumb and index finger. Thereupon, one proceeds to excise the interventricular septum together with the central fibrous body, the lowermost part of the atrial septum and the adjacent segments of the AV fibrous annuli. The serial section is a must in conduction system investigation, since it allows a further tridimensional reconstruction of the examined conducting tissue. The sinus node block is cut serially sectioned in a plane parallel to the crista terminalis. The atrio-ventricular junctional block is serially sectioned in a plane parallel to the two atrioventricular valve rings. Intercalated section every 20-40 □ m interval for the sinus node block and 40-60 □ m interval for the atrio-ventricular block, collecting 3 sections of 8 □ m each at individual levels, were performed. For each level two sections of 8-□ m were saved and stained alternately with hematoxylin-eosin and trichromic Heidenhain (azan). All intervening sections were kept and stained as deemed necessary. For each heart, the average number of histological sections stained and examined is about 200.

035 Helicobacter Pylori and SIDS

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Background
A large proportion of SIDS victims have a stimulated mucosal immune system at the time of death. It has been proposed that an overreaction to a seemingly harmless infection with for example Respiratory syncytial virus or Haemophilus Influenza can lead to SIDS. We have evaluated the role of H. pylori infection in infant death.
Hypothesis
H. pylori infection contributes to the "infectious load" in infectious deaths and in SIDS.
Methods
Stool specimens from 227 cases of infant deaths (0-3 years) have been collected at autopsy between the years of 1993-2003. Stool specimens from live infants were collected in a maternity ward. The specimens were stored at –700C. H. pylori infection was confirmed by the presence of H. pylori antigen in the stool, by means of EIA (Premier Platinum HpSA, Median diagnostics, Cincinnati, United States). SIDS victims and the other cases of infant deaths were further examined immunohistochemically with regard to IgA-, IgM- and IgG- immunocytes in ventricular and duodenal mucosa.
Results
H. Pylori antigen was found in 32% of the infectious deaths, 24 % of the deaths due to non-infectious disease, 19 % of the SIDS cases and in 14 % of the accidental deaths. In the live control group 14 % had H. pylori antigen in stool (preliminary data only). H. pylori infection seems to be more frequent among newborn babies (0-1 months) than in toddlers (12-36 months). No differences in the B-cell response in the gut mucosa were observed with regard to the presence of H. pylori infection.
Discussion
The results indicate that victims of infectious deaths are frequently co-infected with H. pylori, which may be interpreted as a general predisposition to infection. However, the findings do not disclose a role for H. pylori infection in the pathogenesis of SIDS. The control group consisting of accidental deaths and live infants, had a frequency of 14 % H. pylori antigen in stool, which is unexpectedly high compared to prevalence studies among children in other western countries. Further studies of immune reactions in the present fatal cases may reveal mechanisms relevant for the death mechanism.

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036 IL-6 and risk factors in SIDS

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We have previously shown that a substantial amount of the SIDS cases have increased levels of interleukin-6 (IL-6) in their cerebrospinal fluid (csf). This may be due to an overstimulation of the immune system as a response to an otherwise harmless infection. Furthermore, in a previous study, as well as the present one, we have found that the high IL-6 levels probably cannot be explained by a deficient production of the regulatory cytokine IL-10. The objective was to study whether the IL-6 concentration differs according to risk factors. SUBJECTS AND METHODS: 57 SIDS and borderline SIDS cases from the eastern part of Norway were divided into three age groups; 1-6 (44 cases), 6-12 (6 cases) and 12-36 (7 cases) months. IL-6 and IL-10 were measured by ELISA (R&D systems). Information about the cases was obtained from autopsy records and questionnaires to parents. RESULTS: Dividing the cases in high and low IL-6 groups, the high IL-6 group contained the oldest babies ($p< 0.0001$). Furthermore, the oldest babies most often had fever ($p=0.008$), and they also were found dead in a prone position, while the babies in the youngest group more often were found on their back or side ($p=0.032$). The babies/children that were found dead in a prone position more often had symptoms of an infection (signs of a cold and/or fever) prior to death ($p=0.039$). There were no significant correlations between IL-6 levels in csf and signs of infection prior to death or sleeping position at death. DISCUSSION: Several studies have shown that the ability to produce IL-6 is independent of age, so differences in age per se probably do not explain why the oldest children have higher IL-6 concentrations. However, since it has been shown that both fever and passive warming will induce IL-6 production, it is possible that the combined effect of sleeping position and fever, as found in the oldest children, are of importance. The study will be extended.

