Congress Handbook - Programme and abstracts

WHY?

Understanding and preventing infant death and complicated grief

ESPID

10th Congress of the
European Society for the Study and
Prevention of Infant Death

Oslo, Norway
May 27th - 31st 2003

ESPID

The Norwegian SIDS Society

Contents:

On behalf of the Norwegian SIDS Society I welcome you all to Oslo and the 10th Congress of the European Society for the Study and Prevention of Infant Death. As host and organiser, we sincerely hope you will enjoy your stay and the Congress' scientific, social and cultural programme.

When the Norwegian SIDS Society decided to launch our candidacy for the 10th ESPID Congress, our main motivation was to facilitate contact between the different groups and people involved in work aimed at preventing infant death. We are all trying to solve the same puzzle. The sharing of knowledge and experiences is vital if we are to progress and reach our common goals. If all of us – researchers, health professionals and parents – join forces, our chance of solving the puzzle is more likely. International meetings are important for the much-needed exchange of experiences and knowledge.

To host and organise a congress of this magnitude is demanding on people and resources. We are grateful to partners, financial contributors and the people who have kindly contributed to the preparations for the Congress. The Norwegian SIDS Society is proud to have a Scientific Advisory Board and we appreciate the extra work put in by its volunteer members in preparing the Congress' scientific program. Volunteer members and staff members of the Norwegian SIDS Society have also accepted an increased work load. We appreciate their efforts! We also appreciate the contributions from artists taking part in the Congress' cultural programme. Thank you!

We are also grateful for the financial support received from the Royal Ministry of Health and the Norwegian Foundation for Health and Rehabilitation. Thank you very much!

Mr Per Ditlev-Simonsen, the Mayor of Oslo, has invited us to hold the Reception and Opening Ceremony in the City Hall. We appreciate the city's kind gift.

Bjørn Magne Amundsgård
Chairman
Norwegian SIDS Society

In 1949, President Harry Truman promised the people of USA a "Fair Deal".

In some respects, the current situation of the international SIDS community resembles that of the US in 1949: just as President Roosevelt's New Deal policy had counteracted the economic depression of the 1930s, the "back to sleep" campaign launched by the SIDS community around 1990 has dramatically reduced the SIDS rates of previous decades. In a small country like Norway alone, the lives of more than a thousand babies have been saved since 1990.

However, SIDS is still one of the most important causes of death during infancy in industrialised countries; thousands of babies still die suddenly, unexpectedly and without explanation every year. These babies and their families deserve their fair deal – i.e. that researchers redouble their efforts to understand the fatal mechanisms to prevent new tragedies, and that health personnel learn more about complicated grief, to help the families recover.

During the last ESPID Congress, in Istanbul, our eyes were opened to the large number of infants who die in Eastern European countries and in the states bordering Europe. A large number of these deaths could probably be prevented by simple methods, such as better medical follow-up of pregnant women, and of neonates and infants. Families in these countries also deserve a fair deal of our attention.

Recent studies on unexplained stillbirths (intrauterin SIDS) have revealed that there are some similarities and some discrepancies with regard to real SIDS. Since unexplained stillbirths now causes more deaths in the western world than real SIDS – and because the study of the mechanisms behind unexplained stillbirths may also throw light on mechanisms involved in real SIDS, the ESPID Congress will focus unexplained stillbirths in a plenary symposium.

The fight against SIDS has been characterised by cooperation between the parties involved. Synergy between the "grief energy" of parents, the alertness of medical personnel and the passionate, hard work of researchers formed the basis for the great breakthroughs of the 1990s. Accordingly, the ESPID Congress in Oslo aims to fulfil the desires of all groups of participants, offering a fair deal of the overall programme to parents, researchers and health personnel.

Even more importantly, the Congress aims at providing venues for the discussion of common issues such as:

- The need for thorough autopsies to establish the correct diagnosis and to push research forward
- How to approach the tendency to use unsatisfactory diagnoses such as "unascertained" instead of SIDS
- How the new situation, with relatively low SIDS rates, may influence grief reactions in families, as well as the attitudes of health personnel, police authorities and society at large.

The plenary sessions will focus on infant mortality, complicated grief, updates on epidemiology, mechanisms of death and ethical aspects of autopsy.

During the free paper sessions, poster walks and workshops, the organisers aim to provide opportunities to penetrate deeper into issues such as grief reactions in families, neurological and immunological death mechanisms, the handling of organs during autopsy, and new trends in epidemiology.

It is our aim that all participants will experience the Congress as an exciting and intriguing experience, and that all parties will come away feeling that they have been given a fair deal.

May and June is the finest time of the year in the Oslo region. The days are long and the nights are short.

It is springtime, and nature explodes. We wish that participants from all over the world enjoy much more than a fair deal when they experience the beauty and thrill of the Norwegian spring.

Torleiv Ole Rognum
Executive Chairman of ESPID

Welcome to Oslo

It is a great pleasure to welcome delegates to the 10th congress of the European Society for the Study and Prevention of Infant Death.
Welcome to the City of Oslo.

I do appreciate that you have chosen Oslo to host your international congress. A close working relationship between parents, health professionals and researchers are most important when it comes to preventing infant death. Let me take this opportunity to thank each of you for your valuable work.

Many of you visit Oslo for the first time, and it is my hope that you will have the opportunity to get to know Oslo with all the City have to offer. An outstanding standard of living are among the qualities that we greatly value. Oslo has a great varity of cultural attractions like theatres, opera, conser-hall and many museums. The Edvard Munch Museum is especially popular and the Vigeland Scupture Park is unique.

The first settlement within today´s townsite took place in the 8th century and the very foundation of this first settlement was the exceptional advantages of Oslo´s natural harbour. During the Viking era, the little village grew rapidly. A few decades later the City of Oslo was founded.

I hope your congress will meet your expectations. Once again welcome to Oslo.

Per Ditlev-Simonsen
Mayor of Oslo

Congress Committees

Congress Steering Committee:

Jens Grøgaard: Head of National Scientific Board
Torleiv O. Rognum: President of ESPID
Trond Mathiesen: Congress Coordinator
Trine Giving Kalstad: Psychosocial Programme Coordinator
Unn Fossdal: Coordinator of Social and Cultural Committee

National Scientific Committee

Prof. Jens Grøgaard, MD
Prof. Torleiv O. Rognum, MD
Prof. Lorentz M. Irgens, MD
Prof. Rolv Skjærven, DPhil
Lauritz Stoltenberg, MD
Marianne Arnestad, MD
Dag Nordanger, MPsych
Oddbjørn Sandvik, MPsych
Kari E. Bugge, Faculty Tutor, Nursing School
Unn Fossdal, Norwegian SIDS Society
Trine Giving Kalstad, Norwegian SIDS Society
Jon Risdal, Information Consultant

International Scientific Committee:

Caroline Blackwell (AUS)
Peter Blair (UK)
Hazel Brooke (UK)
Peter Fleming (UK)
Roger Byard (Australia)
Geir Sverre Braut (Norway)
Ola Didrik Saugstad (Norway)
Igor Kelmanson (Russia)
Richard Obershaw (USA)
Atle Dyregrov (Norway)
Yildiz Perk (Turkey)
Paul Johnson (UK)
Ed Mitchell (New Zealand)
Jem Berry (UK)
Julia Kjærstad (Norway)
Hugo Lagercrantz (Sweden)
Anat Shatz (Israel)
Andre Kahn (Belgium)
Eric Mallet (France)
Henry F. Krous (USA)
Hannah Kinney (USA)
J. Frederik Frøen (Norway)
Åshild Vege (Norway)

International Abstract Reviewing Committee:

Torleiv O. Rognum (Norway)
Jens Grøgaard (Norway)
Lorentz M. Irgens (Norway)
Dag Nordanger (Norway)
Trine Giving Kalstad (Norway)
Rolv Skjærven (Norway)
Lauritz Stoltenberg (Norway)
Marianne Arnestad (Norway)
Oddbjørn Sandvik (Norway)
Kari E. Bugge (Norway)
Unn Fossdal (Norway)
Åshild Vege (Norway)
Martin Schlaud (Germany)
Monique L'Hoir (Netherlands)
Joseph Milerad (Sweden)
Christian Poets (Germany)
Hannah Kinney (USA)
Andre Kahn (Belgium)
Anat Shatz (Israel)
Hazel Brooke (UK)
Yildiz Perk (Turkey)
Karl Bentele (Germany)

Scientific Secretariat:

Congress Coordinator: Trond Mathiesen
Norwegian SIDS Society
Arbinsgt. 7, N-0253 Oslo, Norway
Tel. (+ 47) 22 54 52 00, Fax: (+ 47) 22 54 52 01
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Organizing Secretariat:

Congress Conference AS - CONGREX
P.O. Box 2694 Solli, N-0204 Oslo, Norway
Tel. + 47 22 56 19 30, fax + 47 22 56 05 41
E-mail: espid@congrex.no

Scientific Information

Course:

There will be one pre-congress course:

- SIDS – Forensic and Paediatric Pathology
  R. Byard, Australia
  H F. Krous, USA
  T.O. Rognum, Norway

The Congress and the pre-congress course have been approved as part of the curriculum for specialist training at the University of Oslo (Pathology, Paediatrics, Social Medicine and General Practice). Ask the Congress Organizer for details (espid2003@sids.no).

Presentation Information:

Oral Presentations: A data projector, slide projector and overhead projector will be provided for the lecturers in the main meeting rooms. Lecturers who need other equipment should inform the Slide Check Room/Speaker Room at least 3 hours before their presentation. Lecturers who wish to use the LCD Data Projector should prepare their slides in Windows Office 2000.

Slides: When preparing your slides, please follow the directions below:

1. Slides should not have more than seven lines each.
2. Characters should be 18-point.
3. The number of words on each line should not exceed 6.
4. Slides must be prepared horizontal.
5. Slides should not include detailed tables.
6. Slides should be numbered.
7. Put your slides in glass boxes.
8. Thin, cardboard-framed slides should not be used.
9. Number of slides should equal half of the lecture time in minutes.

Poster Presentation

There will be a poster session Friday, May 30th at 3.15 pm in the *Haakon salen* at Bristol Hotel.

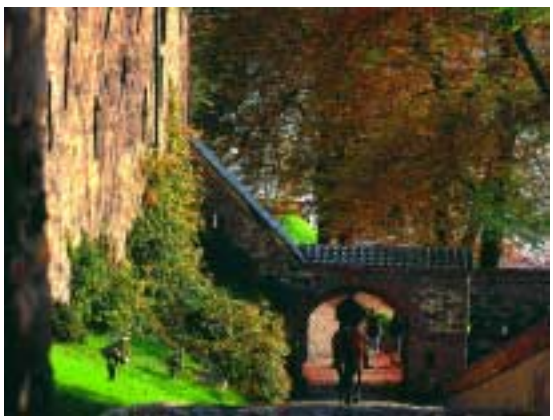
Posters should be prepared in English, measuring 98 cm in width and 136 cm in height. Text should be easily legible from a minimum distance of 1 metre. Participants with poster presentations are invited to put up their posters at noon Wednesday 28th and remove them by the end of the poster session on Friday 30th of May. The posters will hang on the mezzanine in the lobby of the Congress Centre in Bristol Hotel. The mezzanine is very close to the *Haakon salen*. The hall attendants will provide materials and help the participants hang their posters. The poster area will be part of the coffee break area of the Congress.
The posters will be presented in accordance with stand numbers that will be announced by the organizing committee. Participants with poster presentations will get a maximum of 3 minutes to introduce the poster. In this presentation you are allowed to use 2 slides or PowerPoint slides. After having introduced all the posters, there will be a poster discussion. The poster discussion will be instead of an organized poster walk.

Slide check services: All lecturers should bring their slides to the Side Check Room/Speaker Room *Sekel* at least **3 hours before their lectures.** The attendants in the slide check room will help check, prepare and send slides to the meeting room for your lecture. You can pick up your slides from the slide control room again after your lecture.

Awards At the end of the Congress, ESPID Awards will be given to a Senior and a Junior researcher.

Welcome to Oslo – a Congress venue with unique features

Oslo is the capital of Norway and the natural hub of Norway's national and international travel. The city, beautifully situated at the head of Oslo Fjord, is surrounded by forested ridges, giving Oslo qualities one normally wouldn't expect to find in a capital city. With a population of five hundred thousand, Oslo is relatively small compared to most



European capitals. It is, however, one of the largest in terms of area, covering more than 450 square kilometres, most of which is comprised of forests and arable land. The fact that only 1/3 of Oslo is urbanized makes it a very green city - also environmentally, with clean air and clean water, in the tap as well as in the fjord.
Due to the Gulf Stream bringing warm water from the Gulf of Mexico across the Atlantic Ocean and up to the coast of Norway, Oslo's climate is actually better than its northern latitude might indicate. The average daily temperature in May is +15°C (59°F).

First-time visitors are often surprised by the wide range of restaurants and entertainment the capital has to offer. Oslo has a large variety of restaurants, bistros and bars. Most are located within walking distance from Hotel Bristol – the Congress hotel. If you like shopping, you will find everything in Oslo: shopping malls, department stores, small boutiques, shopping streets with hundreds of small shops and retailers, charming antique shops, flea markets, suppliers of exclusive international brand names and local handicraft shops.

Getting around in Oslo is easy. For travel within Oslo we recommend public transportation with buses, trams and the underground.

The staff at the Registration and Information Desk will gladly answer questions about Oslo and what the city has to offer!

Congress hotel: Hotel Bristol.

Hotel Bristol was chosen for its location in the city centre and its good atmosphere. Most sights, museums, restaurants, bars and public transportation hubs are within walking distance from the hotel.

Hotel Bristol has a wide variety of amenities for its guests including a restaurant, café, bar, lounge and spa. Hotel Bristol is also one of the first hotels in Norway to offer a Wireless Local Area Network (WLAN). A WLAN card can be rented and a 24-hour subscription can be bought from the reception.

Address:
Kristian IV's gate 7, 0130 Oslo
Phone: +47 22 82 60 00
Fax: +47 22 82 60 01

"In a world of constant change, Hotel Bristol is dedicated to tradition. As soon as you enter our lobby you will experience the unique style that characterizes our hotel. An air of elegance which we have maintained since we welcomed our first guests in 1920."

Hotel Bristol
Oslo

Registration and Information

Opening hours for the Congress Registration and Information Desk at Hotel Bristol:

May 27th:	1200 – 1900
May 28th-May 30th:	0730 – 1700
May 31st:	0730 – 1400

Phone: + 47 22 82 62 00 Fax: + 47 22 82 60 04

The staff at the Registration and Information Desk can be of help to Congress participants in a wide variety of matters. In addition, the concierge and staff at Hotel Bristol are available for assistance.

Registration fees for participants (specialists/ researchers, nurses, parents and students) cover access to scientific activities, Opening Ceremony, welcome cocktail, Memorial Ceremony, daily lunch, coffee/tea, Programme & Abstract book, Congress bag, Congress badge and Attendance Certificate. Please note that the Attendance Certificate will be issued on request only.
ESPID members have been offered a discounted registration fee. Please note that the discount is offered only if the ESPID membership dues for 2003 are paid.

Tickets for the Oslo Fjord cruise and the Summerland Concert, if available, may be bought at the Registration and Information Desk. For further information see *Social and Cultural Programme*.

The registration fee for accompanying persons covers the Opening Ceremony and the Memorial Ceremony. Please note that the fee for accompanying persons does not include lunch, coffee/tea, access to Scientific Sessions, access to Exhibition Area, Closing Ceremony or any Congress documentation. Tickets for the Oslo Fjord cruise and the Summerland Concert, if available, may be bought at the Registration and Information Desk.

Congress badges

For purposes of security, all participants and accompanying persons should wear their Congress badges at the entrance to the Congress Centre, during the scientific sessions, during lunch and coffee breaks, in the Exhibition Area and during social activities.

Exhibition Area

There will be an exhibition in Hotel Bristol's Congress Centre lobby. Coffee and tea will be served in the Exhibition Area.

Poster Area

The Poster Area is on the mezzanine of Hotel Bristol's Congress Centre lobby.
Participants with poster presentations are allowed to hang their poster at noon on Wednesday May 28th. The poster session will be held on May 30th and posters must be removed after the session.

Oral presentations

The Congress will offer a combination of plenary sessions and parallel sessions. Plenary sessions will take place in *Bristol Hall*. Parallel sessions will take place in *Haakon salen*, *Maud salen* and *Olav salen*. Please see flow chart and scientific programme for details on location of the various oral presentations.

Sightseeing

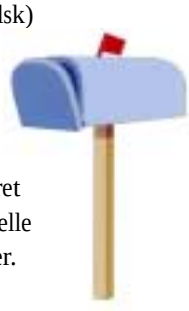
Congress participants and accompanying persons who want to go sightseeing are advised to contact the Registration and Information Desk for information. Tickets can be bought from the reception of Hotel Bristol.

ESPID General Assembly

Personal and corporate members of ESPID are invited to take part in the General Assembly on May 30th at 6 pm in *Olav salen.*

Noe du lurer på?

Ved Information and Registration Desk vil det stå en spørsmålskasse der alle kan legge spørsmål (på norsk eller engelsk) rettet til forskere på kongressen. Disse vil bli forsøkt besvart under den daglige oppsummeringen på norsk som representanter fra Landsforeningens fagråd har ansvaret for, eller de vil bli tatt med til aktuelle workshop-sesjoner for kommentarer.



Please note that the Congress Centre including lobby, mezzanine, and the Congress halls are smoke free areas.

Social and Cultural Programme

We can offer a varied social and cultural programme to the Congress delegates and accompanying persons. Informal social get-togethers are excellent for meeting friends and colleagues and for making new friends. Some parts of the Social and Cultural Programme are included in the registration fee while others require an additional payment.

Opening Ceremony

May 27 at 5 pm, Oslo Rådhus (Oslo City Hall).
The city of Oslo and Mayor Per Ditlev-Simonsen invite all Congress delegates and accompanying persons to the Opening Ceremony at Oslo City Hall. The City Hall is a five-minute walk from Hotel Bristol.

The Norwegian Minister of Health, Mr Dagfinn Høybråten, will deliver the opening address at the ceremony. Sissel Flygel who will read a poem and Åshild Skiri Refsdal who will sing are both mothers and members of the Norwegian SIDS Society. Ellen Marie Carlsen Sunde will accompany Åshild Skiri Refsdal on piano. Participants will be served canapés, fresh fruit, snacks, sparkling wine and mineral water. Participation at the Opening Ceremony is included in the registration fee.

Cruise on beautiful Oslo Fjord

May 27 at 7 pm. The boat departs from Rådhus brygge nr 3 (pier 3) near Oslo Rådhus (Oslo City Hall).
The festivities of the Opening Ceremony will continue with a cruise on Oslo Fjord. All Congress delegates and accompanying persons are invited to join us for this social event. If still available, tickets for the cruise can be purchased from the Registration and Information Desk. The price is NOK 400 and includes a buffet meal with a variety of hot and cold dishes. Drinks can be bought on the ship. Estimated duration of the cruise is 3,5 hours.

The ship offers both indoor and outdoor seating. Evenings in May can be somewhat chilly. You might want to bring an extra sweater or jacket.

Memorial

May 28 at 6.30 pm, Oslo Domkirke (Oslo Cathedral).
Quite a large group of parents who have experienced the loss of a child will attend the Congress. The Memorial Ceremony will take place in Oslo Cathedral, a stone's throw away from Hotel Bristol. Parents are invited to light a candle in memory of their child. Heine Totland and Åshild Skiri Refsdal will perform and you can expect evocative and warm songs and music. All Congress delegates and accompanying persons are invited to take part and commemorate the children.
The Memorial is open to the public.

Summerland Concert

May 29 at 8 pm, Universitetets Aula (University Hall). The doors open at 7.30 pm.
In connection with the death of a child, author Eyvind Skeie wrote Summerland – *a story about hope* in 1983. Since its release, the book has been translated into other Nordic languages as well as German and English. In 2003 the book will be available in an Arabic edition.
Eivind Skeie, in cooperation with composer Galib Mammadov and conductor/producer Per Oddvar Hildre, has recently produced a concert version of the book. Ten new songs have been written to accompany the reading of the text. The Norwegian SIDS Society is proud to announce that the premier performance of the concert version of Skeie's book will take place during the Congress. Accompanied by the renowned musicians Tord Gustavsen, Mats Eilertsen and Rob Waring, Cecilie Jørstad and Torunn Sævik will present the texts and songs.
The Prime Minister of Norway Mr Kjell Magne Bondevik and his wife Mrs Bjørg Bondevik will attend the concert as official guests.

Congress delegates and accompanying persons are invited to the Concert. If still available, tickets can be purchased from the Registration and Information Desk. Congress participants pay the discounted ticket price of NOK 100. Tickets must be bought in advance at the Registration and Information Desk in order to get the discount. The regular price is NOK 150 and tickets will also be offered to the public. The University Hall is a five-minute walk from Hotel Bristol.

Closing Ceremony

May 31 at 11.30 am, Bristol Hall.
Congress delegates are welcome to take part in the Closing Ceremony. Please note that the ESPID awards to Senior and Junior Researcher will be announced at the Closing Ceremony.

Members evening

May 30 at 7 pm.
Members of the Norwegian SIDS Society will be invited to take part in a social event for members only. Details on this event will be announced at the Congress.

"Cultural eye-openers"

Daily at 8.30 am, Bristol Hall.
Each day we will start the Scientific Session with what we call a cultural eye-opener. Please be seated by 8.30 am.

On May 28th Unn Fossdal, mother and member of the Norwegian SIDS Society, will perform together with dybwikdans – a professional ensemble consisting of singer Nils Christian Fossdal and dancers Siri Dybwik and Mari Førde. Accompaniment by Per Mathisen on bass.
On May 29th Nils Christian Fossdal will sing and on May 30th actor Yngve Marcussen will read a poem.

Bristol Lounge

To accommodate informal, social interaction among Congress participants, the Bristol Lounge will be the unofficial meeting place for evenings – a perfect place to meet old and new friends.

Congress locations are marked with red dots.

1. Hotel Bristol
2. Rainbow Hotel Stefan
3. Rainbow Hotel Munch
4. Oslo Rådhus (Oslo City Hall)
5. Cruise. Place of departure: Rådhusbrygge 3 *(pier 3)*
6. Oslo Domkirke (Oslo Cathedral)
7. Universitetets aula (University Hall)

STATOIL

Norwegian SIDS Society

The Norwegian SIDS Society is a parental organization founded in 1985. The Society has a network of trained volunteers throughout the country.

The main objectives are:
- To give care and support
- To spread preventive information
- To support SIDS research

Norwegian SIDS Society
Landsforeningen til støtte ved krybbedød
Arbinsgt. 7, N-0253 OSLO
Tel. + 47 22 54 52 00. Fax. + 47 22 54 52 01
www.sids.no - sids-norge@sids.no

ESPID (The European Society for the Study and Prevention of Infant Death) was founded in 1990 as an international body of clinicians and scientists, who are dedicated to address the problem of infant death. Main objectives are research, prevention and exchanging of information. ESPID has five permanent workgroups for clinical, pathological, epidemiological, fysiological and psychosocial approaches to studies on infant death.

ESPID Executive board:
Executive Chairman Torleiv O. Rognum, Norway
Vice Chairman Joseph Milrad, Sweden
Executive Secretary Christian Poets, Germany
Member-at-Large Jens Grøgaard, Norway
Executive Treasurer Martin Schlaud, Germany

ESPID Work groups:
Epidemiology Work Group:
Lorentz M. Irgens, Norway
Psychosocial Work Group:
Monique L'Hoir, Netherlands
Clinical Work Group:
Christian Poets, Germany
Pathology Work Group:
Åshild Vege, Norway

Sponsors

HELSEDEPARTEMENTET

Royal Ministry of Health

HELSE OG REHABILITERING

Stiftelsen for norske helse- og rehabiliteringsorganisasjoner

Norwegian Foundation for Health and Rehabilitation

Dräger MEDICAL

Partners

City of Oslo

University of Oslo

OSLO DOMKIRKE

Bogerud Blomster

Programme

Tuesday May 27

9.00
Pre-congress Course:

SIDS-Forensic & Paediatric Pathology:
R. Byard
H. F. Kraus
T. O. Rognum

From 12.00
Registration at Hotel Bristol

17.00 Opening Ceremony at Oslo City Hall

19.00 Cruise on Oslo Fjord

10th ESPID Congress, Oslo 2003

Wednesday May 28

8.30 Plenary session: Cultural opening — ***Bristol Hall***

8.45: Infant mortality
Chair: T. O. Rognum (N) & J. Grøgaard (N)
- a global perspective:
J. Zupan (WHO) — No 1

9.30 - in Eastern Europe:
I. Kelmanson (Rus) — No 2

10.00-10.30 Coffee break

10.30 - at the borders of Europe:
Y. Perk (Tur) — No 3

11.00 - Fighting infant mortality - different challenges in East and West?
O. D. Saugstad (N) — No 4

11.30: Unexplained stillbirths
Chair: D. Boyd (US) & A. Shatz (Isr)
A public health challenge?
R. Fretts (US) — No 5
The epidemiology of unexplained stillbirths:
S. Rasmussen (N) — No 6
Comparing unexplained stillbirths and SIDS:
M. Arnestad (N) — No 7
A need for definition?
J. F. Frøen (N) — No 8

Lunch 12.30 - 13.30

13.30 Workshops:

Bristol Hall	***Olav salen***
Unexplained stillbirths Chair: R. Fretts (US) & J. F. Frøen (N) Roundtable	**Sibling support** Chair: K. E. Bugge (N) & J. Kjærstad (N)

Coffee break 14.45 - 15.15

15.15 Oral session,Venue:

Bristol Hall

Autopsy
Chair: H. Brooke (UK) & M. Arnestad (N)
Ethics and Autopsy
P. J. Berry (UK)

Talking about the Autopsy:
A venue for health personnel and parents

16.30 Summary in Norwegian, *Haakon salen*
(L.Stoltenberg & K. E. Bugge)

18.30 Memorial in Oslo Cathedral

Thursday May 29

8.30 Plenary Session: Cultural opening — ***Bristol Hall***

8.45: When grief is complicated:
Chair: M. Weber (Aus) & O. Sandvik (N)
How grief affects social relationships:
R. Obershaw (US) — No 9

9.30 New ways in understanding grief:
A. Dyregrov (N) — No 10

10.15 -10.45 Coffee break

10.45 Research on parents:
K. Dyregrov (N) — No 11

11.00 - The pain of losing a child - Autonomy, Integrity, Vulnerability: Venue between professionals and parent:
G. S. Braut (N): Moderator

Venue participants:
K. Bugge (N) K. Bentele (Ger)
A. Dyregrov (N) J. Kjærstad (N)
A. Kahn (B) A. Shatz (Isr)

Lunch 12.30 - 13.30

13.30 Workshops:

Haakon salen	***Maud salen***	***Olav salen***
Changing effects of risk factors in the post-prone periode: Chair: E. Mitchell (NZ) & L. M. Irgens (N)	**Death Scene Investigation** Chair: R. Byard (Aus) & P. Fleming (UK)	**ALTE** Chair: B. Thach (US) & A. Kahn (B)

Coffee break 14.45 - 15.15

15.15 Workshops and seminar:

Haakon salen	***Maud salen***	***Olav salen***
Changing effects of risk factors in the post-prone periode Chair: E. Mitchell (NZ) & L. M. Irgens (N)	**Family support: Speaking about the unspeakable** T. Andersen (N)	**Classification & diagnosis** Chair: Stoltenburg -Didinger (Ger) & C. Rambaud (F)

17.00 Pathologists discussion of definition & diagnosis criteria

16.30 Summary in Norwegian, *Haakon salen*
(M. Arnestad, L. Stoltenberg, D. Nordanger)

20.00 Summerland Concert

10th ESPID Congress, Oslo 2003

Friday May 30

8.30 Plenary session: Cultural opening — ***Bristol Hall***

8.45: Can we explain risk factors?
Chair: M. P. L'Hoir (NL) & L. M. Irgens (N)
SIDS - Update on risk factors: E. Mitchell (NZ) — No 12
Brain physiology and risk factors:
H. Lagercrantz (S) — No 13

9.45: Symposium
From risk factors to death mechanisms
Chair: M. Schlaud (Ger) & O.D. Saugstad (N)
Temperature regulation & risk factors:
P. Johnson (UK) — No 14
Cardio-respiratory functions and risk factors:
C. Poets (Ger) — No 15

10.15 -10.45 Coffee break

10.45: Symposium cont.
Immunology, CNS and risk factors:
Å. Vege (N) — No 16
Microbiology and risk factors:
C. Blackwell (Aus) — No 17
Serotonergic receptors and SIDS?
T. Sawaguchi (Jap) — No 18
Do cytokines modulate respiration?
J. F. Frøen (N) — No 19
Functional morphology and risk factors:
H. F. Krous (US) — No 20

Plenary discussion

Lunch 12.30 - 13.30

13.30 Workshops:

Haakon salen	***Maud salen***	***Olav salen***
SIDS around the world: Chair: R. Byard (Aus) & A. Jenik (Arg)	**Psycho-social perspectives on bereavement:** Chair: D. Nordanger (N) & K. Dyregrov (N)	**Neurobiological mechanisms** Chair: T. Sawaguchi (Jap) & B. Thach (US)

Coffee break 14.45 - 15.15

15.15 Workshops/oral/poster:

Haakon salen	***Maud salen***	***Olav salen***
Poster presentations Chair: A. Shatz (Isr) & J. Grøgaard (N)	**Free papers** Chair: C. Poets (Ger) & S. L. Tonkin (NZ)	**Immunological mechanisms** Chair: C. Blackwell (Aus) & Å. Vege (N)

16.30 Summary in Norwegian, *Haakon salen*
(L. M. Irgens, D. Nordanger, M. Arnestad)

18.00 ESPID General Assembly, Olav salen

Saturday May 31

8.30 Plenary session — ***Bristol Hall***

Chair: A. Kahn (B) & C. Rambaud (F)

SIDS - the difficult diagnosis:
R. Byard (Aus) — No 21
P. Fleming (UK) — No 22
9.00 SIDS or Unascertained:
H. Brooke (UK) — No 23

9.20 Multi-factorial causes of death:
L. M. Irgens (N) — No 24
P. Blair (UK) — No 25

10.00-10.30 Coffee break

10.30
Chairpersons: K. Bentele (Ger) & Y. Perk (Tur)
Sudden death in toddlers:
H. F. Krous (US) — No 26

11.00 - Future challenges of fighting infant death and complicated grief:
T. O. Rognum (N)

11.30 Closing Ceremony. ESPID Awards

Lunch 12.30 - 13.30

Wednesday May 28

13.30 – 14.45 Workshop: Unexplained stillbirths — **Bristol Hall**

Chair: R. C. Fretts (US), and J. F. Frøen (N)

13.30 Unexplained fetal death in Queensland 1994-1999:
A population-based analysis using routinely collected data.
V. Flenady, R. Hockey, D. Tudehop, M. Coory, J. King Abstract 27

13.40 Cause specific perinatal mortality in Australia and New Zealand
using a new clinical classification system (ANZACPM and ANZNDC).
V. Flenady, A. Chan, R. Haslam, J. King, D. Tudehope, L. McCowan Abstract 28

13.50 A kick from within; The story of a cancelled progress in antenatal care.
J. F. Frøen Abstract 29

14.00 Maternal health in sudden intrauterine unexplained death
- Do urinary tract infection protect the fetus?
J. F. Frøen, R. A. Møyland, O. D. Saugstad, B. S. Pedersen Abstract 30

14.10 A Strategy of Antepartum Testing to Reduce the Risk of
Unexplained Stillbirth. **R. C. Fretts** Abstract 31

14.10 Roundtable
Chair: T. O. Rognum

14.45 – 15.15 Coffee break

13.30 – 14.45 Workshop: Sibling support — **Olav salen**

Chair: K. E. Bugge (N) & J. Kjærstad (N)

A bereavement support model for children,
adolescents and their families in hospitals. **K. E. Bugge** Abstract 32

14.45 – 15.15 Coffee break

15.15 Oral session, Venue: Autopsy — **Bristol Hall**

Chair: H. Brooke (UK) & M. Arnestad (N)

15.15 Ethics and Autopsy. **P. J. Berry** Abstract 33

15.45 Tissue and organ retention - parent's rights. **M. J. Weber** Abstract 34

15.55 The perinatal autopsy: Integration into a multidisciplinary
bereavement program. **V. M. Anderson, S. Ryce, J. Weitzman, B. Naso, E. Benjamin, Y. Guerin, M. Lamarque, C. Bowers** Abstract 35

16.05 Parents' views on information and routines of autopsy after loosing
a child in sudden infant death. **U. M. Heltne** Abstract 36

16.15 Pediatric autopsies – a need for information? **Å. Vege, T. G. Kalstad** Abstract 37

Thursday May 29

13.30 – 14.45 Workshop: — Haakon salen

Changing effects of risk factors in the post-prone periode

Chair: E. Mitchell (NZ) & L. M. Irgens (N)

13.30	Changing risk-patterns for Sudden Infant Death Syndrome, Norway 1967-1998. **R. Skjærven, A. K. Daltveit, L. M. Irgens**	Abstract	53
13.40	Risk factor changes in 15 years (1985-2001) in the Netherlands **M. P. L'Hoir, A. C. Engelberts, G. A. de Jonge, J. H. Ruys, B. A. Semmekrot**	Abstract	39
13.50	An analysis of SIDS risk factors by CHAID. **J. M. D. Thompson, E. A. Mitchell**	Abstract	40
14.00	Co-sleeping and sudden unexpected infant death. **H. Brooke, D. Tappin, R. Ecob**	Abstract	41
14.10	A case controlled study of SIDS in the republic of Ireland: Factors relating to the infant's sleeping environment **C. McGarvey, M. McDonnell, M. O'Regan, A. Chong, T. G. Matthews**	Abstract	42
14.20	Smoking and SIDS over 8 years: Current Irish epidemiology **M. McDonnell, C. McGarvey, M. O'Regan, M. Mehanni, K. Hamilton, A. Chong, T. G. Matthews**	Abstract	43

14.45 – 15.15 Coffee break

13.30 – 14.45 Workshop: Death scene investigation — Maud salen

Chair: R. Byard (Aus) & P. Fleming (UK)

13.30	Multi-agency protocols to coordinate a thorough investigation when a baby dies. **A. Deri-Bowen**	Abstract	45
13.40	Parents experiences related to participation in voluntary death scene investigation. **U. M. Heltne, A. Dyregrov, L. Sveum**	Abstract	46
13.50	The forensic pathologist at the death scene – experiences from the German case-control death scene investigation study on SIDS. **J. P. Sperhake, M. Dreier, A. Fieguth, D. Geissler, B. Giebe, S. Heide, K. P. Larsch, C. F. Poets, M. Schlaud, U. Schmidt, C. Weihs, W. J. Kleemann**	Abstract	47
14.00	First results from the German case-control death-scene investigation study on SIDS. **M Schlaud, M. Dreier, C. Weihs, A. Fieguth, D. Geissler, B. Giebe, S. Heide, K. P. Larsch, C. F. Poets, U. Schmidt, J. Sperhake, W. J. Kleemann.**	Abstract	48
14.10	Better investigation in cases of sudden unexpected death in infants and small children. **L. Sveum, M. Arnestad, Å Vege, T. O. Rognum**	Abstract	49
14.20	Sleep environment in sudden unexpected death in infancy and early childhood. **L. Sveum, M. Arnestad, Å. Vege, T. O. Rognum**	Abstract	50

14.45 – 15.15 Coffee break

13.30 – 14.45 Workshop: ALTE – current status — Olav salen

Chair: B. Thach (US) & A. Kahn (B)

13.30	To be or not to be after ALTE. **A. Edner, M. Wennborg, B. Alm, H. Lagercrantz**	Abstract	44

Presentations from A. Kahn, B. Thach and J. Grøgaard are expected

14.45 – 15.15 Coffee break

Thursday May 29, cont.

15.15	**Workshop:**	**Haakon salen**	
	Changing effects of risk factors in the post-prone periode		
	Chair: E. Mitchell (NZ) & L. M. Irgens (N)		
15.15	Used infant mattresses, parity and sudden infant death syndrome. **D. M. Tappin, H. Brooke, R. Ecob**	Abstract	51
15.25	Smoking during pregnancy and sudden infant death. **T. Bajanowski, J. Beike, M. Vennemann, W. J. Kleemann, J. Sperhake, C. Sauerland, W. Köpcke, B. Brinkmann**	Abstract	52
15.35	Changing prevalence of modifiable risk factors for SIDS in socioeconomically different populations in Hamburg/Germany during a 6-year period (1996-2001). **J. P. Sperhake, I. Zimmermann, W. Korte, K. Püschel, K. H. P. Bentele**	Abstract	38
15.15	**Seminar:**	**Maud salen**	
	Family support		
	Speaking about the unspeakable. **T. Andersen**	Abstract	54
	Seminar both in Norwegian and English		
15.15	**Workshop: Classification & diagnosis**	**Olav salen**	
	Chair: G. Stoltenburg-Didinger (Ger) & C. Rambaud (F)		
15.15	Application of ESPID recommendations for the classification of SID cases a ten years' experience in Styria (Austria). **R. Kerbl, H. Zotter, C. Einspieler, P. Roll, M. Ratschek, G. Köstl, P. Schober, W. Sauseng, I. Bachler, R. Kurz**	Abstract	55
15.25	Neuropathology of nonaccidental head injury in early childhood. Differential diagnosis of shaken baby syndrome versus sudden infant death. **G. Stoltenburg-Didinger, J. Wienholz**	Abstract	56
15.35	Alveolar maturation arrest in two siblings: An extreme and lethal form of pulmonary hypoplasia. **S. Arslanoglu, S. Arena, A. Liberatore, G. E. Moro**	Abstract	57
15.45	Do metabolic diseases play a role in SIDS? **Å. Vege, M. Arnestad, T. O. Rognum, A. Opstad, K. Skullerud**	Abstract	58
15.55	Post-mortem radiography after unexpected death in infancy and early childhood. **C. de Lange, Å. Vege, M. Arnestad, T. O. Rognum, G. Stake**	Abstract	59
16.05	Sudden death in children, acute aspiration of gastric contents, and resuscitation attempts **C. Rambaud, E. Briand, M. Guibert, D. Cointe, F. Capron**	Abstract	60
16.15	Detection of viral genome in myocardial samples from SIDS victims and children with unnatural deaths. **R. Dettmeyer, S. Padosch, C. Haag, A. Baasner, B. Madea**	Abstract	61

Friday May 30

13.30 – 14.45 Workshop: SIDS around the world — **Haakon salen**

Chair: T. Sawaguchi (Jap) & B. Thach (US)

13.30	Problems on sudden infant death (SID) cases in Japan. **Y. Ino, A. Kouno, M. Nakayama, R. Matoba**	Abstract	73
13.40	Problems around the world: France. **C. Rambaud**	Abstract	74
13.50	Is the SIDS approach changing in Latin American countries? **A. Jenik**	Abstract	75
14.00	SIDS – problems in Australia. **R. Byard**	Abstract	76
14.10	The continuing challenge of SIDS in the United States. **H. F. Krous**	Abstract	77
14.20	Modifiable risk factors for SID in Germany. **M. Vennemann, M. Findeisen, G. Jorch, W. Köpcke, T. Bajanowski, B. Brinkmann**	Abstract	78

14.45 – 15.15 Coffee break

13.30 – 14.45 Workshop: — **Maud salen**

Psycho-social perspectives on bereavement

Chair: D. Nordanger (N) & K. Dyregrov (N)

13.30	Mother's need for support and confirmation when loosing an infant. **U. G. A. Nyman**	Abstract	68
13.40	Grief in a cultural context: Coping strategies after sudden death in Norway versus Tigray, Ethiopia. **D. Nordanger**	Abstract	69
13.50	Micro-sociological analysis of social support following traumatic bereavement: Unhelpful and avoidant responses from the community. **K. Dyregrov**	Abstract	70
14.00	Help to grieve - a description of a parent-support-group in Sweden. **U. G. A. Nyman, E. Bergman**	Abstract	71
14.10	Training volunteers to take the FSID Helpline from home. **A. Deri-Bowen**	Abstract	72

14.45 – 15.15 Coffee break

Friday May 30, cont.