030 Correlation between the NMDA receptor and neuronal death in the SIDS medulla.

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N-methyl-D-aspartate (NMDA) receptors have many associations with hypoxia, including a role in the hypoxic ventilatory response (1) and changes in expression and function after hypoxic stimulation (2,3). Changes in NMDA receptor activity, whether too much or too little, can also precipitate cell death. Excessive activation of NMDA receptors leading to cell death is called excitotoxicity. This study tested the hypotheses that NMDA expression is increased in the SIDS medulla (presumably due to hypoxia prior to death), and that increased NMDA expression correlates with increased neuronal death.
Data were compared between populations of non-SIDS (n=10) versus SIDS infants (n=15), and between SIDS victims who usually slept prone (n=4) to those who slept non-prone (n=11).
Immunohistochemistry for NMDA receptor 1 (NR1) protein was performed either alone or combined with TUNEL staining, to detect DNA fragmentation as an indicator of cell death. Sections were formalin fixed/paraffin embedded, and 7mm thick at the level of the mid medulla. Eight nuclei were examined.
In SIDS infants, NR1 protein expression increased in the dorsal motor nucleus of the vagus ($p=0.04$) and decreased in the nucleus of the spinal trigeminal tract ($p=0.03$) compared to non-SIDS infants. SIDS infants who usually slept prone had decreased NR1 protein in the vestibular nucleus compared to those who usually slept non-prone ($p=0.02$).
No differences were observed between the SIDS and non-SIDS group for NR1 protein and TUNEL co-localisation (NR1/TUNEL), but SIDS infants who usually slept prone had increased NR1/TUNEL in the arcuate nucleus (AN) compared to those who usually slept non-prone ($p=0.05$).
As a group, SIDS infants have altered NMDA expression in brainstem nuclei, including those that regulate cardiorespiratory function. The alterations seen in SIDS infants may be the result of environmental insults, and this preliminary study provides evidence for excitotoxic injury in the AN of SIDS infants who usually slept prone.
1-Ohtake et al., (2000), Am J Respir Crit Care Med. 162: 1140-47.
2-Hoffman et al., (1994), Neurosci Lett. 167: 156-60.
3-Machaalani and Waters (2002), Brain Res. 951: 293-300.

031 Sampling Techniques of the Brainstem in Sudden Infant Death Syndrome (SIDS) and Sudden Unexpected Perinatal Death

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Recent observations have identified, both in SIDS and sudden unexpected perinatal death victims, frequent developmental abnormalities in the brainstem, particularly in the arcuate nucleus, an important cardio-respiratory center of the ventral medullary surface. The aim of the present work is to describe the anatomo-pathological techniques that can be adopted for the study of the respiratory cardio-vascular and arousal coordinating structures of the brainstem. A simplified procedure for the routine study of brainstem structures, starting in fetuses after the 25th week, newborns and in infants will be presented, applicable in all histopathological laboratories. However, it requires a careful and precise sampling. The sampling procedure consists of the examination of the 3 portions of the medulla oblongata, in order to observe the inherent nuclei (arcuate, olivary, hypoglossus, ambiuus, dorsal vagal, tractus solitarii nuclei, etc.) in their cranial, intermediate and caudal portions, and of the pontine-mesencephalic portion from the cranial portion of the pons to the caudal portional of the midbrain, in order to examine the parabrachial complex/Kölliker Fuse nucleus. This method is applicable in every laboratory. The medulla oblongata is divided into three blocks. The first cranial one is extended by the border between medulla oblongata and pons up to the upper pole of the olivary nucleus. The second, intermediary one, correspondent to the sub-median area of the inferior olivary nucleus, has as point of repere the obex and it is extended 2-3 mms above and under the obex itself. The third, caudal one, includes the lower pole of the inferior olivary nucleus and the lower adjacent area of the medulla oblongata. The first and the second blocks, including respectively the upper third of the medulla oblongata with the adjacent portions of the pons and of the medulla oblongata adjacent to the obex, are sectioned in a cranial-caudal direction. The third block, correspondent to the lower portion of the medulla oblongata, is sectioned instead in a caudal-cranial direction. The fourth sample from the pons and midbrain is sectioned in caudo-rostral direction. From each of these paraffin-embedded blocks, 6 groups of 12 serial sections are obtained. Thus, comparing with the complete method, this simplified one allows a remarkable reduction in the number of the histologic slides to perform. The four blocks are kept and sectioned as deemed necessary. With this simplified procedure it is possible not only to recognize the different nuclei, and particularly the arcuate nucleus in its variable extension, but also to compare them at correspondent levels.