13.30 – 14.45 Workshop: Neurobiological mechanisms — **Olav salen**

Chair: T. Sawaguchi (Jap) & B. Thach (US)

13.30 Chemo receptor responses in infants with apnea of infancy.
M. Katz-Salamon, M. Wennborg Abstract 62

13.40 Arousals and cerebral blood volume in preterm infants.
H. Zotter, B. Urlesberger, W. Müller, R. Kerbl Abstract 63

13.50 Breast fed infants are more easily aroused from active sleep
R. S. C. Horne , P. M. Parslow, D. Ferens, A-M. Watts, T. M. Adamson Abstract 64

14.00 Physiological relationship between autonomic reactions and arousals in infancy. **P. Franco, J. P. Lanquart, P. Van de Borne, S. Chabanski, S. Adams, D. Verheulpen, J. Groswasser, A. Kahn** Abstract 65

14.10 Effects of sleep position and sleep state on postnatal development of heart rate responses at arousal. **R. Tuladhar, R. C. S. Horne, R. Harding, S. Cranage, T. M. Adamson** Abstract 66

14.20 Skin conductance activity in infants born by smoking and non-smoking mothers during the first 10 weeks of life. **K. G. Hernes, L. Mørkrid, A. Fremming, S. Ødegården, Ø. Martinsen, H. Storm** Abstract 67

14.45 – 15.15 Coffee break

15.15 Poster presentation and discussion — **Haakon salen**

(3 minutes presentation):
A. Shatz (Isr) & J. Grøgaard (N)

- Combined effects of prone position and tobacco exposure on arousals in infants. **P. Franco, N. Seret, J. N. Van Hees, S. Scaillet, A. Mazurkiewicz, J. Groswasser, A. Kahn** Abstract 79
- Could swaddling decrease cot death? Facts and findings on swaddling. **M. P. L'Hoir, B. E. van Sleuwen, A. C. Engelberts** Abstract 80
- A comparison of efficacy between two natural exogenous surfactant preparations in premature infants with respiratory distress syndrome **M. Yalaz, S. Arslanoglu, M. Akisu, N. Kultursay** Abstract 81
- Candida septicemia as a problem in neonatal intensive care units. **H. Cetin, M. Yalaz, N. Tansug, C. Ozturk, S. Hilmioglu, M. Akisu, A. Onag, N. Kultursay** Abstract 82
- A rare cause of neonatal pneumonia: Chryseobacterium meningosepticum. **Y. Perk, N. Yucel, M. Vural, S. Altun, B. Ilikkan, G. Aygun, B. Kocazeybek** Abstract 83
- Nonbronchoscopic bronchoalveolar lavage for diagnosing ventilator-associated pneumonia in ventilated newborns. **N. Köksal, M. Hacmustafaoglu, S. Celebi, S. Bagci, I. Ildrm** Abstract 84
- Tympan temperature loss during crying in premature infants. **V. S. Hurgoiu** Abstract 85
- Is subdural hygroma in childhood a controversial lesion? **J. B. Lundemose, M. Gregersen** Abstract 86
- Accidental suffocation from an anti-allergen quilt. **S. H. Hansen** Abstract 87
- Oral nystatin propylaxis significantly reduces candidal colonization in vlbw infants. **S. Arena, S. Arslanoglu, C. Coccia, M. Andena, G. E. Moro** Abstract 88
- Routine use of fentanyl infusions for pain and stress reduction in mechanically ventilated premature infant. **S. Bagci, N. Köksal, B. Baytan** Abstract 89

15.15	**Free papers**	**Maud salen**	
	Chair: C. Poets (Ger) & S. L. Tonkin (NZ)		
15.15	Character of bacterial colonization of newborns in a neonatal intensive care unit and its relationship with nosocomial infections. **Y. Perk, N. Yucel, B. Ilikkan, G. Aygun, M. Vural, B. Kocazeybek, M. Samasti**	Abstract	94
15.25	Sudden infant death syndrome, historical aspects of a still unsolved riddle - a vanishing epidemic? **C. V. Isaksen**	Abstract	95
15.35	Epidemiology of apparent life threatening event (ALTE) in the Tyro. **U. Kiechl-Kohlendorfer, D. Hof, U. P. Peglow, B. Traweger-Ravanelli**	Abstract	96
15.45	Subtle upper airways abnormalities in infants with apnea. **A. Shatz**	Abstract	97
15.55	The effect on premature infants restrained in car seats of maintaining a neutral upper airway position. **S. L. Tonkin, C. McIntosh, C. Dakin, W. Hadden, A. J. Gunn**	Abstract	98
16.05	Low birth weight and sleep behaviour in two-month-old infants. **E. I. Adulas, I. A. Kelmanson**	Abstract	99
16.15	SIDS and LQTS: A genetic and epidemiological analysis of 41 SIDS victims. **H. Wedekind, T. Bajanowski, T. Wülfing, P. J. Schwartz, B. Brinkmann, G. Breithardt, MD, E. Schulze-Bahr**	Abstract	100

15.15	**Workshop: Immunologcial mechanism**	**Olav salen**	
	Chair: C. Blackwell (Aus) & Å. Vege (N)		
15.15	Investigation of the il-10 gene in sudden infant death. **S. H. Opdal, A. Opstad, Å. Vege, T. O. Rognum**	Abstract	90
15.25	Apoptosis in erythroid precursor cells in umbilical cord blood (CB). **A. Rojahn, A. G. Bechensteen, W. Sollien, Å. B. Westvik, P. Kierulf, J. Kjeldsen Gragh**	Abstract	91
15.35	Urinary tract infection as a risk factor for sudden infant death syndrome. **M. McDonnell, C. McGarvey, M. O'Regan, M. Mehanni, K. Hamilton, A. Chong, T. G. Matthews**	Abstract	92
15.45	Neonatal levels of 8-isoprostane, a marker of oxidative stress, in pregnancies associated with augmented risk for SIDS. **K. Braekke, N. K. Harsem, A. C. Staff**	Abstract	93

1

Infant mortality - a global perspective

J. Zupan
World Health Organization
Department of Reproductive Health and Research

2

Highlights of infant mortality in Eastern Europe

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St. Petersburg State Paediatric Medical Academy, Russia

Infant mortality rate and its structure in the eighties and the nineties varied across time and regions in the in Central and Eastern European countries. Mean for the period value for total infant mortality was the highest in Republic of Macedonia (36 per 1000 live births), while the lowest one was in Slovenia (7 per 1000). In all the countries, there was a trend towards reduction in total infant mortality rate across the time. By the end of the nineties, the figures ranged between 5 per 1000 live births in Slovenia and 23 per 1000 live births in Romania. Certain conditions originating in the perinatal period were among the leading causes of infant mortality with the highest mean values in Russia (7.5 per 1000 live births) and Latvia (7.3 per 1000 live births) and the lowest one in Slovenia (2.5 per 1000 live births). Congenital anomalies were another principal contributors to infant mortality: highest for the period mean value was in Moldavia (5 per 1000 live births) and the lowest one in Czech Republic (1.5 per 1000 live births). Infant mortality rate of the diseases of the respiratory system markedly differed across the regions: it was relatively high in Romania and Moldavia (mean for the period values 7 per 1000 live born infants), while in Czech Republic and Slovenia these figures were close to zero. Infectious and parasitic diseases were registered with the highest mean rate of 1.5 per 1000 live births in Moldavia and with the lowest mean rate in Czech Republic (0.1 per 1000 live births). Mean infant mortality rate of the diseases of the nervous system did not markedly varied across the countries, and the figures ranged between 0.1 per 1000 live births in Slovenia and 0.5 per 1000 live births in Romania and Macedonia. Not all the countries provided reliable official figures on infant mortality rates of the sudden infant death syndrome (SIDS), but when available, they ranged between 0.1 and 0.5 per 1000 live births. Total infant mortality rate in Russian Federation was declining during last decade.

While in 1985 the rate was 20.7 per 1000 live births, in 1996 it was 17.4 per 1000. Infant mortality rate in Russian Federation varies across the country, and the lowest figures were reported in St. Petersburg where they reduced to as low as 7.0 per 1000 live births by the end of 2002. Leading causes of infant mortality do not essentially vary across the time and regions, and, according to figures of 1996, they were perinatal conditions (7.5 per 1000 live births) followed by inborn malformations (4.1 per 1000), respiratory diseases (2.2 per 1000) and infections (1.2 per 1000. No reliable statistics on the total rate of SIDS in Russian Federation is available so far, principally due to misinterpretation of this diagnosis. However, in the city of St. Petersburg where this entity is diagnosed and certified in a conventional manner, mean rate for the period 1983-2002 was 0.5 per 1000 live births. The major preventable risk factors of SIDS in St. Petersburg are maternal smoking during pregnancy, a defect in developmental stimulation, putting infant prone to sleep, lack of breast feeding, overheating.

3

Infant mortality at the borders of Europe

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Turkey is the unique bridge between the two continents, not only geographically but also culturally. A candidate for E.U. with 67.000.000 inhabitants, a relatively young republic founded in 1923. Our country reveals different properties from east to west with demographic data and health care practices and services, hence infant morbidities and mortalities. At the borders of Europe, the data of our region including some of our neighbours will be discussed briefly.

Turkey is a developing country with Gross National Income per capita 2540 $ and with the average annual rate of inflation 74 % for the year 2001. Two percent of the population have the income of less than one dollar a day.

Three percent of the governmental expenditures are allocated for health, whereas this is 10 % for education and 8 % for defence. More than half of (66 %) the population live at the urban areas, although the rate of moving rural to urban is decreasing by half during the last decade

(4.6 versus 2.4 of growth rate).

Eighty-two percent of population has the facility of using adequate water, 81 % in urban and 86 % in rural regions. Totally 90 % of the population have the chance of using adequate sanitary (97 % for urban, 70 % for rural).

Within these parameters, life expectancy is 70 years and life expectancy of females as a percentage of males is 108 years. The status of the mothers is one of the most important contributing factors for infant health. Adult literacy rate is 82 % for the females whereas this is 94 % for the males. For 2001, antenatal care coverage is 68 % and skilled attendant at delivery is 81 %.

As for 2001annual number of births is 1.424.000 and total fertility rate is 2.4. Of all births, 15 % are low birth weight infants. Infant mortality rate has improved to 0.36 % whereas it was 0.163 % in the year 1960. The leading causes of infant mortality are; acute lower respiratory diseases and diarrhea.

Breastfeeding is strongly promoted in our country during the last decade. Ministry of Health encourages the hospitals to be "Baby Friendly". There are 141 Baby Friendly Hospitals throughout the country, but according to the data of Unicef as for 2001, 7 % of infants< 6 months are exclusively breastfed. 34% of infants who are 6-9 months of age are breastfeeding with complimentary foods, whereas 21 % of children are breastfed until two years.

Immunisation is provided by the governmental health care centres as well as private sector. As for 2001, for one-year-old infants, 89% is immunised for BCG, 88% for DPT3 and polio, 90% for measles and 77 % for hepatitis3.

All of our neighbours more or less have comparable data. Infant mortality rates of Azerbaijan, Georgia, Iran, Iraq, Syrian Arap Republic are, 74, 24, 35, 107, 23 respectively whereas it is 6 for Israel and 5 for Cyprus. Israel and Cyprus have the highest gross national incomes per capita (16710 and 12370 $), whereas this is about 650- 1000 $ for Georgia, Azerbaijan and Syrian Arap Republic. Iraq has the highest infant mortality rate with a gross national income per capita being 2170$.

Fertility rate for Azerbaijan, Georgia, Iran, Iraq, Syrian Arap Republic are; 1.6, 1.4, 2.9, 4.9, 3.8 whereas these are 1.9 and 2.8 for Cyprus and Israel respectively for 2001. For Turkey, fertility rate is 2.4 with a population of 67.000.000 coming after Iran who has the population of 71.000.000.

For low birth weight rate, Iraq has the highest rate; 23 % then comes Turkey (15%), Azerbaijan (10%). The others are between 6-8 %.

Breastfeeding rates are similar for Turkey and Azerbaijan. In Iran exclusively breastfeeding is the highest rate amongst the neighbours, being 66%.

In conclusion, infant mortality rate is one of the parameters indicating the well being of the society. If a population has the higher income, it brings together well education that is parallel to educated mothers who will bring up their children in a healthy manner provided by the secure environmental conditions.

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4

Fighting infant mortality - different challenges in East and West?

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There are wide variations in neonatal mortality and morbidity throughout the world with 8-9-fold difference in death rate between the poorest and the most developed areas. Worldwide there are four major causes of newborn death: infection, birth asphyxia, prematurity and congenital malformations. Also within Europe there are wide variations in newborn mortality. In Eastern Europe with its 3 mill annual births 27000 die in the neonatal period (9/1000) compared with 17000 in Western/ Northern/Southern Europe with its 4 mill births (neonatal death 4/1000). Comparing various countries reveals, however, even bigger gaps in neonatal mortality varying between 3-4 and 25/1000.

Worldwide 16% (20 mill) newborn are born with low birth weight (< 2.5 kg), in Europe this figure is 6% (0.47 mill) varying between 4 and 8% in different countries.

Survival of infants with extremely low birth weight has improved dramatically the last decades in the most developed countries, still the mortality and morbidity are very much high for these fragile infants in those parts of Europe with more limited health care resources.

A reduction in neonatal mortality to 4/1000 across Europe would save 14,000 lives each year. Improvement in socio-economic conditions, improved organization and higher medical standard of perinatal care are key factors in order to reduce neonatal morbidity and mortality. More resources are also needed. Today life saving treatment is not given to a number of newborn infants due to its high costs. It should be a prioritised and common goal for every government of the world to guarantee easy access and free therapy to every newborn child wherever she/he is born.

5

Unexplained stillbirth

R. C. Fretts
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Introduction:

Currently fetal deaths in the United States occur 10 times more often than Sudden Infant Death Syndrome (SIDS). Many fetal deaths can be attributed to maternal disorders, such as diabetes or hypertensive disease; to fetal pathology, such as congenital anomalies or severe fetal growth restriction; to placental pathology, such as abruptio placenta; or due to complications of labor and delivery. Yet many antepartum deaths remain unexplained. Unexplained fetal deaths have been difficult to study because, by definition, an unexplained fetal death is a diagnosis of exclusion and occur in pregnancies were not considered high risk.

Scope of the problem:

Unexplained fetal death is the most frequent type of fetal demise, representing between 25 and 60 percent of all fetal deaths. It is also one of obstetrics' most distressing outcomes since preventative strategies have not been identified. Unfortunately, most national statistics do not systematically classify fetal deaths by cause. The McGill Obstetrical Neonatal Data from Montreal Canada was used to evaluate the changing patterns of specific fetal deaths from 1961-88. This study showed since the 1960's that the causes of fetal death that were reduced the most were fetal deaths attributed to isoimmunization, intrapartum asphyxia, malformations, and intrauterine growth retardation (Figure 1). From the 1960's to the 1990's there was also a significant reduction in the risk of unexplained fetal deaths from 3.8/1000 to 1.4/1000. Some of this reduction may be due to improved diagnostic techniques that lead to an increased proportion of fetal deaths having an identifiable cause, (such as screening for elevated glucose, fetal-maternal bleeding, and autoimmune disease). However, the proportion of fetal deaths that remain unexplained has remained relatively constant and estimated to represent between 25 and 35% of all stillbirths.

Risk factors for unexplained fetal deaths:

Older women have an increased in conditions that are associated with an increased the risk of fetal demise, such as hypertension, diabetes, placenta previa, abruptio placenta or multiple gestations. Nevertheless, even after controlling for these risk factors advancing maternal age remains a significant independent risk factor for fetal death.

Historically a significant proportion of the increased number of stillbirths seen in older women occurred in fetuses with lethal congenital malformations. Folic acid and prenatal screening, with the availability of abortion of affected pregnancies have reduced these fetal deaths and this rate reduction has been greater in women 35 years of age or older when compared to younger women. Older women however, had an increase in the most frequent type of demise, the unexplained fetal death.

Other risk factors for unexplained fetal death have been reported in two large cohorts from Norway and Canada. The Norwegian cohort found multiple factors associated to be associated with an increased risk of unexplained antepartum fetal death: maternal age 35 years of age or older (OR 5.1, 95% CI 1.3-19.6), smoking 10 or more cigarettes (OR 3.1, 95% 1.2-8.07), education less than 10 years of age (OR 3.8, 95% CI 1.5-9.5), and a body mass index prior to pregnancy to be greater than or equal to 25 kg/m2 (OR 2.8, 95% CI 1.5-5.2). The Canadian study for years 1978-1996 found similar results to the Norwegian data with maternal age of 40 years of age or older a risk factor (OR 3.7, 95% CI 1.3-10.5), women with less than 13 years of education were also at increased risk (OR 2.9 95% CI 1.1-5.5). Fetuses weighing between the 3rd and the 10 % experienced an increased risk (OR 2.8, 95% CI 1.5-5.2), as did fetuses that weighed greater than the 90 % (OR 2.4 95% CI 1.6-4.4). The presence of pre-pregnancy obesity was significant only when study periods were combined (OR 2.9, 95% CI 1.9-4.7).

Infection and immunologic exposure:

Romero and others have proposed an "inflammation response syndrome", where by fetal losses and preterm delivery are associated with an inflammatory process medicated by cytokine production that results in prostaglandin production and preterm delivery. One of the major anti-inflammatory cytokines is interleukin1 receptor antagonist (IL-1ra). It competes with interleukin 1 (IL1) for binding to its receptors on target cells. Romero reported that homozygosity of the IL-RA subtype2 occurred 4 times more often in unexplained stillbirths than those with a live birth (p= 0003). Romero and colleagues also found that women who had an otherwise unexplained stillbirth without any evidence of obvious infection had a higher number of "memory T cells" (CD45RO) than "naïve T cells" (CD45RA) when compared to live born controls. This finding suggests that, in spite of evidence of overt infection, these women demonstrated prior exposure to infectious antigens. While unexplained stillbirth cannot be fully explained by a single gene polymorphism, the role of the inflammatory response may be important when looking at the interaction between environment and genetic predisposition.

Timing of unexplained fetal death:

The risk of unexplained fetal deaths increases late in gestation, with two-thirds of all unexplained fetal deaths occurring after 35 weeks of gestation. A stratified analysis of the timing of unexplained stillbirth by maternal age, showed a notable increase risk in late pregnancy when compared to younger women (Figure 2). Risk factors such as advanced maternal age, pre-pregnancy obesity and black race appear to cause this risk to rise more rapidly thus making the risk for these women similar to women who have a post-dates pregnancy. The reasons for this risk increase is unknown, theories include cord compression, placental dysfunction and inflammation.

Sstrategies for prevention:

Antepartum surveillance with judicious delivery of fetuses with poor fetal testing has been shown to improve outcomes in pregnancies with growth-restricted fetuses. Antepartum testing is also widely used in patients perceived to be at increased risk for fetal death, with the utility of the testing related to the underlying risk of stillbirth. Using a decision analysis model that began at 37 weeks of gestation, we found that a strategy of antepartum testing in women with an estimated risk of unexplained stillbirth to be 3 times greater than general population required between 400-700 antepartum test, between 20 and 50 inductions and 8-10 additional c-section per fetal death averted.

Summary

- Unexplained fetal deaths are the most frequent types of fetal deaths.

- Most of unexplained fetal deaths occur after 35 weeks of gestation, with the rate of unex plained fetal death increasing in gestation.
- Risk factors for unexplained fetal death is advanced maternal age, obesity, and low socio-economic factors. Other possible risk factors are smoking, and primiparity, and fetuses that weigh less than the 10% and greater than the 90% and black race.
- There are no randomized control trials that assess the potential benefit of antepartum for the purposes of reducing fetal deaths in low risk women.
- While initiating antepartum testing earlier in gestation may reduce the number of late unex plained fetal deaths that occur, this strategy will also result in a significant increase of inductions, with a possible increased risk of C-section.

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Figure 1

Changing Pattern of Fetal Death, 1961-1988. (Reference 2)

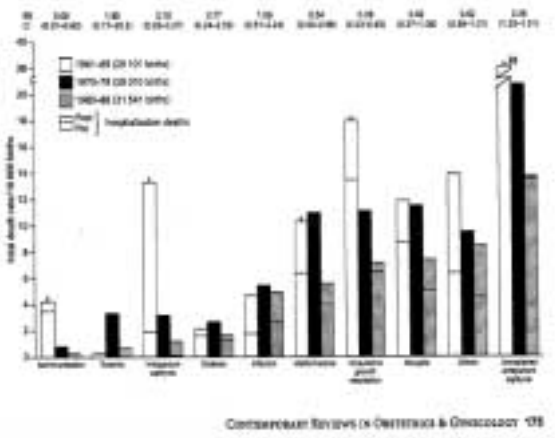
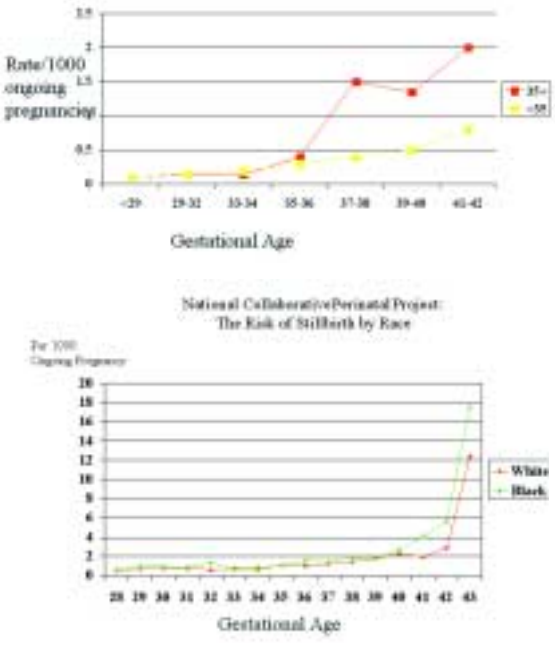


Figure 2

Unexplained Antepartum Death and Advance Maternal Age



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The epidemiology of unexplained stillbirth

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Objective:

To relate unexplained stillbirth with maternal and fetal characteristics.

Method Design:

Population based study based on records of 1,676,160 singleton births with gestational age ≥28 weeks. Unexplained stillbirth was defined as fetal death before labour without known foetal-, placental-, or maternal pathology.

Results:

Although unexplained stillbirth in general declined from 2.4 per 1000 births in 1967-76 to 1.6 in 1977-98, the proportion among all stillbirths increased from 31-32% to 43% during the same period of observation. Unexplained stillbirth occurred later in gestation than explained. From 39 weeks gestation, the risk increased progressively to 50/10,000 per undelivered pregnancies in women aged ≥35 years and <10/10,000 in women <25 years. In birth order ≥5 the risk was particularly high after 39 weeks gestation. For birth weight percentile 2.5-9.9 and ≥ 97.5, unexplained stillbirth was 4 and 3 times more likely to occur, respectively. We found an additive effect of maternal age and birth weight percentile 2.5-9.9. Women with less than 10 years education had higher risk than women with 13 years or more (OR=1.6). Weaker associations were observed with female gender, unmarried mothers, and winter season.

Conclusion:

Unexplained stillbirth occurred later in gestation than explained and was associated with high maternal age, multiparity, low education, and moderately low and high birth weight percentile. The increased risk in post-term pregnancies and the additive effect of maternal age and birth weight percentile 2.5-9.9 suggests that older women would benefit from monitoring of fetal growth, and induction of birth near or at term may be considered.

7

Comparing "unexplained stillbirths" and SIDS

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Previous studies have shown that some reported risk factors for unexplained stillbirths are the same as reported risk factors for SIDS, i.e. low socio-economic status and maternal smoking during pregnancy (1, 2). Late stillbirths are also reported to have approximately the same recurrence risk in families as SIDS (3). In 1995, Walsh and Mortimer (4) formulated a hypothesis that SIDS and unexplained stillbirths are related phenomena. They compared autopsy records (not all cases included autopsied), clinical details and epidemiological data from 149 SIDS cases with 93 unexplained stillbirths. They found similar gender and seasonal distributions, no epidemiological differences regarding maternal age, smoking or socio-economic factors, and identical post mortem features. They saw their findings to be compelling evidence that these disorders represent a continuum of a single spectrum of disease.

Several studies have looked at maternal smoking and the effect on fetal and infant death and found an increased risk of both. Tuthill et al (5) looked at the effect of smoking on the outcome of the pregnancy. They found significantly increased risk of both SIDS and unexplained stillbirths, with the odds risk of SIDS almost 3 times that for unexplained stillbirths. Guildea et al (6) studied social deprivation among families who had experienced stillbirths and infant deaths. Four groups showed a significant increase in mortality rate with increasing social deprivation: unexpected prelabor deaths with 46%, infectious deaths with 65%, specific conditions including accidental deaths with 68%, and SIDS deaths with 307%. All these studies support an epidemiological relationship between SIDS and unexplained stillbirth cases.

However, in a comparative study of SIDS and unexplained stillbirths (7), we found significant differences in the epidemiological patterns, the strong opposite effect of increasing maternal age being the most conspicuous example. Other variables, such as multiparity and male gender showed

increased risk of SIDS only, while maternal overweight/obesity showed increased risk of unexplained stillbirths only. The differences between SIDS and unexplained stillbirths for these variables were significant. For some factors, such as high fundal height measurements and proteinurea, cases of unexplained stillbirths seemed to take an intermediate position between SIDS cases and controls, with increased risk only for SIDS cases compared with controls. Maternal smoking during pregnancy showed increased risk of both SIDS and unexplained stillbirths cases, but with a significant difference between them, as the adjusted risk for SIDS was 2.9 times higher than for unexplained stillbirth. It seemed that both SIDS and unexplained stillbirths had an increased risk associated with low maternal education. For the unexplained stillbirths this factor was confirmed in the multivariate analysis. For SIDS on the other hand, low maternal education did not come out as a significant risk factor in the multivariate analysis. This must be seen in the context that low maternal education was highly positively correlated with both maternal smoking, low maternal age and also with high parity.

Even though maternal and fetal characteristics differ in SIDS and unexplained stillbirths, and these conditions obviously peak at different critical time periods (unexplained stillbirths at the end of gestation, and SIDS at 2-4 months of life), both can still be affected by related exogenous stressors during these vulnerable periods. Sub clinical infections and nicotine, both documented risk factors for SIDS and for unexplained late fetal death, are both thought to be involved in creating hypoxic brain damage in utero (8). This may suggest reduced tolerance and survival after hypoxia, both in intrauterine life resulting in unexplained stillbirth, and in infancy resulting in SIDS. Grafe and Kinney (9)state that findings of cerebral damage in SIDS victims are identical, though less severe than findings in stillborns with vascular lesions. They suggest that such findings are markers of antenatal brain injury due to hypoxia-ischemia. Also, Fifer and Myers(10)report that recent research suggest important continuities in fetal to infant neurobehaviour and indices of autonomic control. They speculate that a dysfunctional development of the autonomic nervous system could play a role in the pathogenesis of unexplained stillbirths, as recent evidence suggests it does for SIDS. Furthermore, a possible similar genetic predisposition, triggered by environmental exposures, for SIDS and unexplained stillbirths cannot be ruled out.

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8

Unexplained stillbirths – a need for definition?

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Sudden infant death syndrome has been defined as a diagnosis of exclusion for more than three decades. The success of prevention seen throughout the developed world could not have been achieved without a defined task, and common grounds for international collaboration. In the shadow of SIDS, with far less public awareness and less research effort, the obstetric and perinatal professionals have struggled with unexplained stillbirths. – One of the major causes of perinatal mortality in developed countries for decades, and today surpassing the rates of SIDS many times. Still without any broadly accepted definition. The figure below illustrates the problem: Various authors report 9 to 70% (!) of their stillbirths to be unexplained, and they can obviously not be discussing the same subject – although they seemingly do so.

The problems of definition already begin with the definition of 'stillbirth': The WHO have defined a stillbirth as: "Death prior to the complete expulsion or extraction from its mother of a product of conception, irrespective of the duration of pregnancy; the death is indicated by the fact that after such separation the fetus does not breathe or show any other evidence of life such as beating of the heart, pulsation of the umbilical cord or definite movement of voluntary muscles." Although the child is no longer a fetus after the complete separation from its mother, this definition (or equivalent) seems to be globally accepted.

However, there are no references to gestational age in this part of the definition, and there is no consensus on when a fetal death evolves from 'abortion' to 'stillbirth'. Two neighbouring countries like Norway & Sweden are examples of extremes, where the Medical Birth Registry of Norway defines stillbirths as fetal death after 16 completed weeks of gestation, while the Swedish registry only includes births after 28 completed weeks of gestation. However, the WHO have further defined the limit of birth vs. abortion as a bodyweight greater than 500g or a gestational age of 22 completed weeks or more, and recommended that also mortality rates for births after 28 completed weeks of gestation should be reported to facilitate international comparisons. As 22 weeks also represents the lower limit of extra uterine viability (as long as lung function is required for gas exchange), and 28 weeks represents the entry to the third trimester, this is a recommendation followed by the majority of authors. The most important exception is the USA where the limit is set at 20 completed weeks of gestation. If these differences are not addressed properly, it will remain a futile task to compare studies on stillbirths from e.g. Norway and Sweden, as the mechanisms causing fetal death change significantly e.g. from the 16 – 27 weeks period to the 28 – 42 weeks period.

If the problems of defining stillbirths could be overcome, we would be able to approach mechanisms. Unfortunately, most of the epidemiology studies related to stillbirths have studied stillbirth as a single entity – as if they all had the same cause of death. This does not reflect the plethora of causes of death in fetal life. Several classification systems for perinatal causes of death have been used, but none of them have been constructed specifically for stillbirths. These classifications have not been useful in exploring the epidemiology and mechanisms of unexplained stillbirths. Several classification systems have no category for these deaths at all, and have "pooled" all unclassifiable and unexplained perinatal deaths in one group, while none of the classifications that include a category for unexplained stillbirths have defined these deaths as a diagnosis of exclusion. The use of these classifications is the reason why many authors report more than 50% of stillbirths as "unexplained" – due to the lack of more suitable categories. An intriguing example of the effects of changing classification systems is the Irish study by Walsh and Mortimer in 1995, reporting that the percentage of unexplained stillbirths was approximately 10% in the 1970ies, and increased to 70% in 1993 when newer classifications were used (figure below).

The lacking definition of 'unexplained' has had the consequence that a majority of studies referring to unexplained stillbirths in reality are reporting "unexplored" cases – lacking the cornerstone of SIDS research; that unexplained is not identical to unexplored. In all studies that have reported more than 30% unexplained stillbirths, large proportions of the stillbirths where not autopsied, or the placenta was never examined. In comparison, in

studies where extensive post mortem test protocols were used; the percentage of unexplained stillbirths has been reported to be as low as approximately 10% (figure below).

In studies where the vast majority of unexplained stillbirths had been subjected to post mortem examination, and where unsuitable classification systems were avoided, unexplained stillbirths have proved to have different determinants and risk factors than other causes of fetal death – underlining the need for a definition of this important cause of death. As a proposal for a minimum that should be required to classify an antepartum death as "unexplained", with the original SIDS definition as a template, we have defined sudden intrauterine unexplained death (SIUD) as: Intrauterine death before the onset of labor of a fetus of ≥22 completed weeks of gestation or ≥ 500g body mass, which is unexpected by history, and in which a thorough autopsy of the fetus, together with gross and histological examination of the umbilical cord, placenta and membranes fails to demonstrate an adequate cause of death.

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When Grief is complicated – How grief affects social relationships

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New ways in understanding grief

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In 1989 Camille Wortman and Roxane Cohen Silver wrote a very challenging paper entitled "The myths of coping with loss" in Journal of Consulting and Clinical Psychology. Over the nearly 15 years that has passed since that article was printed, many of old notions and beliefs about grief have come into question. Not only have the long held beliefs in phases or stages in grief been questioned, but also have many other assumptions about the grieving process such as: Is distress and depression always inevitable? Is failure to experience distress indicative of maladaptive grief? And maybe most challenging: Is working through loss important? One of the most radical suggestions for both professionals and bereaved is the notion that one maybe does not have to confront one's personal feelings and reactions to the death of a loved one to adjust to the loss. Maybe the lack of distress even may be indicative of resilience, as some researchers have suggested. In the modern hectic world it is appealing to think that one may skip the "grief work" and go on enjoying life as before. What do we know in this respect? How can such notions fit with the experience of losing a child? Are there positive health effects of disclosing one's reactions? Some studies have reported benefit from writing about one's loss while others fail to find support that emotional disclosure facilitates adjustment. May it be that writing only benefits those who suffer from complicated grief?

Such issues are complex and not easily answered and answers may depend upon the type of loss experienced, the circumstances surrounding the loss, and the personal make-up of the bereaved. What is definitely clear from new research on grief is that there is a deep and long-lasting toll from losing a child suddenly and under traumatic circumstances. New research from Denmark even shows that such losses reduce the life length of bereaved parents. Regardless of the challenges to old grief theories, there continues to be a substantial part of bereaved parents who continue to experience serious problems following the death of their child, both individually and as families.

How can we effectively help parents and families following traumatic loss? Besides educating family, friends and coworkers on how to provide better social support, new knowledge point to the importance of having good crisis procedures in place to secure adequate crisis follow-up. However, since so many develop complicated forms of bereavement, family difficulties (including problems in siblings) and posttraumatic stress problems, it is equally important to secure good long term follow-up. Over the last years Holly Prigerson and Shelby Jacobs have conducted research over what is now termed "Complicated grief". This research has taught us that complicated grief differs from depression, that it has its special features that demands its own treatment formats. Awaiting recognition as a new category in DSM V, efforts already have been invested in developing specific grief treatments to more effectively help those with complicated grief reactions.

The last decade has seen a convergence of clinical experience and research evidence from the field of traumatic stress and the field of bereavement. The coming together of these fields has resulted in a better understanding of the interplay of grief and trauma, and in the development of more specific strategies to help those who suffer loss in general and those who develop various forms of complicated grief.

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Bereaved parents; experience of research participation

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Despite the ethical codes guiding bereavement research, few studies have been conducted to evaluate the perceived stress experienced by the bereaved, and to explore what methodologies are experienced to cause least distress. This article investigates how bereaved and traumatised populations experience research participation, and voice their recommendations for future research. The data is from a nationwide three-phase study with parents who lost their child by suicide, SIDS, or accident between 1.07.97-31.12.98. Whereas the first phase reported quantitative results of perceived psychosocial health and focused on offered and ideal support (N=262), the second phase investigated the same issues through in-depth interviews of a sub sample (N=69). Phase three was conducted as a short questionnaire with 64 parents, evaluating research participation in the two previous phases. The results show that 100% of the parents experienced participation as positive; very positive, and none regretted participation. They linked the positive experiences to being allowed to tell their complete story, the form of the interview, and a hope that they might help others. Apparently, processes of meaning re-construction and increased awareness of the bereavement process were facilitated by the interviews. However, _ of the interviewees reported that it was more or less painful to talk about the traumatic loss. Regression analysis showed that being a woman and high levels of psychic distress were the most important predictors of painful interview experience. In order to protect bereaved and vulnerable populations from harm, already existent ethical codes must be strictly applied, and the researchers must listen respectfully to recommendations from the bereaved themselves.

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Can we explain risk factors? Update on risk factors

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Sudden Infant Death Syndrome (SIDS or cot death) deaths dropped dramatically in the late 1980s and early 1990s in most developed countries, following the advice to parents not to put their baby to sleep on its tummy. This observation showed that SIDS, although still unexpected, was no longer non-preventable. Many other pieces of advice have been given, some with strong evidence, but others with no or limited support. This update provides a brief review as to what we currently know about modifiable SIDS risk factors.

Sleep position

Recent evidence suggests that the side sleeping position doubles the risk compared with back sleeping position, probably by infants turning to the prone position (secondary prone). Infants who usually sleep supine, but are placed prone (unaccustomed to prone) are at very high risk of SIDS. Healthy infants are not at increased risk of aspiration if placed on their back to sleep.

Smoking

Maternal smoking is very strongly associated with SIDS and this association is probably causal. Most countries have also included avoidance of tobacco smoking as part of their SIDS prevention advice. A recent meta-analysis has found that infants of mothers who smoked in pregnancy are at almost a 5-fold increased risk of SIDS compared with infants of non-smokers. The effect of environmental tobacco smoke exposure is less clear. A meta-analysis of studies where the mother did not smoke but the father did, found the risk of SIDS is increased by 1.4-fold compared with non-smoking parents. The increased risk is probably predominantly due to an in utero effect of tobacco smoke rather than postnatal environmental tobacco

smoke. Now that few infants sleep prone maternal smoking is the major risk factor for SIDS. The challenge is to develop effective strategies to reduce smoking in pregnancy, as simply telling mothers that their babies are at increased risk of SIDS is ineffective at changing behaviour.

Other SIDS prevention messages have been promoted, but with less agreement.

Breastfeeding

Most studies have shown that SIDS is lower in breast fed infants, but as breastfeeding in most developed countries is associated with socioeconomic advantage, adjustment for socioeconomic factors results in a reduced level of protection. Some have concluded there is no decreased risk from breastfeeding, whereas others have argued for a protective effect.

Pacifiers (dummies)

Unexpectedly several studies have been shown that pacifiers are associated with a reduced risk of SIDS and their use is promoted in the Netherlands in bottle fed infants. However, this benefit needs to be balanced against the possible detrimental effects, such as a reduction in breastfeeding and increased incidence of otitis media.

Bed sharing

It is now well established that there is an increased risk of SIDS with bed sharing for infants whose mothers smoked during the pregnancy. Whether or not there is an increased risk for infants whose mothers were non-smokers has not been firmly established. If there is an increased risk it is likely to be quite small (pooled odds ratio = 1.4). The controversy is that in some cultures bed sharing is an established practice. Furthermore, others have advocated bed sharing to improve breastfeeding rates.