033 Experimental Simulation of the Geomagnetic Activity Correlated

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Sudden Infant Deaths (SIDS) occur in temporal clusters that include large geographical areas. For a two year period the monthly incidence of SIDS in Ontario was significantly correlated with specific bands of intensity of global geomagnetic activity. Subsequent analyses indicated that geomagnetic pulsations within the 0.1 Hz to 0.5 Hz range were specifically correlated with these bands of intensity. We have both experimental and correlational data that weak (around 20 nanoTesla), complex magnetic fields whose temporal structure simulates neurophysiological patterns can increase the incidence of electrical seizures in epileptic rats. Our working hypothesis is that the specific temporal pattern of some geomagnetic pulsations over several hours can "drive" neurons (e.g., respiratory neurons that are sensitive to 0.5 Hz stimulation) within the vulnerable brain stem to produce SIDS. Over several blocks of experiments 20 pregnant rats that were exposed to 10 nT, 0.5 Hz sine-wave magnetic fields during the 48 hr preceding parturition produced about 2 fewer pups per litter than did the 20 pregnant females exposed to the control conditions (effect size about 15%). Fields generated in the east-west direction were associated with more deaths in male pups and fewer neurons within the solitary nucleus but not the parasolitary nucleus. Some rats when reintroduced as healthy adults to the same experimental magnetic configuration in which they were born died suddenly when sudden decreases in the earth's magnetic field occurred. The latter results are consistent with the association between increased SIDS on the same days as increased admissions for cardiac arrhythmias in adult humans. The results suggest that specific geomagnetic configurations are associated with SIDS and that similar phenomena can be reproduced in the laboratory

037 Postnatal metabolic and neurochemical development of brain stem respiratory nuclei in rats: Potential implications for Sudden Infant Death Syndrome

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Ninety percent of SIDS occurs in the first 6 months of life, with a peak at 2-4 months, implying strongly that a vulnerable period exists in postnatal development. Our laboratory has been probing for clues of a critical period by analyzing the metabolic and neurochemical development of brain stem respiratory nuclei in normal rats from postnatal day (P) 0-21. We found that at P11-13, there was a rise-fall-rise pattern of metabolic activity revealed by cytochrome oxidase (CO) in the pre-Bötzinger complex (PBC), the presumed center of rhythmogenesis. The abrupt fall in CO activity on P12 coincided with a fall in the expression of excitatory neurotransmitter (glutamate) and receptors (NMDAR1) and a rise in the expression of inhibitory neurotransmitter (GABA) and receptors (GABABR and glycine receptor). Similar changes were found in other brain stem respiratory nuclei (such as the ventrolateral nucleus of the solitary tract and upper airway motoneurons of the nucleus ambiguus), suggesting that the system is under excessive inhibitory drive and reduced excitatory drive at P12. Carotid body denervation induced a significant decrease in CO activity and a distinct delay, as well as prolongation of the maturational process, especially when induced close to P11-13. The fact that carotid body denervation could only delay and prolong, but not eliminate, the rise-fall-rise pattern of metabolic development indicates that it is largely genetically programmed. Developmental changes in synaptic transmission could also involve receptor subunit switches reported in various brain regions. We found that the expression of GABAA □ 3 subunit declined with age, whereas that of □ 1 subunit increased with age, and the two trends intersected at P12 in the PBC. Such apparent switches may be the basis for less efficient inhibitory transmission before P12 and a more mature inhibitory effect after P12. Thus, during the presumed critical period, the animal may be less able to overcome the detrimental effects of exogenous respiratory insults. If such periods exist in humans, and if exogenous stressors are introduced in an infant that has some vulnerable attributes, it is possible that catastrophic events, such as sudden death, may result. (Supported by Children's Hospital Foundation, Milwaukee, WI).