Room sharing

However, there is some evidence suggesting SIDS risk is lower in infants that share the same bedroom as their parents, providing they are not also sharing the same bed.

Thermal stress

Thermal stress has been implicated in SIDS and has lead to the advice not to let baby get too hot. Recent work suggests that excess infant clothing or bedding increases the risk of SIDS only in prone sleeping infants. Infants are at increased risk of thermal stress when sleeping prone because the face, which is the most important skin surface for heat loss, is partially insulated by the mattress. Thermal factors are no longer important as few babies sleep prone.

Head covering

Some 15 to 20% of SIDS cases are found with their head covered by bedding. Head covering might cause SIDS by rebreathing of expired gases or by thermal stress. This has led to several suggestions as to how head covering might be avoided. These include tucking bedding in firmly, no bedding, placing infants at the foot of the cot, using the Dutch sleeping sack and avoiding the use of duvets. The evidence to support these recommendations is limited.

Immunisations

In the past there has been concern that immunisations causes SIDS, as the peak age for SIDS coincides with the age for immunisation. However, studies have shown that immunisations are not associated with an increased risk of SIDS, indeed some have shown a reduced risk of SIDS.

Conclusions and research questions

Despite SIDS mortality rate being markedly reduced, there are still groups that are at high risk, particularly those that are disadvantaged.

There continue to be many questions. The cause or causes of SIDS remain unknown, although the most likely mechanisms include airway obstruction, rebreathing of expired gases, thermal stress and an arousal defect. There is now little support for the (central) apnoea hypothesis, which was the major postulated mechanism in the 1970s and 1980s. Physiologists need to show how the establish risk factors might operate. The reason(s) for the high SIDS rate in disadvantaged and indigenous communities need to be explored.

We also need to continue to promote the established messages, which are based on strong scientific evidence, and ensure all new mothers receive this information. Innovative methods of delivering these messages and changing behaviour among disadvantaged and indigenous groups need to be found and evaluated.

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Hypoxic stress and neurotransmitter imbalance as risk factors

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Hypoxic stress causes immobility, apnea and bradycardia in the immature organism rather than the fight or flight response as typical for the adult. The classical neurotransmitters acetylcholine and catecholamines mediate many of these responses and are of crucial importance to sustain homeostasis. Disturbance of their expression and receptor action by for example nicotine can affect the hypoxic defence.

Objective:

To assess the effect of manipulation of the sympatho-adrenal and cholinergic systems on the physiological responses to hypoxia in rodents.

Method Design:

Hypoxic ventilatory response and survival have been studied in rat pups after adrenalectomy and pharmacological blockade. Arousal and ventilation has also been analysed in mutant mice lacking the ß2-subunit of the nicotinic acetylcholine receptor. Ventilation was monitored by whole-body plethysmography.

Results and Conclusions:

Adrenalectomy and adrenergic receptor blockade (phentolamine) reduced gasping time and survival in rat pups (1). A similar effect can be seen after nicotine exposure; probably due to blunting of the adrenal catecholamine release (2) Arousal from sleep was diminished and breathing drives were accentuated in nicotinic acetylcholine receptor KO-mice (3). These findings indicate that there is an intricate balance between sympathoadrenal and cholinergic systems to sustain breathing and arouse during hypoxia. This can be disturbed by exposure to nicotine and may be part of a sequence of events underlying the increased risk of SIDS infants exposed to nicotine.

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Temperature Regulation and Risk Factors

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High environmental temperatures, abnormalities or immaturity of thermoregulatory mechanisms have been considered risk factors for SIDS. While plausible, much of the evidence for this is circumstantial, lacking in data on other interrelated factors or simply not there. It was suggested from epidemiological studies in the UK that overheating was an independent risk factor although further analysis and subsequent data did not support this view. At the same time work in Tasmania showed that overheating/ raised environmental temperature was not an independent risk factor for SIDS even when infants slept prone or had infection. Nevertheless, avoidance of overheating was included in advice to prevent the risk for SIDS to new mothers in the UK.

A considerable amount of experimental work in human and other mammalian infants in which ambient temperature, hypoxia, sleeping position, and smoking were varied, has mainly been cited as supporting the view that increased environmental temperature compromises a number of 'defense' mechanisms. These include, arousal, primary or secondary to such challenges as hypoxia, baroreceptor responses and autonomic control. In particular, respiratory instability such as periodic breathing has been used as marker of respiratory risk.

It is contended that many of the studies involve sleeping practices are abnormal or unusual for the species being tested, including man. It seems appropriate that normal physiology should be established in the normal postnatal environment for the species under study, before mimicking social practices adopted in many western societies. Prone sleeping of the solitary human infant is a relatively modern development, with the former practice following the mistaken advice of pediatricians who became aware that preterm infants exhibited more effective breathing and oxygenation when nursed in the prone position. What was good practice for the

preterm infant was considered suitable for all infants, and thus practice changed in the industrialized world. Only after an increase in SIDS was observed after 1970 was this mistaken advice slowly rectified. Research then followed to show that prone sleeping did not confer the presumed benefits to the infant born at term.

Most mammalian neonates and infants have continual interaction with peers (litter mates) or parents, as is the case in most non-industrialized societies where the infant is carried by the mother or member of the family. Co-sleeping is usual but seldom in the comfort (danger) of a western bed. Newborn Chinese infants traditionally slept swaddled in a heated sand box next to the stove and then its mother in the stove-heated family bed (Kong). SIDS is rare in this social setting and even, paradoxically in the crowded westernized environment of Hong Kong. However, carriage and stimulation of the infant by family members prevails, seemingly overcoming the effects of an adverse living environment

There is good evidence that thermal control, which is heavily dependent on non-shivering thermogenesis in early infancy, takes many weeks to mature in the human infant, during which time the thermoneutral range is narrow and increases from that even in fetal life. Cardiorespiratory control has to meet the greatly increased metabolic demand of independent thermoregulation, feeding, activity and growth from that in fetal life. Recognizing the multifactorial influences on physiological function during postnatal development is a pre-requisite to identifying independent risk factors.

If clinical and physiological investigation and resultant advice were to be tailored to the cultural behaviour of the family concerned, more clarity in risk avoidance might be achieved. Too many surrogates for the real factors, some still unknown, continue to be used. For example, smoking was found to relate to abnormalities in micro diet in young socially disadvantaged mothers that independently influenced lung function in their infants. Thus specific advice to parents to moderate ambient temperature may be too simplistic. Hypothermia is probably a greater threat to infant survival worldwide than hypothermia. Attention to more holistic life-style practices may be more justified.

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Cardio-respiratory control and risk factors for SIDS: How do they fit together?

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Despite major advancements in our knowledge on prevention SIDS continues to be an enigma. Any hypothesis trying to explain these deaths must be compatible with their epidemiology; e.g., with the fact that SIDS peaks at 2-4 months of age or that cigarette smoke exposure, prone sleeping and the facedown position are major risk factors. In analyses of cardio-respiratory recordings obtained during SIDS, most deaths were preceded by a progressive slowing of the heart, while central apnoea occurred much later. These findings would be compatible with hypoxic cardiac depression potentially resulting from (face-down-) position-related airway obstruction (Ped Res 1999; 45:350). The prone position also favours upper airway collapsibility even in the absence of a facedown position (Am J Respir Crit Care Med 2002; 166:760). Both the prone position and passive smoking are also associated with a decreased propensity for arousal, which could partly explain why these infants did not free themselves from the asphyxiating condition (Ped Res 2001; 49:402). Finally although the majority of death recordings analysed so far show gasping, the latter clearly remained ineffective. This gasping failure may be related to the characteristic age distribution of SIDS: In animal studies, gaping was a strong mechanism in neonatal and adolescent animals, but not during infancy (Ped Pathol 1986; 5:325). Yet, it remains unexplained why some infants seem to arouse, and can thus free themselves, from an asphyxiating condition, whereas others apparently cannot. Until more light is shed onto this issue, all we can do to reduce these deaths further is to educate parents to prevent their infants from getting into conditions where they may need the autoresuscitative mechanisms that apparently fail during SIDS.

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Immunology, CNS and risk factors

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Since the 1950'ies, with Werne and Garrows (1,2) publication, it has been focused on the fact that many of the Sudden Infant Death Syndrome (SIDS) cases have signs of inflammation on microscopic examinations and that they have symptoms consistent with a slight infection. In 1989, Forsyth (3) demonstrated elevated levels of immunoglobulins in the lungs in SIDS victims. We have demonstrated that there is a mucosal immune stimulation in the trachea, duodenum and salivary glands (4,5).

Cytokines

In 1989, Maehlen and co-workers (6) showed that peripheral stimulation altered the immune response locally in the central nervous system (CNS) through retrograde axonal transport. The same year Guntheroth (7) proposed Interleukin-1 as a link between sleep apnoea and SIDS during respiratory infections.

On this background we started to look at interleukins as a possible contributors to SIDS. For a start we looked at the proinflammatory cytokines TNF-a, IL-1b and IL-6 in cerebrospinal fluid. There were not many SIDS cases with demonstrable TNF-a and IL-1b, and we therefore concentrated on IL-6, which was elevated in a substantial amount of the SIDS victims. Half of the SIDS victims had IL-6 levels comparable to what was seen in cases of infectious death, while the other half showed very low or not detectable levels, like the control cases of accidental death (8).

The presence of the interleukins in the cerebrospinal fluid implies that there has taken place an immunological reaction. The question is where this reaction actually has been going on. One possibility is the central nervous system itself, as it is known that a variety of cells, among them endothelial cells (9), microglia and astrocytes (10) can produce interleukins. There is, however, also possible that interleukin-6 have been produced in the periphery and then have been transported to the CNS. In 1999 we published a study were we showed that there was a relation between IL-6 cerebrospinal fluid (CSF) levels and laryngeal IgA and epithelial HLA-DR response in sudden infant death syndrome cases (11). The site of the interleukin production has not been clarified, but there is, as mentioned above, two options: It may be produced by inflammatory cells in the laryngeal mucosa and then transported to the central nervous system, or retrograde axonal signals may induce IL-6 production in the CNS.

Risk factors

The prone position could be particularly harmful in this context, as this may increase the risk of overheating. IL-6 is an endogen pyrogen and will by itself impart a raised body temperature. Sleeping prone may also change the bacterial flora in the nasopharynx. A study performed on adults has shown that nasal swabs obtained form adults lying in a prone position yielded higher bacterial counts than swabs obtained after their lying supine (12). Furthermore, this would increase the risk of toxin production from staphylococci, as proposed by Blackwell and coworkers, or the presence of endotoxin from other bacteriae (13).

In addition to the hazard just mentioned, an airway infection may also imply a laryngeal stimulation. Lindgren and Grøgaard have showed that laryngeal stimulation in respiratory syncytial virus (RSV) infected lambs gave an increased inhibition of minute ventilation and delayed recovery of regular breathing (14) and that RSV infected lambs had a significantly reinforced reflex apnoea response compared with non-infected infants (15). Other infectious agents may possibly act in the same way. Furthermore, an infection will usually give increased secretion in the airways, and sleeping in the prone position may make it more difficult to clear the airways, thus imparting an irritative effect on the laryngeal mucosa. Stoltenberg and co-workers (16) showed that laryngeal irritation mediated by insufflation of ammonia-saturated air gave apnoea, and that the duration of apnoea was prolonged and the autoresuscitation was modified after intravenous and intratechal injection of another proinflammatory cytokine; IL-1b.

Smoking is also a well-known risk factor for SIDS. Froen and co-workers have shown that nicotine interferes with normal autoresuscitation after apnoea in new-born piglets when given in doses within the range of what the child of a smoking mother could receive through tobacco smoke and breast milk. This is seriously aggravated when combined with the presence of IL-1b that is

released during infections (17). Furthermore, nicotine and endotoxin have detrimental effects in the brains of the newborn piglets during severe hypoxemia (18).

The age of the SIDS victim has changed slightly over the years. During ”the epidemic” there was an age peak between 2 and 4 months, while this is not so prominent any more. We have shown that sleeping prone and having an infection seems to be especially dangerous to the small infants between 2 and 4 months of age (19).

Conclusion: The combined effect of a slight respiratory infection and a prone sleeping position could lead to a laryngeal stimulation which in turn could induce apnoea and hypoxia. IL-6 production could give hyperthermia and add to the irregular breathing. In turn these effects could give bradycardia and hypotension and ultimately lead to death.

After all these years of research, we still do not know what actually leads to SIDS. We have not been able to demonstrate a specific agent, though in many cases an infection seems to play a role. It therefore seems reasonable to focus on the infant's inflammatory response as this may help us to understand the cause of death in at least some of the cases.

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From risk factors to death mechanisms. The role of microbiological factors

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Most of the highly significant risk factors for SIDS parallel those for serious bacterial infections in infants and young children, particularly infections of the respiratory tract: age range; exposure to cigarette smoke; minor virus infection; socio-economic background; ethnic group. These factors for SIDS could enhance susceptibility to infection by affecting 1) frequency or density of colonisation of mucosal surfaces by toxigenic species implicated in SIDS, and 2) modulation of inflammatory responses to infection or toxins.

Toxigenic bacteria and/or their toxins have been identified in SIDS infants in different countries or implicated by epidemiological studies. Cytokines produced in response to some bacterial components help to control infection in non-immune hosts; but, if these responses are not controlled, they cause tissue damage or even death. Staphylococcus aureus best fits the mathematical model proposed by the common bacterial toxin hypothesis. Pyrogenic toxins produced by these bacteria can kill healthy adults or older children, and one or more of these toxins have been identified in tissues from over half of SIDS cases, which we have investigated from five different countries.

Our research group has investigated the hypothesis that some SIDS deaths are due to uncontrolled inflammatory responses to infectious agents and/or cigarette smoke, which occur during a developmental period when infants are less able to damp down inflammation. Genetic, environmental and developmental factors associated with SIDS are discussed in relation to colonisation of infants by toxigenic bacteria, effects of cytokines on physiological responses that could lead to death and the ability to control inflammatory responses induced by the toxins. Recent findings indicate that parents of SIDS infants have stronger interleukin-b responses to staphylococcal toxins and genetic analyses indicate that anti-inflammatory responses of SIDS infants might be low. The following are areas for future investigations into the role of infection in sudden death in infancy: 1) interdisciplinary investigations of genetic and environmental factors (particularly exposure to cigarette smoke) which could contribute to uncontrolled inflammatory responses of infants to infectious agents; 2) co-operative studies with ethnic groups in which there are high SIDS mortality rates to examine risk factors in relation to bacterial colonisation and inflammatory responses; 3) thorough microbiological investigations and careful interpretation of these data in cases of sudden death in infancy; 3) wider availability of specialist techniques for detection of bacterial toxins in tissues and body fluids.

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Changes of correlation between pathological information on serotonergic receptors and physiological information on sleep apnea in SIDS victims

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Objective:

A linkage study between retrospective pathological data on serotonergic receptors and prospective physiological data on sleep apnea has been designed.

Method Design:

Among 27000 infants studied prospectively to characterize their sleep-wake behaviour, 38 infants died under 6 months of age (including 26 infant victims of SIDS, 5 with congenital cardiac abnormalities, 2 from infected pulmonary dysplasia, 2 from septic shock with multi-organ failure, 1 with a prolonged seizure, 1 from prolonged neonatal hypoxemia, 1 from meningitis and brain infarction). The frequency and duration of central and obstructive sleep apneas recorded some 3-12 weeks before the infants' death were analysed. Brainstem materials from these 38 infants were studied and immunohistochemical analyses of 5-hydroxytryptamine (5-HT) 1A receptor were evaluated quantitatively. The pathological and physiological data were linked for each infant and correlation analyses were carried out for the SIDS group and for the control group.

Results & Conclusion:

In the control group, the following 3 significant correlations were found:

(1) A significant positive correlation between the density of 5HT1A receptor in the locus cereleus and the frequency of obstructive apnea (P=0.030),

(2) a significant positive correlation between the density of 5HT1A receptor in the pontine nucleus and the duration of central apnea (P=0.046),

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Do cytokines modulate respiration?

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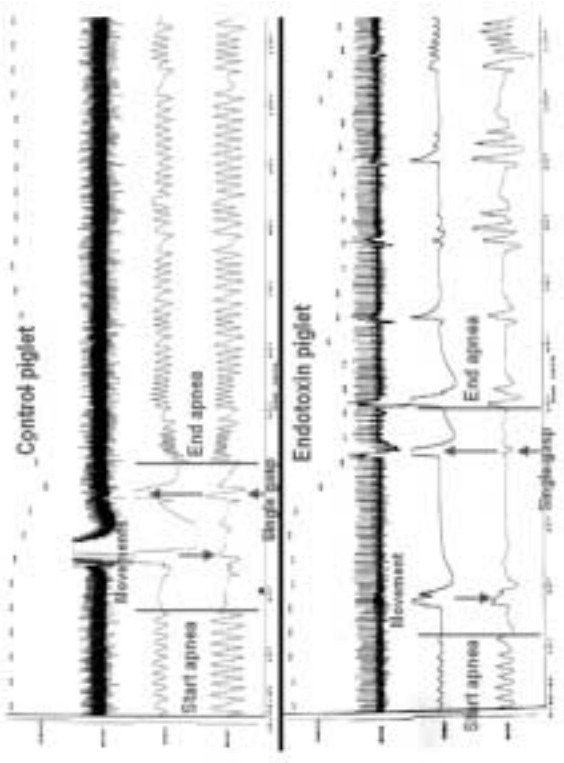
A multitude of risk factors for sudden infant death syndrome has been reported. Many of these identifiable characteristics linked to SIDS victims, can only be found in a minority of deaths, or are weak associations remote from the actual cause of death - and thus difficult to interpret. This reflects that many deaths probably are multifactorial - including a plethora of causes. However, SIDS seems to be closely associated to several epidemiologic and post mortem findings that are important in elucidating the mechanisms involved in a significant proportion of these enigmatic deaths - among these; infections and hypoxia.

Several speakers in this symposium will have presented the associations to infections seen in SIDS. Indeed, a multitude of reports have demonstrated that infants that succumbed to SIDS often had clinical signs of apparently harmless infections before death, or post mortem findings demonstrating that they had infectious agents or an activated immune system in their blood, respiratory tract and central nervous system. Many strains of virus and bacteria have been implicated or proposed as important. However, although the exact pathogen or groups of pathogens may be important to identify for future prevention, it may be less important in understanding the mechanisms involved in SIDS. Some pathogens contain toxins that are harmful to the body directly, but for most of these infectious agents that are thought to be relatively harmless, whether they are common bacteria or rare strains of virus, the clinical effects on the core functions of the human organism are caused by the same mediators: cytokines. As soon as an "intruder" is detected these small proteins are released in cascades from cells in the immune system. Depending on the intensity of the immune response, cytokines will affect important organ systems as the cardiovascular system, the hypo-thalamic-pituitary-adrenal-axis and central regulatory areas in the nervous system – affecting the infant's temperature regulation, blood pressure, heart rate, metabolism & energy production, sleep stages & arousal – and respiration.

Signs of hypoxia are also commonly reported from post mortem examinations in SIDS – both acute signs, as well as elevated hypoxanthine levels indicating more prolonged episodes of hypoxia, and findings in the brain and bone marrow indicating previous or long lasting periods of hypoxia. As respiratory failure remains the prime cause of lacking oxygen, various models have tested the effects of infections and inflammation on respiration in the infant. In particular, the effects of infectious or inflammatory challenges on laryngeal chemo reflex apnea have been studied in the newborn piglet, lamb and human. Studies by Lindgren and Grøgaard on the human infant (environmentally acquired infection) and lambs (inoculated) with respiratory syncytial virus demonstrated that infections aggravated the reflex and prolonged apnea, and it was speculated that respiratory syncytial virus infection could alter the sensitivity of the laryngeal chemo receptors locally. However, it seems that these effects are common to all infections (at any location) causing a systemic inflammatory response, as also intravenous injections of the proinflammatory cytokine IL-1_ or small doses of toxin from common intestinal bacteria (E.coli endotoxin O55:B5) had similar effects in the newborn piglet. Although apneas were prolonged, it may be more important that the cytokine response to a pathogen seriously affected the newborn's capability to restore adequate respiration after a hypoxic event, as seen in the figure below. While normal control animals had a brief apnea after laryngeal stimulation (seen as an interrupted airflow through the nostrils (NOF lower line)), rapid hyperventilation started immediately to restore oxygen saturation (SaO2 upper row of numbers). On the other hand, animals pre-treated with endotoxin had prolonged apnea, repeated

spontaneous apneas, periodic breathing, and their oxygen saturation was significantly lowered even ten minutes after the initial reflex apnea.

These findings of inflammatory responses causing a loss of autonomic respiratory control should be seen in relation to the other effects of cytokines on hyperthermia, hypotension, sleep, delayed arousal, increased vulnerability of the brain during hypoxia, and the fact that hypoxia also cause the release of cytokines, - to be seen as a participant in a vicious circle leading from apparently harmless infections to death of the predisposed infant.



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Functional morphology and SIDS risk factors

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This presentation will address the definition of SIDS and emphasizing that this diagnosis should not be made without a complete autopsy. San Diego SIDS/SUDC Research Project research that correlates risk factors, death scene findings, and post-mortem observations will be presented. The topics will include the relationship of SIDS with neck extension, found face position, pulmonary arteriolar smooth muscle, VEGF, and respiratory inflammation. Finally, the benefit of the autopsy, if done competently and thoroughly, to study fundamental questions in SIDS and to healthy grieving will be emphasized.

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Accident, abuse or SIDS – the difficult differential diagnosis

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As we are all well aware the standard definitions of SIDS do not provide diagnostic pathological features that can be used to establish that a particular infant death has been caused by this poorly understood entity. The 'diagnosis' almost arises by default. As there are no pathognomonic features that can be identified at autopsy, there have been a number of calls for the term 'SIDS' to be abandoned. In the face of overwhelming epidemiological evidence that demonstrates very characteristic features in SIDS infants, such a step would be extremely counterproductive. For example, problems in establishing the diagnosis have not stopped successful prevention campaigns. Unfortunately, however, the general pathological findings in SIDS infants remain entirely non-specific. While it can be argued that SIDS infants are more likely to have thymic petechiae or pulmonary edema than infants

dying of other causes, this is not of a degree that is of any practical consequence. Experimental data demonstrating brain stem neurotransmitter abnormalities, genetic links to cardiac conduction or cytokine disorders, and possible effects of infectious agent interaction etc. may help to clarify mechanisms of death and identify subsets of infants whose deaths can subsequently be explained, but again do not help in the day-to-day diagnosis of cases. Autopsy examinations do help to exclude natural diseases, and cases where there have been injuries from accidents or inflicted trauma. In isolation, however, deaths due to sleeping accidents or inflicted asphyxia may not be able to be differentiated from SIDS. For these reason standard protocols for post-mortem examinations, ancillary testing and death scene examination have been formulated. Their use has been shown to increase diagnoses of accidental asphyxia and drug toxicity. In addition, cases where features are of concern, but are not diagnostic of inflicted injury, have been placed in the 'undetermined' group. This leaves us with the remaining small group of infants whose deaths can still be justifiably attributed to SIDS, whose deaths require us to continue research at all levels. The challenge is to ensure that investigations into infant deaths do not remain 'scanty and inexpert' in certain areas as has recently been asserted.

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SIDS: A difficult differential diagnosis. The paediatric perspective.

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The introduction of the term Sudden Infant Death Syndrome as a label for infant deaths, unexpected by history, and not fully explained by autopsy has been of major value in establishing the epidemiological features of such deaths, and some of the factors that are associated with, and may contribute to such deaths. Some investigators have used this label as an indicator that all (or certainly most) such deaths have a common, understandable aetiology and represent the effects of a single pathophysiological process.

The reproducibility of the epidemiological risk factors between different studies in widely differing populations has led to the view of SIDS as an "epidemiological entity" (1). In the CESDI studies of Sudden Unexpected Deaths in Infancy (SUDI) however virtually all of the identifiable epidemiological features of SIDS were also present to an almost equal extent in infants who died suddenly, but for whose deaths a complete explanation was found (2).

The "triple risk" hypothesis of SIDS envisages the aetiology as being a combination of vulnerable infants going through a vulnerable phase of development, and experiencing a particular stress (a "trigger factor") at a time of heightened vulnerability (3). Such a model can incorporate both "explained" and "unexplained" deaths as being potentially the consequences of similar aetiological processes. In the former group the "trigger factor" (e.g. an overwhelming infection) would be readily identifiable, and similar to the types of external stress that may cause death at other ages. In the latter group the "trigger factor" would not be easily identifiable, though a number of potentially contributory factors might be identifiable, each of which would not, in their own right be considered a complete or sufficient "cause" of the death. For example, in an infant at a developmental stage of increased vulnerability, a minor upper respiratory viral infection, if accompanied by heavy wrapping, in a warm environment, particularly if the infant were sleeping prone might lead to a lethal disturbance of CNS thermal balance or of peripheral vasomotor tone and thus to death. Alternatively, similar circumstances might lead in another infant to trivial disturbances of temperature or blood pressure only.

The triple risk hypothesis predicts that amongst infants who die suddenly and unexpectedly there would be a potentially continuous spectrum ranging from infants for whose deaths there was a complete and sufficient explanation (e.g. the presence of an overwhelming infection with a highly pathogenic organism) to infants in whom no identifiable pathological abnormality of any type could be identified. In between would be probably the majority of infants who die suddenly and unexpectedly, for whose deaths contributory factors, but not a complete or sufficient explanation could be found.

With this perspective, the identification of the "cause" of a sudden unexpected death in infancy is less a question of seeking to identify the presence or absence of a single identifiable potentially causative factor, and more one of trying to identify all potentially contributory factors in the infant, the environment, the recent history, and the family, as well as a thorough investigation of the ways certain combinations of factors may contribute to the risk of death.

Such an investigation requires:

1. A careful history, taken by someone with expertise and experience in the care of normal and sick infants,

2. A detailed and a carefully conducted "death scene" investigation, with comparison of the environment in which the death occurred to the environments in which other, apparently healthy infants of similar social and cultural backgrounds sleep.

3. A post-mortem examination, conducted to an agreed, evidence-based protocol (4), and

4. A multiprofessional case discussion meeting in which all of the identified factors are brought together and their potential interactions reviewed by professionals with expertise in pathology, physiology, paediatrics, normal infant care, and the social and cultural norms for this society.

Figure 1.
The Avon Clinicopathological classification of Sudden Unexpected

Infant Deaths.

Classification**	**0**	**I A**	**I B**	**II A**	**II B**	**III**
Contributory or potentially "causal" Factors	Information not collected	No factors identified	Present but not likely to have contributed to ill health or to death.	Present, and may have contributed to ill health, or possibly to death	Present and certainly contributed to ill health, and probablycontributed to the death	Present, and provides a complete and sufficient cause of death
Social factors						
Non-accidental injury/ evidence of abuse or harm						
Past Medical history						
Family history						
History of final events						
Death-scene examination						
Radiology						
Toxicology						
Microbiology / Virology						
Gross pathology						
Histology						
Biochemistry						
Metabolic investigations						
Special investigations (e.g. histochemistry) Other (specify)						
Overall classification **						

** This will equal the highest individual classification listed above. NB an entry (0, I, II, or III) MUST be made on every line of the grid.
A brief free text explanation of each notable factor should also be given.

The outcome of this process should be to identify if possible a single sufficient "cause" of the death, but more often will be to identify several potentially contributory factors, none of which is sufficient in its own right to fully explain the death (4).

The Avon Clinicopathological classification of SUDI (5) is a system by which each SUDI can be classified in each of several areas, to distinguish those for which no abnormality was found, those for which an abnormality was found that might have contributed to ill health or possibly to the death. The outcome of the classification is thus a grid, in which an entry is made in each of a large number of cells, signifying whether any significant factors in that area were identified, and whether those identified were thought by the expert review committee to be of greater or lesser significance in relation to the causal process contributing to the death. Any gaps in the thorough investigation of a death will thus be identified, and the potential limitations in the accuracy of the conclusions reached will thus be immediately apparent.

The use of such a multidimensional system of classification allows investigation and the development of our understanding of how interactions between factors, themselves not sufficient to cause death, may together contribute to SUDI.

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SIDS or Unascertained?

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Over the past ten years in Scotland we have seen a dramatic move away from the use of Sudden Infant Death Syndrome (SIDS) as a certified cause of death, one which we gather is being observed in other countries. SIDS is being replaced by alternatives such as "Unascertained", "Undetermined" and "Sudden Unexpected Death in Infancy" (SUDI). Pathologists have become increasingly unhappy about using the term SIDS until all post mortem test results are available. Their reluctance is undoubted fuelled by increased scrutiny within medicine and by recent revelations in the press about deaths initially diagnosed as SIDS which were subsequently proven to be unnatural. While appreciating these pressures, this change in practice presents major problems for both researchers and the bereaved family.

Firstly, there is no agreement among pathologists as to the circumstances in which the alternative terms should be used. One pathologist will use "Unascertained" to indicate that he suspects that the parents may be guilty of abuse while another will use the term simply to indicate that there is no obvious cause for the death. Two families can therefore have the same wording on the their child's death certificate, meaning very different things. In England and Wales the percentage of sudden unexpected infant deaths certified as "Unascertained" increased from 4% in 1996 to 26% in 2001.

Secondly, in countries with a coronial system the use of the term "Unascertained" will very probably result in the case going to inquest, where details of the bereaved family's personal lives are aired in a forum open to the public and the press. The extra pressure on the already devastated family can only be imagined.

Thirdly, even when the post-mortem test results finally prove negative, the death certificate is rarely changed. In Scotland in 2002 a cause of death was subsequently found in 4 of the 40 cases originally certified as "SUDI" or "Unascertained". Of the

remaining 36 cases, only 4 were changed to SIDS. The rest all remained as "SUDI" or "Unascertained". One would like to think that the failure to change the diagnosis of these cases was the result of careful multi-disciplinary review of the pathology findings, medical history and the event scene investigation. However, since case review is in an embryonic state in Scotland the indications are that the diagnosis may have remained unchanged for a wide variety of reasons:

- Lingering concerns that the death may have been unnatural
- Incidental pathology findings
- Concerns about parental lifestyle
- Baby in bed with parents at death
- Mechanism for communicating change in diagnosis failed

These possibilities are far too varied to make amendment a secure process.

Finally, the changing practice of certification will not only make examination of trends in time within countries difficult and international comparison impossible but it will have a significant effect on research. There is a major interest in genetic studies, for example into possible vulnerability genes such as Interleukin 10, for which pure diagnostic groups are needed. The tiny number of cases now diagnosed as SIDS is going to be far too small – and how do we choose appropriate cases from the remaining muddle of terminology?

Where does this leave the bereaved family? Twenty years ago we all struggled to establish SIDS as a certifiable cause of death. We told parents that it was "a certificate of innocence", showing that the death had been fully investigated, that they had not missed signs of significant illness in their baby and that they were not to blame in any way for the death. Does "Sudden Unexpected Death in Infancy" or "Unascertained" imply the same absence of guilt? Of course not, since the term is currently used both in the case of the infant co-sleeping with a breastfeeding, non-smoking mother and the case of the infant with drug and alcohol-abusing parents whose care of the baby is, at the very least, sub-optimal. Who is going to take responsibility for explaining the implications of the current terminology to parents?

It is clearly most unlikely that pathologists, certainly within the UK, are going to revert to the use of SIDS as an initial diagnosis. They believe that this practice was flawed. However, the new system of certification is perhaps even less satisfactory. It is up to pathologists to take ownership of this problem and resolve it as a matter of some urgency. They must provide a terminology, which recognises cases, in which neither pathology findings nor death scene investigations explain the death, as well as those where genuine, objective findings exist. And they must ensure a guaranteed system for amendment of death certificates if appropriate. Otherwise, those who refer to SIDS as "a dustbin" will find that there is a new dustbin called "SUDI" or "Unascertained", within which it will be very difficult to carry out any research - particularly of a genetic nature.

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Multi-factorial Causes of Death

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Usually, a "cause of death" in medicine represents a category of cases defined according to their medical diagnoses, e.g. infections, cancer, cardiovascular diseases and even SIDS. In this paper, cause is considered as the factor(s) that caused the disease or its determinant(s). From a preventive point of view, a cause of a disease may be defined as a factor the manipulation of which will cause similar fluctuations in the occurrence of the disease.

Multi-factorial causation of disease or death implies a series of questions

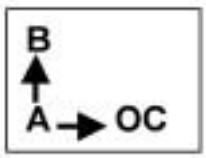
1. are the "causes" mechanistically independent implying that the relative risks pertaining to each cause may be added?

2. are the "causes" mechanistically dependent in that one determinant may reduce or increase the effects of another determinant implying that relative risk of two determinants in combination differs from the added relative risks (effect modification)? In a situation in which a disease never occurs without one of the determinants present, this determinant is considered as necessary.

3. are the “causes” mechanistically dependent in a way that the association is due to confounding?

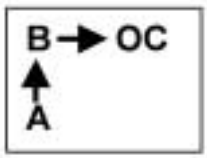
3.1 determinant A causes determinant B which causes the outcome example:

A = Social deprivation
B = Smoking
OC = SIDS



3.2 determinant A causes determinant B and the outcome example:

A = Smoking
B = Low birth weight
OC = SIDS



The technical solution is often adjustment performed in multivariate logistic regression models, which enables one to clarify to which extent the variation in the OC variable is explained by variation in one of the two independent variables possibly leaving some variation to the other variable. A one to one association between A and B would in such a model rule out the effect of the other variable even if both were causal. Consequently, such adjustment should be performed with great caution. Instead, the effect of factor A should be assessed in various levels of factor B in stratified analyses.

In the following, these principles are applied to the multifactorial web of SIDS.

- a basic pathophysiological trait

SIDS may be considered as a mechanism of death with a remarkably homogeneous clinical pattern, which suggests the use of the term syndrome. This mechanism probably represents a basic pathophysiological trait, the nature of which is unknown even if elements pertaining to respiratory, immune and cardiovascular regulatory systems have been discussed (1). Probably, the triggering of this pathophysiological trait may elicit a final common path that leads to the pattern of death known as SIDS. Even if SIDS is a rare phenomenon, this basic pathophysiological trait may be present in a large number of infants. The only difference between them may be the threshold for triggering the final common path. The rapid decline of SIDS in many countries during a period of 1-3 years by a ratio of 3-4 subsequent to the “Back to sleep” campaign strongly suggests that this trait may be present in a large fraction of seemingly normal infants.

Indicative of a basic pathophysiological trait is, in addition to the homogeneous clinical pattern, the unique age-at-death distribution with a peak between the 2nd and 3rd month of life. Probably, this distribution is related to the maturation of regulatory systems involved. Alternatively, the distribution would be the result of the timing of risk factors triggering the final common path, which seems most unlikely.

Furthermore, the increased occurrence in males (OR: 1.5) (2), which is higher than the male preponderance in most infant conditions, is probably also related to the basic pathophysiological trait. The involvement of a single X-linked gene, which has been proposed (3), is not very likely since the recurrence risk among siblings is not very high.

The trait may be considered a necessary determinant in SIDS

- prenatal factors influencing the basic pathophysiological trait

Prenatal factors may influence the threshold of the basic pathophysiological trait. Thus, the effect of the prone sleeping position (trigger) is much greater in infants with low birth weight (< 2,500g), preterm infants and term infants with intrauterine growth retardation (4). The same interaction is observed for markers of prenatal, perinatal and neonatal hypoxia such as smoking, as well as for conditions with placental dysfunction (preeclampsia, abruptio placentae). Smoking and prone sleeping combined have a relative risk of

55.3 against 16.7 for prone and non-smoking. Prenatal, perinatal or neonatal hypoxia may reduce an infant's arousal mechanisms. The potential of arousal usually varies during the day, which may be responsible for the peak of SIDS deaths early in the morning. However, in infants of smoking mothers, this peak is not observed, compatible with a reduced arousal in these infants (5). Twins have an increased risk of SIDS (RR = 2.4) (2), however, whether this is due to a prenatal placenta mediated effect or a parity-care mediated effect is not clear.

- postnatal factors triggering the basic patho-physiological trait

Postnatal factors may trigger the basic patho-physiological trait in infants with a relatively low threshold. Before the success of the "Back to sleep" campaign, prone sleeping was the triggering factor responsible for the majority of the SIDS cases calculated in terms of attributable fraction. Prone sleeping may be related to mechanisms such as re-breathing, high temperature, reduced arousal and circulatory or ventilatory obstructions. After the campaign, the relative risks of prone sleeping have increased concomittantly with the decreasing prevalence of this risk factor, a commonly observed general phenomenon. Usually, this is caused by confounding since the infants exposed to prone sleeping today are also more than previously exposed to a series of other risk factors as well. Still, the attributable fraction related to prone sleeping is now much lower than previously, due to its low prevalence.

A different type of triggering is related to care which may involve a series of established risk factors, such as the weekend effect, low maternal age, high birth order, unmarried mother and low social class/short education. The essential element in the care complex has not yet been identified, but may be related to lacking parental responses in one way or another. Thus, it is interesting to note that no increased risk for SIDS is observed for an infant born to a mother who lost her first child in the perinatal or postperinatal period; the risk is rather reduced.

The lack of specificity in SIDS (6), that "explained" deaths may occur suddenly and unexpectedly, may be due to the possibility that diseases, like infections or even birth defects, may trigger the trait or reduce its threshold.

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SIDS: Multifactorial Causes of Death, Changing the epidemiological perspective

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If the research of SIDS infants is to be judged on the number of lives saved it has been an unmitigated success. If however, it is to be judged on how well we now understand the aetiology of this syndrome after four decades of intensive research, it has so far failed.

Defined and classified in the 1960's the number of studies investigating SIDS infants now numbers more than 100 in the epidemiology field alone. Many studies have reported numerous characteristics and risk factors strongly associated with SIDS when compared with live randomised controls. It has been generally assumed that these factors are specific to SIDS to the extent that the syndrome has been described as an "epidemiological entity"[1]. However in the UK CESDI study we collected data not just on SIDS infants but all Sudden Unexpected Deaths in Infancy (SUDI) including deaths explained at post-mortem largely because of undetected infection or accidental asphyxia. Distal factors characterising SIDS infants, mothers and families, nearly all of them markers of vulnerability, were no longer unique when compared to explained SUDI infants. Of 22 factors compared all were in the same direction and only two were significantly more characteristic of SIDS infants, the unique age distribution and higher prevalence of parental smoking [2]. More recently we have re-analysed the data comparing SIDS infants with socio-economically matched live controls and shown that commonly reported maternal characteristics of SIDS infants; younger mothers, single mothers and bottle-feeding mothers were of identical prevalence amongst similarly deprived control families [3]. This is not to suggest that these features are not characteristic of SIDS but it does suggest that many of them are not unique to SIDS and therefore cannot be specifically attributed to some sort of causal pathway. Distinctive background features that may be part of some causal mechanism include infant vulnerability either from birth or the deprived circumstances in which the infant lives, an age distribution peculiar in the sense there are so few deaths in the first few weeks of life with a shifted peak at 3-4 months and excessive infant exposure to tobacco smoke.