038 SIDS is not associated with a temperature dependant Brugada syndrome mutation.

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Inherited cardiac arrhythmias have been suggested as a cause of some cases of SIDS, and mutations in the long QT syndrome genes have been described in a few infants that had succumbed to sudden unexpected death. Brugada syndrome is an autosomal dominantly inherited disease with low penetrance, which is more prevalent in males. It is characterised by abnormal electrocardiographical (ECG) changes that can induce sudden cardiac death due to ventricular fibrillation in patients with structurally normal hearts. Most deaths occur during sleep, as is the case in SIDS. Gene defects linked to the Brugada syndrome are found on the cardiac sodium channel (SCN5A) gene, where mutations causing the long QT syndrome are also found. Case reports have suggested that fever induces Brugada syndrome. One mutation on the SCN5A gene, T1620M, has proven to be arrythmogenic only at temperatures over the physiological range, suggesting that some patients are more at risk during a febrile state. Approx. 20% of SIDS cases from southeast Norway have had fever in the last days prior to death. The objective was to search for the T1620M mutations in SIDS cases from our region, and if present to relate the findings to history of fever prior to death. SUBJECTS AND METHOD: 198 pure-SIDS cases, 26 borderline-SIDS cases with signs of infection prior to death, and 38 cases of sudden unexpected death due to infectious disease were investigated. DNA was prepared from tissue samples, PCR performed to amplify the DNA product and restriction enzyme added to search for the mutation. RESULTS: The T1620M mutation was not found in the cases studied. DISCUSSION: Several mutations in different cardiac genes known to cause sudden cardiac death have been described in the last few years, and some of them are thought to be involved in sudden unexpected death in infancy and childhood. The failure to demonstrate the T1620M mutations in our subjects does not rule out involvement of inherited cardiac arrhythmia as an explanation in some of our SIDS cases.

039 Sudden infant deaths in car seats

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Episodes of decreased oxygenation have been recorded in infants positioned in car seats. It is believed that former premature infants traveling in car seats are at risk of life-threatening events and even death. However, there is no data available on the prevalence of such events. The aim of our study was to determine exactly the incidence of infant sudden death occurring in car seat or similar devices. We therefore reviewed the circumstances of death in all cases of sudden unexpected death between 0 and 1 year of age from 1991 to 2000 in the province of Quebec. RESULTS: There were 527 deaths during that time period for which we knew exactly the circumstances and where exactly the infant died; this represented 96% of the total number of sudden deaths in infancy. Of these deaths, 408 were unexplained and 119 were explained deaths. There were 16 deaths in a car seat or a similar device, of which 9 (2%) occurred in the unexplained death group and 7 (6%) in the explained death group (NS, chi-square analysis). The post-conceptional age of the infants who died in car seats was not different from that of infants dying in beds (51.7 wks vs. 52.1 wks). The proportion of prematurely born infants was not different between groups (in car seat ; 18.7%; in bed: 15.2%, NS). Most of the deaths occurring in car seats (or similar devices) did not occur during travelling but at home. CONCLUSION : Very few infant deaths occurred in car seats in our population and the demographic characteristics of infants dying in car seats are similar to those of infants dying in beds.

040 Sudden unexpected death in children 2 to 18 years

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Sudden and unexpected death in children is not a frequent phenomenon although the exact incidence is poorly reported. Careful investigation of such deaths may yield interesting information, especially when compared to analogous deaths in the infant age group. We therefore reviewed the coroner's investigation of all sudden deaths having occurred during the ten-year period from 1991-2000 in the province of Quebec (Canada) in children from the age of 2 to 18 years. Of the 3127 cases reviewed, and after eliminating all accidental deaths and deaths that were not truly sudden and totally unexpected, we were left with 90 cases for which there was no clue as to the cause of death before the investigation. A definitive cause of death was found in 71 instances (79%). The most common category for the causes of death was a heart condition (27 cases) with hypertrophic cardiomyopathy, coronary artery anomaly and myocarditis being the most frequent findings. An infectious cause was found in 11 cases and a CNS problem in 8 cases. Only 44% of the cases occurred during a sleep period, the other deaths having occurred during wakefulness with some during a physical activity (mostly cases with a heart condition -15 instances). During the same period in the province of Quebec, there were 527 sudden unexpected deaths in infants (7 to 365 days), of which 119 were fully explained (23%); 93% of all cases occurred during sleep. In summary, sudden unexpected deaths are rare occurrences in children and a cause of death is much more often found than when the sudden unexpected death occurs in infancy.

041 Sudden Infant Death While Being Breastfed

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Sudden infant death while breastfeeding is a rare event. We present two infants who were discovered unresponsive while breastfeeding. The first, a 1 month, 14 day old male was being breastfed by his mother in a movie theater when she discovered him unresponsive and apneic with red fluid coming out of his nose. CPR was unsuccessful after 5 hours of effort. Postmortem examination revealed pleural and thymic petechiae. Toxicology and metabolic screening, blood cultures, and vitreous electrolyte analysis did not provide an explanation for the death. The second case was a 5-week old male who suddenly stopped breathing while being breast-fed and being carried in a sling. The mother noted a small drop of blood in one of the nares and generalized pallor. CPR was instituted and he expired 4 days later in the hospital. Salicylic acid, acetaminophen, alcohol, cocaine, opiates, methamphetamine, and phencyclidine were not detected in the blood, and the basic drug screen was negative. Postmortem examination revealed myocardial necrosis, diffuse alveolar damage, thymic atrophy, but no retinal hemorrhages. Vitreous electrolyte analysis and metabolic screening were not performed. Airway obstruction while breastfeeding was considered the cause of death of both infants. These cases are compared with previously reported ones. We conclude that absent attention to the infant's airway patency, breastfeeding is capable of causing lethal airway obstruction.

042 Sudden Infant Death After Being Found Awake

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Abundant literature suggests that sudden infant death syndrome (SIDS) is usually preceded by sleep. We present 6 cases of sudden infant death known to be preceded by wakefulness. These 6 cases comprise 1.2% of 462 cases of SIDS in the San Diego SIDS/SUDC Research database. The mean age was 83.7 ± 49.8 days with the range from 29 to 147 days. The male to female ratio was 2:1. One was born prematurely, 4 at term, and the gestational age was unknown in 1. "Wheezing gasps" or "rasping respirations" were observed in 2, 2 became suddenly unresponsive, 1 moaned and became limp, and 1 became "bluish grey" and semiconscious. Only 1 of 5 with a known history was exposed to gestational tobacco smoke, and 1 was exposed postnatally to tobacco. Two were placed prone for sleep, 1 on the side, and the position was unknown in 3. Three were found prone, 2 were found supine, and the position was not described in 1. The face was to the side in 2, up in 2, down in 1, and unknown in 1. The head was not covered in 1, and unknown in 5. Two cases were bedsharing, 3 were alone, and the status was unknown in 1. The death of one case was delayed 6 hours after successful resuscitation; CPR was attempted but unsuccessful in 5. Oronasal blood was not described in 5 and was unknown in 1. Conjunctival petechiae were not identified in 5 and was unknown in 1. Intrathoracic petechiae were present in 5, and unknown in 1. Metabolic screening studies were negative in 1, and not performed in 5. The demographic and pathologic profiles of these cases are similar to those of SIDS in general. Comprehensive scene investigation and postmortem examination, including ancillary studies, are critical to explaining these deaths.

043 Sudden Unexplained Death in Childhood (SUDC). A Report of 34 Cases.

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Systematic studies of sudden unexplained death in children are available given its rarity. We report 34 children in whom the cause of death remained unexplained after review of the circumstances of death and postmortem examinations. The ages of the children ranged from 384 days to 15 years with 30 cases between 12 and 36 months. There were 21 males and 13 females. 26 were white, 4 were Asian/Pacific Islander, 2 were Hispanic, and 2 were black. A family history of SUDC was absent in 24 in which information was available. A history of SIDS occurred in 3 families, was absent in 21, and was unknown in 10. A history of epilepsy was present in 2 families, was absent in 21 and was unknown in 11. A family history of seizures was present in 7, was absent in 11, and was unknown in 16. A history of seizures affecting the SUDC child was present in 10 (8 of these were febrile), absent in 22 and unknown in 2. 28 SUDC children were thought to be sleeping before becoming unresponsive and 6 were awake. 3 were bedsharing, 25 were not and it was unknown in 6. The body was found prone in 24, supine in 2, side in 2, and unknown in 6. The face position was found down in 9, up in 1, to the side in 12, and unknown in 12. Postmortem gross examinations and toxicology analyses were performed in all cases. Postmortem cultures were done in 23 and not done in 11. Metabolic screening was performed in 20, not done in 14. Vitreous electrolyte analyses were done in 15 and not done in 19. Postmortem radiographs were done in 17 and not done in 17. Intrathoracic petechiae were present in 14 and absent in 20. SUDC is rare, shares features with typical SIDS, and is dependent upon comprehensive review of the medical histories, circumstances of death, and postmortem examination to make the diagnosis.