When SIDS research first began the epidemiological net was necessarily cast wide in search of what seemed an infinite number of feasible causal hypotheses. Over the years the search has narrowed and focussed on the usual practice and specific sleeping environment of the infant. From distal factors we have moved to proximal risks and found several that are significantly predictive of SIDS. Most notable is the prone sleeping position, and although we do not yet understand the mechanism of death this has given rise to the triple risk model of SIDS causality; an underlying vulnerability, a critical development period (i.e. infancy) and an external stressful event such as not being able to move from the prone position or infant head covering [4]. This has lead to much work investigating possible causal mechanisms such as fatal rebreathing [5], hypoxia [6] and heavy wrapping in the presence of a viral infection [7] yet ignores that the majority of SIDS infants who are not found prone or with their head covered. In the CESDI study there appears to be three distinct groups of SIDS, those found prone or with their head covered or both (37%), those found co-sleeping in bed or an a sofa (24%) and those found non-prone and face uncovered in the cot (33%). The first two groups are not mutually exclusive, a further 6% of co-sleepers were found prone or covered, but this small proportion suggests the risks of prone sleeping and head covering are primarily risks associated with the cot rather than the parental bed. This also suggests that given the continued success of intervention campaigns in eliminating the use of the prone position and potential for head covering, subsequent studies should observe an increasing proportion of co-sleeping SIDS deaths. Any understanding on the aetiology of SIDS must take into account these three groups as not only is it emerging that proximal risk factors differ between these groups but distal characteristics such as age are also different. In the CESDI study the median age of SIDS infants found prone or covered was 16 weeks, for those found non-prone and uncovered, 14 weeks, and for those bed-sharing the median age was 8 weeks.

Future research will be difficult, not least because of the smaller numbers and the diagnostic shift from SIDS to unascertained, but will also be

different given the higher prevalence of deprived families and co-sleeping deaths. If we are to understand the aetiology of SIDS and reduce the number of deaths still further subsequent epidemiology studies must adapt in several ways. Firstly we must focus in even more detail on the circumstances surrounding the death. The strongest predictors of SIDS are factors specific to the last sleep not the usual practice within this environment. Habitual use of a pacifier for instance is similar between SIDS and control infants, it is the lack of a pacifier amongst habitual users for the last sleep that is important, similarly the risk associated with prone sleeping is much higher amongst those new to this position for the last sleep rather than those infants who always sleep prone. Future studies therefore proportion of co-sleeping SIDS deaths. Any understanding on the aetiology of SIDS must take into account these three groups as not only is it emerging that proximal risk factors differ between these groups but distal characteristics such as age are also different. In the CESDI study the median age of SIDS infants found prone or covered was 16 weeks, for those found non-prone and uncovered, 14 weeks, and for those bed-sharing the median age was 8 weeks.

Future research will be difficult, not least because of the smaller numbers and the diagnostic shift from SIDS to unascertained, but will also be different given the higher prevalence of deprived families and co-sleeping deaths. If we are to understand the aetiology of SIDS and reduce the number of deaths still further subsequent epidemiology studies must adapt in several ways. Firstly we must focus in even more detail on the circumstances surrounding the death. The strongest predictors of SIDS are factors specific to the last sleep not the usual practice within this environment. Habitual use of a pacifier for instance is similar between SIDS and control infants, it is the lack of a pacifier amongst habitual users for the last sleep that is important, similarly the risk associated with prone sleeping is much higher amongst those new to this position for the last sleep rather than those infants who always sleep prone. Future studies therefore need a sleep scene investigation of control infants to put findings of any detailed death scene investigation into perspective. Secondly we must utilise future controls and build on what we already know. Thus as well as an age-matched randomised control group we need a second control group not only matched by age but by sleeping environment (parental bed or cot) and socio-economic status. Thirdly, future SIDS studies should include all SUDI from the outset of the investigation and evaluate each case separately, not only will this improve diagnostic consistency but, given the relative rise in prevalence of explained SUDI deaths, yield a viable comparative group for pathological investigation. If SIDS is indeed as looks likely a multifactorial cause of death we ultimately need to define and untangle the different subgroups we collectively call SIDS. The fourth and final point therefore, is that the epidemiologist and pathologist need to work closer together, pathological findings, which may appear non-specific to SIDS as a whole may be specific to subgroups identified through the epidemiology.

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Sudden death in toddlers

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The sudden unexpected death of one's infant or child is surely the greatest tragedy afflicting the human experience. Fortunately it is rare, especially after the first birthday, but to the individual family it is an all-consuming event. The grief of survivors is often complicated by guilt and the frustration of not knowing the cause of their child's death.

The medical literature is replete with information about sudden, unexpected death in infancy and sudden infant death syndrome (SIDS). The United States National Library of Medicine (PubMed) lists more than 5,000 citations about SIDS that address its epidemiology, pathology, hypotheses of causation, and declining rates after implementation of infant care practices designed to eliminate the risk factors. In contrast, literature about the sudden, unexpected, and unexplained death of children beyond 12 months of age (SUDC) is very limited. This is perhaps not surprising given the incidence of SIDS is approximately one hundred times that of SUDC (CDC Wonder database). The Table summarizes several reports about SUDC.1-8

By definition, SIDS is not diagnosed after the first birthday, at least in the United States.9 Although the differences in developmental physiology and anatomy of young infants and teenagers are substantial, none compel distinguishing an 11 month-29 day old infant from a 12 month-1 day old toddler when considering a diagnosis of SIDS! Nevertheless, sudden unexpected death in children is rare after the age of 1 year. Ninety percent of SIDS deaths occur before 6 months of age. Epidemiologic studies have identified risk factors that place young infants at increased risk of SIDS, but their role in sudden, unexpected death beyond infancy is currently unknown.

We propose that SUDC be defined by incorporating the same elements of the SIDS definition,9 i.e., SUDC is the sudden death of a child greater than 1 year of age that remains unexplained after a thorough case investigation, including performance of a complete autopsy, examination of the death scene, and review of the clinical history. Therefore, similar to SIDS, SUDC is a diagnosis of exclusion.

A triple-risk model encompassing the developmental state of the young infant, subtle underlying pathology and external risk factors has been hypothesized to explain SIDS.10 Whether this same model can be applied to sudden death in childhood is unknown.

Therefore, we have begun a descriptive analysis of the clinical, epidemiological, death scene, and postmortem findings in children beyond the age of 1 year who died suddenly and unexpectedly. To date, 21 cases have been evaluated. Their ages ranged from 367 days to 15 years, with 18 children being between 1 and 3 years. There were 12 males and 9 females. Sixteen were white. Nineteen of 21 were thought to be sleeping when they died. Co-sleeping and sweating were identified in 2 and 1, respectively. Fourteen (78% of 18 for which data was available) were ill within 2 weeks of death with 9 (50%) experiencing fever within 48 hours of death. Poor appetite and vomiting within 48 hours of death occurred in 38% and 22%, respectively. A known past history of febrile seizures or "other" seizures occurred in 4 and 2, respectively. None had a history of apnea, but 1 had a history of breath holding spells. Family histories of febrile seizures or "other" seizures, apnea, breathing holding spells, heart "problems", or SIDS occurred in 11 cases. Nineteen died at home. Six, 1 and 1 of the cases were placed supine, side, or prone for sleep the night, respectively, during which the death occurred. Fourteen of 17 for which position was described were found prone and 6 were face down.

Autopsies and toxicology were performed in all cases; the mean and median numbers of microscopic sections were 38 and 33, respectively, with a range of 13 to 105. Postmortem radiographs were obtained in 12, being extensive in 1. Postmortem cultures in 13, metabolic screens in 12, and vitreous chemistry in 6 cases did not explain the cause of death in any of the cases. Intrathoracic petechiae were described in 8. Microscopic evidence of gastric aspiration in each of the lung sections was seen in 4 out of 20 cases (20%), but was considered to be the likely cause of death in only one.

A study diagnosis of SUDC was made in 16 cases. A cause of death was identified in the other 5. The original and study diagnoses concurred in 13 cases. Discordant and/or unrecognized diagnoses occurred in the remainder. The discordant original and study diagnoses, respectively, were: nonspecific myocarditis vs. arcuate nucleus hypoplasia, possible

acute epiglottitis vs. SUDC, possible brainstem encephalitis vs. brainstem encephalitis, possible cardiac anomalies vs. SUDC, arrhythmogenic right ventricular dysplasia vs. SUDC, acute viral infection vs. SUDC, cardiac dysrhythmia vs. SUDC, foreign body aspiration vs. probable foreign body aspiration, and SUDC vs. cerebral and cerebellar neuronal migration disorder.

The cause of death was ascribed to brainstem encephalitis or arcuate nucleus hypoplasia in 2 of 13 cases in which microscopic sections were ultimately available. It is noteworthy that microscopic sections of the brainstem were taken initially in only 12 of 20 cases; the brainstem was available in another case that had not been submitted for microscopic examination. Of the initial 12 cases with microscopic sections already available, the medulla, pons and midbrain were examined in 12, 6, and 5 cases, respectively.

We conclude that medical history review, death scene investigation and reconstruction, and thorough post-mortem examination are critical in establishing a cause of death. Toxicology and metabolic screening are necessary but typically unhelpful. On the other hand, extensive microscopic examination of the brain including many levels of the entire brainstem, offers an important opportunity to identify a cause of death in these children, even in the absence of gross abnormalities of the central nervous system.

Parents were and continue to be the driving force for legislation, parent support groups, research and conferences, including this very one! In so many ways, this SUDC story is unfolding in a fashion that parallels the early days of SIDS with parent survivors taking the lead in pursuing legislation for standardized medico legal investigations, pushing for funding agencies to make monies available for research, and establishing services for survivors. An example of progress already made is reflected in the following email message from the mother of one of the children we have studied: “I wanted to let you know that after much discussion with doctor at the Office Of Medical Investigators regarding my daughter’s cause and manner of death, a new computer coding for sudden unexplained death in childhood has been established. The Office of Medical Investigators is going back two years to recode deaths that fit this description.” It is our hope that our efforts will stimulate other investigators to pursue the many unanswered questions about this unique and rare population of childhood deaths.

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Author, year	Study Size	Ages	Comments
Molander, 19821	43	1-20 years	The cause of death was not be established in 4 cases with ages of 2 yrs, 13 yrs, 14 yrs, and 17 yrs. SUDC in this study represented an incidence of 0.007 per 1,000 live births. Three of the 4 cases were similar to SIDS.
Neuspiel, Kuller, 19852	207	1-21 years	207 cases of sudden unexpected natural death. Incidence was 4.6 per 100,000 live births For the 1-4-year-age group, 15 of 62 cases had undetermined cause of death; for 5-13-year age group, 3 cases had undetermined causes of death.
Siboni, Simonsen, 19863	1,920	2-30 years	Only one case with an undetermined cause of death among 78 sudden unexpected natural deaths, a 22-year-old female who suffered a cardiac arrest a month after childbirth.
Southall, 19874	9,856	1-5 years	5 children aged 16 months to 4 yrs died suddenly. The SUD incidence after age 1-year was 1/16th (0.125 per 1,000 live births) the SIDS incidence of (1.95 per 1,000 live births). One-third of deaths in this sample of children 1 to 5 years were sudden and unexplained.
Hoffman, et al, 19885	757 SIDS	2 weeks-2 years	Included 16 cases (2%) between the ages of 52 and 103 weeks classified as “definitely” or “probably” SIDS.
Keeling, Knowles, 19896	169	2-20 years	11 deaths were unexplained comprising 1% of the necropsy population or 6.5 % of natural deaths, a much smaller proportion than pertains in infancy. Of the 11 cases, 2 were originally called “SIDS” including a 2 year old and a 3 year old.
Norman, et al, 19907	126	Birth – 17 years	Two case sets of interest were described: 1) “SIDS-like cases with confounding factors” – a 14 _ month old was in this category 2) “metabolic cause unknown” –a 15 month old was in this category. At one point, the latter case was apparently described as having “findings of typical SIDS.”
Helweg-Larsen, Garde, 19938	68	1-5 years	11 of the 27 natural sudden deaths were classified as unexplained or “late SIDS,” which equals an incidence of 0.04 per 1,000 live births compared to the Danish SIDS incidence of 1.6 during the 1st year of life.

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Unexplained fetal death in Queensland 1994-1999: A population-based analysis using routinely collected data

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Background:

With decreasing perinatal mortality rates, the relative contribution of unexplained antepartum deaths (UAD) appears to be on the increase. A recent analysis of perinatal deaths in the year 2000 within three states of Australia (South Australia, Queensland and Victoria) involving 129 735 births and 1 302 perinatal deaths (867 fetal deaths) found an overall rate of UAD of 2.0 per 1 000 births which is more than three times the rate of SIDS. UAD were the largest group of fetal deaths contributing 30%to all fetal deaths and the second largest group of perinatal deaths (20% of all perinatal deaths) second only to deaths from congenital anomaly, which accounted for 25% of all perinatal deaths.

Objective:

To describe the population of unexplained fetal deaths in Queensland over a 6 year period (1994-1999) to provide a basis for the development of future research aimed at reducing the risk of (UAD).

Methods:

A clinical classification system for perinatal deaths developed to identify areas where improvements can be made by determining the single most important factor, resulting in the death was applied to all perinatal deaths over the period 1994-1999. Within this classification, fetal deaths prior to the onset of labour or membrane rupture where no cause for the death was able to be determined following review of all relevant available clinical information are classified into the category of "Unexplained antepartum death". All UAD were compared with infants surviving the neonatal period with respect to maternal characteristics, which formed part of the routinely collected data in Queensland.

Results:

Over the study period 1994-1999, there were 2 096 fetal deaths of which 29% were UAD. A total of 286 269 infants survived the neonatal period and 610 were classified as unexplained antepartum fetal deaths. The results of preliminary univariate analysis are given below.

	Infants surviving neonatal period n=286, 269	Unexplained antepartum death n=610		
	n (%)	n (%)	RR (95% CI)	
Maternal age				
<18	5933 (2.1)	15 (2.5)	1.22	0.54
35+	36 701 (12.8)	89 (14.6)	1.17	0.20
Primiparity	112 634 (39.4)	284(46.6)	1.34	<0.01
Indigenous	15 334 (5.4)	51 (8.4)	1.62	<0.01
Antenatal care type				
Shared care	108 664 (38)	168 (27.5)	1.19	0.04
Place of birth				
Major public hospital	93 160 (32.5)	251 (41)	1.45	<0.01
Antenatal Visits				
0 or 1	4725 (1.7)	40 (6.6)	4.16	<0.01

Conclusions:

Preliminary analysis indicates Indigenous status, primiparity, Shared antenatal care, birth in a major hospital, and less than 2 antenatal visits are associated with UAD when compared with infants surviving the neonatal period. The results of multivariate analysis will be presented.

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Cause specific perinatal mortality in Australia and New Zealand using a new clinical classification system (ANZACPM and ANZNDC)

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Background: A clinical classification system for perinatal deaths (Australia and New Zealand Antecedent Classification of Perinatal Mortality - ANZACPM; and the Australia and New Zealand Neonatal Death Classification - ANZNDC) has been developed by a group of clinicians and researchers in Australia and New Zealand to address perceived deficiencies with existing perinatal mortality classification systems. The key purpose of the classification system is to identify areas where improvements can be made by determining the single most important maternal, fetal or neonatal factor, which triggered the chain of events resulting in the death.

Objective: To apply a clinical classification system to a large cohort of perinatal deaths, to facilitate identification of categories where preventive strategies may be developed to further reduce the perinatal mortality rate.

Method Design: Three states (South Australia, Victoria, and Queensland) contributed to the analysis of perinatal deaths which occurred in the year 2000. The ANZACPM was applied to all perinatal deaths and additionally the ANZNDC to all neonatal deaths, which occurred in three states in the year 2000 using information obtained from death certificates, the State perinatal collection form, autopsies and other relevant clinical information. The categories were then assessed with respect to potential preventability. Comparisons were also made between the Indigenous and non-Indigenous populations.

Results:

A total of 129 735 births and 1302 perinatal deaths occurred in the participating states in the year 2000 giving an overall perinatal mortality rate 10.04 per 1000 births. The three leading causes of death were consistent in all states, being congenital abnormalities (25%), unexplained antepartum death (20%) and spontaneous preterm labour (18%).

	Type of perinatal death								
Cause of death	Fetal death			Neonatal death			Total		
ANZACPM	**n**	**%**	**rate[1]**	**n**	**%**	**rate[2]**	**n**	**%**	**rate[1]**
Congenital abnormalities	171	19.7	1.32	149	34.5	1.16	320	24.6	2.47
Unexplained antepartum death	257	29.5	1.98	0	0.0	0.00	257	19.7	1.98
Spontaneous preterm	92	10.6	0.71	141	32.6	1.09	233	17.9	1.80
Specific perinatal conditions	84	9.7	0.65	31	7.2	0.24	115	8.8	0.89
Antepartum haemorrhage	59	6.8	0.45	24	5.6	0.19	83	6.4	0.64
Maternal conditions	71	8.2	0.55	8	1.9	0.06	79	6.1	0.61
Fetal growth restriction	66	7.6	0.51	9	2.1	0.07	75	5.8	0.58
Hypertension	27	3.1	0.21	18	4.2	0.14	45	3.5	0.35
Perinatal infection	23	2.6	0.18	19	4.4	0.15	42	3.2	0.32
Hypoxic peripartum death	18	2.1	0.14	21	4.9	0.16	39	3.0	0.30
No obstetric antecedent	2	0.2	0.02	12	2.8	0.09	14	1.1	0.11
Total	**870**	**100.0**	**6.71**	**432**	**100.0**	**3.35**	**1302**	**100.0**	**10.04**

1 per 1 000 births, 2 per 1 000 livebirths

Conclusions:

There was good correlation between three states in the major categories of perinatal mortality using a new clinical classification. Analysis of cause of death from three Australian states highlights that preventive strategies and research need to be focussed on three categories: congenital malformations, unexplained stillbirths and spontaneous preterm birth, which together comprise nearly two thirds of perinatal mortality.

Acknowledgments:

State Committees contributing data - Queensland Maternal Perinatal Quality Council, Victorian Consultative Council on Obstetric and Paediatric Mortality and Morbidity, South Australian Maternal, Perinatal and Infant Mortality Committee.

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A kick from within; The story of a cancelled progress in antenatal care

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Objective:

Interest for maternal fetal movement counting as a screening for fetal well being boomed during the 1970ies and 80ies. Several reports demonstrated how introduction of counting charts reduced stillbirth rates significantly. However, in 1989, a large study appeared in The Lancet that would annihilate research in this field. Charts were deemed ineffective, but in retrospect, the evidence was missing. This review tells the story of fetal movement counting and revisits the significance of fetal movement counting in predicting outcome and reducing stillbirth rates.

Methods:

A search was performed in October 2002 in MEDLINE, The Cochrane Library and The BIBSYS database. All 1379 publications including the term fetal movement(s) were screened to identify materials on pregnancy outcomes and its associations to maternal perception of fetal movements. Reference lists and ISI web of Science were searched for further materials in PhD theses and major textbooks in obstetrics. Suspected preliminary or redundant materials were excluded. Only publications from Western countries after 1970 were included.

Results:

Twenty-four materials were identified. Outcome of reduced fetal movements was reported from nine risk pregnancy materials and eight total populations where fetal movement charts were used, and seven total populations in which no charts were used. Five studies were case-control studies on the effect of fetal movement charts. Available data demonstrate that reduced fetal movements are associated to adverse pregnancy outcome; both in high and low risk pregnancies. Increased vigilance to maternal perception of movements, e.g. by performing movement counting studies, reduce stillbirth rate, in particular stillbirths deemed avoidable. The use of fetal movement charts in a population decrease the rate of avoidable stillbirths.