044 Changes of concepts and diagnostic shifts over time

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Literature review shows that over the past decennia different 'causes' were thought to be responsible for the sudden unexpected infant deaths. Comparison of mortality statistics in time and place gives insight in the changes of concepts and in diagnostic shifts. Davison (1945) reported one diagnostic shift. Before 1927 a coroner could act on the strength of a doctor's opinion as to the cause of death (asphyxia or suffocation) without necessarily having the confirmation of a post-mortem examination. The medical opinion as to the cause of death, based on circumstantial evidence, history and external examination only, was usually accepted. Since the Coroner's (Amendment) Act in 1926 the coroner was given authority to order post-mortem examination without necessarily committing the case to an inquest if death was found to be due by natural causes. Since that day every suspected suffocation in Birmingham was subject to post-mortem examination. This resulted in a remarkable change of classification. In 1918-1924 in a population of 550.000 130 cases were classified as asphyxia while in bed with parents or others. From 1938-1944 in a population of 1.000.000 only 18 cases had been classified as asphyxia while 229 cases were classified as respiratory infection (77 with otitis media). Comparison of mortality tables for the year 1944, for infants under 1 year in Texas, New York and California show a great variability in causes, which could only be explained by assuming that the criteria for these diagnoses were most indefinite.

What was learned from the past, present and what does it mean for the future?

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045 Respiratory malacia: possible cause of sudden death in infancy and early childhood?

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Background: The literature on the role of laryngo-tracheo-bronchomalacia (LTBM) in sudden death in infancy is very sparse. Usually it is not considered a sufficient cause of death. The condition causes functional weakness of the laryngeal and/or tracheobronchial tree due to congenital or acquired abnormalities of airway cartilage (malakia=greek for softness). Anatomically it is separated into laryngo-, tracheo- or bronchomalacia, but a combination of all three is frequent. The etiology of the congenital variant is not well understood, but it may appear in combination with other malformations. The acquired may be caused by localized pressure by vessels, preventing normal growth. Morphologically the cartilage rings are shorter and softer than normal, causing collapse, especially during forced expirations. Symptoms include wheezing, barking cough, frequent respiratory infections and cyanotic episodes. Because of the symptomatic similarities to asthma, it is often misdiagnosed. Treatment might be conservative or include surgery, depending on etiology and severity. Case Report: A 15-month-old boy woke in the middle of the night with wheezing and respiratory distress and became lifeless within minutes. Resuscitation was undertaken with temporary response, but he died the following day. At the age of four weeks congenital laryngo-tracheo-bronchomalacia had been diagnosed and treated with bronchodilators.

Autopsy revealed narrowing of the lower trachea and right bronchus, with aspiration of gastric contents. Histology showed immature cartilage in the airways with an abnormal structure, diagnosed as LTBM. Neuropathological examination revealed changes of hypoxic-ischemic encephalopathy (HIE). The cause of death was determined; HIE caused by upper airway obstruction due to collapse of the trachea and bronchus.

Discussion: The airway malacias might be an unrecognised cause of sudden death in infancy and early childhood, and should be considered, especially in cases where there is a history of respiratory distress. LTBM often causes symptoms within the first months of life, and may be subsequently misdiagnosed as asthma, thus being underdiagnosed by clinicians. There are a number of associations with other malformations such as esophageal atresia, certain chromosomal defects and vascular malformations that should alert pathologists to the possibility of occult upper airway abnormalities, when found at autopsy.

052 Increased Pulmonary MMP's In Hypoxic Piglets During Resuscitation with 100% O_2

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BACKGROUND: Newborn infants resuscitated with ambient air seem to recover more quickly compared to 100% O_2. Matrix Metalloproteinase's (MMPs) play a role in extracellular matrix remodelling in lung ischemia-reperfusion.
OBJECTIVE: To assess MMP and antioxidant capacity in piglets after global hypoxia and subsequent resuscitation with ambient air or 100% O_2. MATERIAL AND METHODS: Piglets (12-36h of age) were resuscitated for 30min by ventilation with 21% or 100% O_2at different $PaCO_2$-levels after a hypoxic insult, and thereafter observed for 150 min. In pulmonary tissue extracts, MMPs were analysed by gelatine zymography and broad matrix degrading capacity (total MMP). Total endogenous antioxidant capacity in pulmonary tissue extracts was measured by the oxygen radical absorbance capacity (ORAC) assay. RESULTS: Total matrix metalloproteinase (MMP) activity was significantly increased in resuscitated than in the control group ($p = 0.017$), and the MMP-2 expression was significantly increased in lung tissue from all resuscitated animals as compared to baseline assessed by zymography (MMP-2: $p=0.001$). In addition, MMP-2 levels were significantly higher in piglets resuscitated with 100% O_2 (MMP-2: $p=0.014$) compared to piglets resuscitated with ambient air. mRNA expression of MMP-2 were higher in the resuscitated compared to baseline, $p = 0.001$. CONCLUSION: MMP-2 is highly increased in the lungs after hypoxia-reoxygenation injury. There is a significantly higher MMP expression when resuscitated with 100% O_2 compared to ambient air. Ambient air is less damaging to the lung tissue. These enzymes are presumably activated by oxidative stress and most pronounced activation is found in piglets resuscitated with 100% O_2.

A	**Antibody**	A protein produced in the blood which fights diseases by attacking and killing harmful bacteria or viruses
	Apnea (central)	Cessation of breathing movement
	Apnea (obstructive)	Continuation of breathing movement despite obstruction of the airways
	Arousal response	Awakening in reaction to something
B	**Bacteria**	A type of very small organism that lives in air, earth, water, plants, animals, and humans, often one which causes a disease:
	Bradycardia	A slow down of the heart beat
	Brainstem	Part of the brain at the base of the skull, which contains many control systems for functions such as breathing and heart beat.
C	**Carbon dioxide, CO_2**	The gas formed when carbon is burned. CO_2 is therefore a product of normal metabolism; it is eliminated by the lungs during breathing
	Cathecolamines	A hormone produced by the body; for instance, adrenaline is a cathecholamine produced when you are frightened, angry or excited, which makes the heart beat faster and prepares the body to react to danger
	Conduction system	System that allows electricity (or heat) to go through; applied to the heart it represents the system that makes all parts of the heart to beat.
	Cytokines	A type of proteins secreted by white blood cells during inflammation; they act as mediators of information between cells.
D	**Diagnosis**	A judgment about what a particular illness or problem is, made after making an examination
	DNA	Deoxyribonucleic acid: the chemical at the centre of the cells of living things, which controls the structure and purpose of each cell and carries the genetic information during reproduction
	Drosophila	A fruit fly extensively used in genetic studies
E	**Endotoxin**	A toxin produced in the blood stream or tissues by bacteria
	Epidemiology	The study of populations in order to determine the frequency and distribution of disease and measure risks. In the context of SIDS, epidemiology often means studying the circumstances of death in babies who die suddenly and unexpectedly, studying their families and environmental factors and comparing these with babies who survive
	Etiology	The scientific study of the cause of diseases
G	**Gene**	A part of the DNA in a cell which contains information in a special pattern received by each human, animal or plant from its parents, and which controls its physical development, behaviour, etc:
	Genetics	The study of how, in all living things, the characteristics and qualities of parents are given to their children by their genes. In the context of SIDS, genetic studies can help to answer questions such as why do some babies resist infection, which overcomes other babies?
	Gestation	The period of the development of a child or young animal while it is still inside its mother's body,
	Gestational age	The duration of the period of development at birth. Normal is 40 weeks (9 months). A gestational age of 36 weeks means that the baby was born 4 weeks early.

H	**Hemoglobin**	A substance in red blood cells which combines with and carries oxygen around the body, and gives blood its red colour
	Histology	The scientific study of the structure of tissue from plants, animals, and humans.
	Hypothesis	An idea or explanation for something that is based on known facts but has not yet been proved To hypothesize: to give a possible but not yet proved explanation for something
	Hypoxemia	Low levels of oxygen in the blood
	Hypoxia	Low levels of oxygen in tissues
I	**Immune system**	The system that protects against a particular disease by particular substances in the blood including the Immunoglogulins
	Immunoglobulin	Substances in the blood that are produced by the body to protect against disease
	Incidence	An event or the rate at which something happens
	Inflammation	A red, painful and often swollen area in or on a part of your body
	Interleukin	Naturally occurring compounds that are members of the family of cytokines. They are a signal substance between cells of the body.
L	**Larynx**	The hollow organ in the neck at the entrance of the airways
	Lymphocytes	A type of white blood cells involved in the defense system of the organism to fight infection.
M	**Metabolic disease**	Disease that involves complex biochemical processes. In the context of sudden unexpected death, such diseases have been identified in a small percentage of cases.
	Metabolic rate	The speed at which the bodies used energy
	Metabolism	All the chemical processes in your body, especially those that cause food to be used for energy and growth.
	Microbiology	The study of very small living things, such as bacteria
	Microorganism	A living thing which on its own is too small to be seen without a microscope; examples are bacteria and viruses
	Morphology	The scientific study of the structure and form of humans, animals and plants.
	Mucosal	That which concerns the mucous membrane
	Mucous membrane	The thin skin that covers the inner surface of parts of the body such as the nose and mouth and produces mucus to protect them
	Multi-disciplinary research	Research of scientists and experts from different fields working together to address complex problems such as sudden infant deaths.
N	**Neuron**	A nerve cell that carries information between the brain and other parts of the body
	Neurotransmitters	A group of substances that are released on excitation of the cells in the brain (the neurons) and either excite or inhibit target cells.
O	**Oesophagus**	The tube which takes food to the stomach
P	**Pathologist**	An expert in the study of diseases, especially someone who examines a person's body to discover if there were abnormalities and how they died
	Pathology	The scientific study of disease
	Pathway	A set of connected chemical reactions in biology
	Perinatal	Perinatal refers to the period immediately before and after birth.
	Petechiae	Small red spots on the skin or on the surface of organs within the chest or

		abdominal cavity. The red spots are produced by blood outside vessels.
	Pharynx	The soft part at the top of the throat which connects the mouth and nose to the oesophagus (the tube which takes food to the stomach) and the larynx (the hollow organ in the neck, at the entrance of the airways).
	Physiology	The scientific study of the way in which the bodies of humans, animals and plants work. A physiologist is a scientist who studies physiology. Physiology includes the study of body functions such as heart beat and breathing and their development.
	Plasticity	Soft enough to be changed into a new shape
	Postneonatal	Postneonatal refers to the period comprised between 28 and 365 days of life
	Prone	Refers to sleeping on stomach
	Protein	One of the many substances found in food such as meat, cheese, fish or eggs, that is necessary for the body to grow and be strong
	Pyrogen	Substance or agent that produces fever
R	**Rebreathing**	Rebreathing refers to breathing in the air we exhale. Babies can experience excessive levels of the gas CO_2 when they rebreathe air trapped in soft bedding and pillows, for instance.
S	**Serotonin**	A neurotransmitter and hormone with multiple activities. The serotoninergic system is known to modulate mood, emotion, sleep and appetite and thus is implicated in the control of numerous behavioural and physiological functions.
	Statistics	The science of using information discovered from studying numbers. Statistics are information based on a study of the number of times something happens or is present, or other numerical facts. Vital statistics: a group of official facts, which show such things as the number of births, deaths and marriages in a particular country.
	Streptococcus and Staphylococcus	Bacteria, many types of which cause disease
	Supine	Refers to sleeping on the back
	Synergy	The combined power of a group of things when they are working together which is greater than the total power achieved by each working separately
T	**Thymus**	The thymus produces lymphocytes and is therefore involved in the immune function. The thymus is in the chest cavity, just in front of the heart.
	Toxicology	The scientific study of the characteristics and effects of poisons. In the context of SIDS, toxicology screening is used to determine whether abnormal levels of drugs or other substances are present in the blood.
	Toxin	A poisonous substance, especially one which is produced by bacteria and which causes disease
V	**Vital statistics**	A group of official facts which show such things as the number of births, deaths and marriages in a particular country, area, etc.

The definitions have been adapted from the Cambridge Advanced Learner's Dictionary, on-line version (http://dictionary.cambridge.org/) and from the Webster's new World Medical Dictionary, on-line version (http://www.medterms.com)

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