Conclusions:

Maternal fetal movement counting is a cheap, easy, daily, valuable and harmless screening for fetal well being, and can reduce fetal mortality rates, but a resurrection in research activity to optimise its benefits is urgently needed.

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Maternal health in sudden intrauterine unexplained death - Do urinary tract infection protect the fetus?

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Objective:

Unexplained stillbirths are major contributors to the developed worlds perinatal mortality. Maternal health has profound impact on pregnancy outcome, and maternal risk factors for sudden intrauterine unexplained death (SIUD) such as high maternal age, overweight/obesity, smoking and low education, suggest that maternal health influences the risk. Surprisingly, studies have reported significantly better maternal health in unexplained stillbirths than in controls. These materials were however not corrected for the available period of observation (duration of pregnancy), nor fully validated by post mortem examinations. We have revisited this hypothesis.

Design:

All 76 cases of SIUD and 165 explained stillbirths among singletons in Oslo, Norway (1986-1995), were compared to 582 controls. Details on the collection and identification of valid cases have been published previously (1). Information on maternal health was registered from antenatal health-cards and the Medical Birth Registry of Norway. The effect of pregnancy duration was studied before logistic regression analysis corrected for pregnancy duration was performed.

Results:

The time of observation was significantly shorter in stillbirths than in live births, and all subsequent analyses of non-chronic disorders were therefore corrected for the duration of pregnancy.

Urinary tract colonization or infection in pregnancy was associated with a reduced risk for subsequent SIUD (OR 0.29 (0.12-0.74) - irrespective of the use of antibiotics - while insufficient physiological hemodilution during pregnancy (lowest hemoglobin > 13g/mL) increased the risk for SIUD (OR 9.50 (1.30-69.3)). However, the risk for SIUD remained unaffected by the total number of indicators of impaired maternal health during pregnancy, in contrast to significant impact on other stillbirths.

Conclusions:

Urinary tract colonization or infection during pregnancy may subsequently offer strong protection against SIUD. We hypothesize that the subsequent maternal immune response offers transplacental protection against lethal fetal infections by common pathogens of urinary tract infections. Other health indicators have little impact on SIUD.

References:

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A Strategy of Antepartum Testing to Reduce the Risk of Unexplained Stillbirth

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Objective:

To evaluate the trade-offs of a strategy of antepartum testing late in pregnancy to prevent unexplained stillbirths.

Methods:

We used a markov model to simulate three strategies: No testing; weekly testing after 37 weeks and induction with a positive test; and no testing but induction at 41 weeks of gestation. We used risk estimates of stillbirth and the risk of c-section from three databases.

Results:

We adjusted the odds-ratio for fetal death from 1.0 (that estimated for the general population) to high estimate of 5.0. A strategy of antepartum testing was associated with the greatest number of unexplained stillbirths averted but was also associated with an increase in the inductions and c-sections. Because stillbirth is relatively uncommon, weekly antepartum testing with a fixed specificity resulted in similar numbers of positive tests and was not significantly influenced by the risk of fetal death. We found that the estimated number of fetal deaths averted increased as the risk estimate was increased. Using a model of a low risk women (less than 35 years of age, OR=1) a strategy of antepartum testing initiated at 37 weeks of gestation required 2252 antepartum tests, 184 inductions and 31 additional cesarean for each fetal death averted with an estimate of 0.5/1000 fetal deaths averted. For a primiparous women 35 years of age or older (OR 3.3), with no antepartum testing an expected 6.4 /1000 unexplained fetal deaths would occur between 37 weeks of gestation and 41 weeks of gestation. For primiparous women 35 years of age or older, a strategy of antepartum testing initiated at 37 weeks would avert 4.9 fetal deaths/1000 births and would require 703 antepartum tests, 50 inductions and 10 additional c-sections per fetal death averted. If no antepartum testing was performed but all primiparous women 35 years of age or older were induced at 41 weeks, 1.5 fetal deaths/1000 births could be averted requiring 258 inductions, and 110 additional c-sections per fetal death averted.

Conclusions:

A strategy of antepartum testing in high risk women result in a significant reduction of the risk of unexplained stillbirth but would require approximately 50 inductions and 10 additional c-sections per fetal death averted.

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A bereavement support model for children, adolescents and their families in hospitals

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A bereavement support model for children, adolescents and their families in hospitals.
Theoretical and Practical Implications:
The Goals for the Support Program;

The overall goal is to minimise complicated grief
- Improve support routines in Hospital for bereaved parents, siblings and families, acute and longitudinal
- Improve competence in bereavement support for healthworkers in Hospital.
- Tighten connections between Hospital, community care and SIDS organisation.
- Establish a long term program for bereaved children, adolescents and their families
- Create a model for a national program in Hospital bereavement support.

The Support Program;
- Phone contact and supervision for families and health workers
- Family care, creating supportive atmosphere and bereavement knowledge for all members
- Bereavement support groups for children, adolescents and parents. A structured program.
- Teaching programs for health workers, 1. Family support in general, 2. Supervising bereavement support groups, 3. Complicated grief and therapeutic approaches.
- Evaluation and a national model for professional support.

Theoretical Model;
Coping model for children and adolescents
- emotional and social competence
- involvement and care

Family theories
- family structures, phases, relations etc.
- ethnicity and society
- coping model for families

Practical Implications;
- Children and adolescents are the centre of focus for the family.
- All members of the family have to share their bereavement story and we connect the stories.
- Information and support in the bereavement process, and differences in family members reactions.
- Focusing on coping, family coping and society.

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Ethics and Autopsy

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Until recently many pathologists did not recognise ethical dilemmas in their day-to-day autopsy practice, secure in the belief that what they did was legal, good pathology, and in the best interests of parents and the general public. In recent years such well-meaning paternalism has become unacceptable, and past practices may conflict with present day ethical standards and parents' wishes. This brief review will discuss some ethical issues that arise in the context of autopsy following cot death. It does not pretend to provide definitive answers. Consensus can only be approached by discussion involving all interest groups, especially parents, taking into account the legal and cultural values of the individual, community or country.

All medical practice should be ruled by basic underlying ethical principles such as beneficence (conduct likely to produce benefit), non-maleficence (avoidance of actions likely to cause harm), respect for autonomy of the individual, and justice (treating all individuals as of equal value). To these might be added professional integrity, which requires that an individual's work should be of a high standard as well as requiring him or her to prevent poor practice by others. A poor autopsy is an unethical autopsy.

All societies instinctively treat the human body with respect. In ethical terms the body is said to have both intrinsic value reflecting our respect for the individual that was, and instrumental value to relatives as a focus of the grieving process, or to society e.g. as a source of tissue for education and research.

Consent is central to ethical medical practice, and in pathology consent is necessary to respect the intrinsic value of the body of the deceased and the autonomy of relatives. In many countries consent is not required when death has been sudden and unexpected because it is held that the public good over-rides parental rights in this situation. This does not absolve professionals from their other ethical obligations to have respect for the integrity of the body, to acknowledge the autonomy of parents by providing explanation and where possible choice, and to treat them with justice.

Parents should be given sufficient and sensitive explanation of what post-mortem entails to meet their individual needs. It is often said that it would be unkind to explain the process of autopsy to parents in any detail at the time of recent bereavement. It is rarely ethical to withhold information. This kind of "white lie" is fraught with hazard and should pass several tests; we should never tell an untruth; withholding information should be clearly in the parents' best interests, and not our own; we should not underestimate the ability of parents to deal with the truth; our decision should be able to withstand scrutiny by others outside the medical profession. Seen from this perspective, deliberately withholding information about post-mortem examination is unacceptable.

The post-mortem examination must not exceed that strictly sanctioned in law unless specific permission has been obtained from the child's parents for additional studies. The ethical pathologist will wish to ensure that he/she has met the parents' need to know what has taken place at the post-mortem examination, and what samples he kept. When the legal process has been concluded, relatives have a right to possession of the body, and morally this includes any tissue samples or organs that were retained for further examination. Active steps should be taken to offer their return, and explanation of alternative uses to which they may be put.

Pathologists should not forget that photographs, paraffin embedded tissue blocks, microscope slides, small samples of frozen tissue, and fibroblast cultures which they take for granted may be of great significance to parents. There is active discussion in several countries about the extent to which paraffin blocks and slides still represent the tissues from which they were made, and whether they should be retained indefinitely by pathology departments as part of their records and for future medical, legal and research purposes.

While carrying out a post-mortem examination a pathologist may encounter a condition with implications for the family but beyond the remit of investigation sanctioned in law (for example, a genetic condition unrelated to the cause of death). In the author's view minimum samples should be taken to ensure that adequate tests can be done to protect the interests of the family, but that they should be told at the earliest opportunity and their permission sought before it is too late to return the samples to the body. A more extreme view is that nothing should be removed from the body without prior authorisation, even if it would be in the parent's interests to do so.

The number of medicolegal autopsies carried out varies widely between different countries without any apparent differences in harm or benefit to those communities. In some countries with high rates of medicolegal post-mortems some examinations are arguably unnecessary, and so unethical if carried out against the parents' wishes. Examples might include a child who dies on admission to hospital and pre-mortem blood cultures are expected to grow meningococcus. In such circumstances the pathologist might decide to offer a written report without formal post-mortem examination if allowed in law.

Research is a subject of great concern both to parents who have suffered a cot death and to pathologists. It is perfectly proper to use archival post-mortem slides and blocks for teaching and research. However, this requires parental consent. Since it is not possible to specify in advance the precise uses to which they will be put, it is sufficient for most purposes to obtain prospective general consent to their use for teaching and research. Approval for all research projects must be obtained from the institutional research ethics committee. Occasionally research using archival blocks and slides will inevitably be traceable to a small group of individuals (e.g. those with a very rare disorder) or have implications for families (e.g. research involving genetic conditions). In such cases it may be necessary to go back to their representative group or the parents themselves for additional consent.

If it is proposed to take extra tissue prospectively for a research project then this requires separate explanation, ethical approval by the institution's research ethics committee, and an information sheet and separate consent form as for any other clinical research project.

Careful consideration should be given to the need for secure coding or full anonymisation of any material used in research.

Specific consent is also necessary for the retention and display of organs and tissues in medical museums or collections. Some parents value the opportunity to "gift" a diseased organ to a museum or for research, much in the way that blood or organs for transplantation are donated. The emphasis should be on the positive act of giving, rather than permission to keep, and may have a redemptive aspect for the giver enabling some good to come from death.

Post-mortem examination is likely to be least intrusive and of most benefit to parents if it is seen as a partnership in which the special knowledge of the pathologist and the values of individual parents are combined to enable parents to choose, so far as is possible, what happens to their child. Parents or groups of parents who have lost children through SIDS may then feel able to promote post-mortem examination and its use in research to try to prevent such deaths in the future.

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Tissue and organ retention - parent's rights

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In Australia autopsies are mandatory for all sudden unexpected deaths. The majority of Australians do not know how autopsies are performed although this is changing with several television programs now 'exploring' the world of forensic pathology and investigation. In the mid 90's our organisation faced a challenge from a parent as to the rights and ethics of retaining tissues from infants, autopsied by law and without consent, for use in research projects. We set about defining the ethical and moral issues and what were the parent's rights.

In 2001, following similar investigations in the UK the media raised these issues nationally and autopsy practices were scrutinised both internally within the relevant organisations and publically via the media.

The resultant recommendations from this investigation have created additional stress for many families at one of the most devastating times in their lives.

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The perinatal autopsy: Integration into a multidisciplinary bereavement program

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Objective:

To study the value of the perinatal autopsy as part of the bereavement process. To provide an adequate autopsy report as an instructional tool for anticipatory guidance in future pregnancies.

Method:

A senior nurse clinician, conducts prenatal classes also functions as the leader of a multi-disciplinary bereavement team for perinatal loss. Women are informed of the benefit of an autopsy and instructed to make an appointment in 30 days for a post mortem conference. Team members include: obstetrics, social service, nursing, and pathology. Family, friends and physicians in training participate in an encounter that averages 30 minutes. Questions are addressed and a copy of the autopsy report provided. Mothers call if additional concerns arise and are advised to inform their obstetrician of the autopsy report if they become pregnant again.

Results:

During a 4-year period with an average of 2000 births per annum, the autopsy rate for perinatal loss was greater than 50 %. This figure was constant for fetuses over and under 500 grams. Fifty percent of mothers who requested autopsies attended post mortem conferences. A total of 196 stillbirths had 106 autopsies performed and 60 post mortem conferences conducted. Of the total stillbirths, 103 were greater than 500 grams and 93 less than 500 grams.

Conclusions:

1. Post mortem examination and placental evaluation can provide a plausible explanation of perinatal loss in over 95 % of cases. 2. The family is grateful for assistance in their effort to understand their personal tragedy. 3. The pathologist is an information therapist who has the respect of the lay public. 4. The ability to grasp complex medical information and ask penetrating questions is independent of educational level, language barriers and socio-economic status. 5. Legal action is not promoted by expert pathological examination. 6. Involvement in the bereavement program improves the quality of perinatal pathology practice and contributes to improvement of obstetrical outcomes.

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Parents' views on information and routines of autopsy after loosing a child in sudden infant death.

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Objective:

This presentation sums up parents' views on which information they should be given on autopsy before and after the procedure, and how this information should be given. Some of the parents' reactions to the changes in their dead child after the autopsy will also be described.

Method:

19 couples of parents participated in an evaluation study of family reactions to voluntary death-scene investigation, after sudden infant death. In the evaluation study several of the parents commented on the way they had been informed before the autopsy, and how they had reacted to changes in the appearance of their child after the autopsy. They also evaluated the way they had been informed of the results of the autopsy.

Results:

Some of the parents experienced the information they received as inadequate or confusing, some of them thought the information was given in an insensitive or random way. All parents evaluated the information they were given of the results of the autopsy as understandable and thorough. The fact that this information was given by the pathologist who performed the autopsy was evaluated as especially positive.

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Pediatric autopsies – a need for information?

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Background:

During the last years there has been a growing public interest in, and a demand to know more about various medical issues. The media, in Norway as well as in other countries, have had reports about autopsies of varying quality and insufficient information to the bereaved families. In particular, the families have felt deceived not being informed about removal of vital organs that have not been put back in the body after the autopsy.

On this background there has been a growing wish and need to improve the standard of the pediatric autopsy. Thus, two years ago, a group of doctors with special interest in pediatric pathology and representatives from parental organisations started their work to make a protocol with guidelines for the performance of pediatric autopsies. The protocol includes technical as well as ethical demands.

Why autopsy?

An autopsy is in most cases necessary to be able to establish a reliable diagnosis, and thus for as good information to the parents as possible. Furthermore, it is very important for the education of health personnel and for the preventive health work.

What information is needed?

According to Norwegian regulations, the hospitals have a duty to follow up the bereaved families. Two brochures, one for the families and one for the health personnel have therefore been made as an aid in the information about autopsy. It deals with he reasons for autopsy, how is it performed and what happens to the organs after the investigation.

About the organs:

All internal organs are removed and examined before they are put back into the body. An exception is the brain and heart, which have to be treated with special fixation baths before they can be examined further. This treatment is usually not finished until the funeral or cremation of the child has taken place. Therefore, after all examinations have come to an end, the organs will be incinerated, cremated or buried, depending on the local practices and the wish of the parents. Usually, only small specimens of tissue, blood etc will be stored for further evidence, research or teaching purposes.

The main goal of the working group has been to secure a good professional and ethical performance of the pediatric autopsies, as well as finding a way of meeting the bereaved families need for and right to proper information.

[1)] On behalf of the national working group: Roger Bjugn, Bjarne Beck, Aage Erichsen, Bente Guildal, Gunnar Houge, Christina V. Isaksen, Trine Giving Kalstad, Tor-Arne Hanssen, Torvid W. Kiserud, Helga Maartmann-Moe, Karen Rosendahl, Ståle Sund, Åshild Vege.

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Changing prevalence of modifiable risk factors for SIDS in socioeconomically different populations in Hamburg/Germany during a 6-year period (1996-2001)

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Objective:

In many countries, risk reduction campaigns in the late eighties and early nineties have led to a considerable decline in the incidence of SIDS. Despite the fact that there never was a countrywide campaign in Germany, until today the SIDS rate has reached the same level as it has in other countries (with the exception of the Netherlands). To develop the most effective strategies for further local campaigns it is of importance to know about the prevalence and distribution of modifiable risk factors in the population of a given community. Continuous monitoring of parents' risk-related behaviour over the years makes it possible to recognize negative changes as early as possible.

Method Design:

In 1996, 1998, and 2001, parents of healthy infants were interviewed about their smoking-, feeding-, and bedding-habits (n=2001, 2027, and 1752, respectively) in the federal state of Hamburg. The interviews were carried out in pediatric practices and offices of mothers' health care consulters. Socio-economic status (»high«, »normal«, or »low«) was determined by the average socio-economic standard of the district (consisting of various indicators) of each participating practice. The SIDS rate was estimated relying on the evaluation of death certificates and data provided by the Institute of Legal Medicine in Hamburg.

Results:

In Hamburg, the SIDS rate was 0.90 (per 1000 life births) in 1996 and 0.63 in 2001 with considerable differences between the different socio-economic groups (e.g. 0.90 in the low status group vs. 0.29 in the high status group in 2001). The proportion of parents who preferred to put their baby in the prone sleeping position decreased from 8.1% in 1996 and 5.6% in 1998 to 3.2% in 2001. The group with low socio-economic status had a higher prevalence of prone sleeping than the high status group (4.6% vs. 1.1% in 2001). Smoking during pregnancy decreased from 22.2% and 19.7% to 19.3%, respectively, with a higher prevalence in the low status group (23.7% vs. 12.7% in 2001). More results from this study will be presented.

Conclusions:

In Hamburg, there are considerable differences in the SIDS rates depending on the socio-economic status. Most likely this phenomenon goes back to different prevalences of risk factors in socioeconomically different districts. The prevalence of prone sleeping is continuously decreasing which can be considered as the main reason for the decline of the SIDS rate in the past years. Further efforts are needed in order to provide better preventional strategies for the population with low socio-economic status.

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Risk factor changes in 15 years (1985-2001) in the Netherlands

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Abstract:

The Netherlands is a country with a low cot death incidence of 0.17/1000 live born infants in 2001. The complex of risk factors has changed over the years. The following prevalence of well-known risk factors were found:

In 29 of the 136 included infants in the cot death study between 1996 and 2001 the parents had been taken these risk factors in account: the infants slept supine, did not use a duvet, parents did not smoke and no sedatives were used. The mean age of these infants did not differ from the other cot death infants. Twenty-three of the twenty-nine cases (presumably) died between 8 o'clock a.m. and 8 o'clock p.m., mainly during the day. Fourteen of the 29 infants died in secondary prone sleeping position, of which 11 with the face straight down. There seems to be a subgroup of cot death cases, which mainly die during the day.

In the period September 1st 1996 to September 1st 2001 the Dutch Cot Death Committee collected data on 136 cot deaths. In the age group 3-5 months only 25% of the cot death infants used a Dutch sleeping bag. In the same age group 47% of the baby's at well baby clinics had slept in such a sleeping bag. The sleeping bag thus offers significant potential for prevention. There was no significant preventive effect of usually sleeping with a dummy. For sleeping with a dummy in reference sleep there unfortunately were no control data. Furthermore, in the age of 0-4 months 24% died while sharing a bed with parents and a larger than expected amount (16 versus 4) died while in a day care setting.

Conclusion:

In conclusion, these data show that prevention is effective, and that the search for new risk factors should continue.

	cot death infants				population based study
age n	1985/87 <6mnd	1984/91 <24 mnd	1995/96 <24mnd	1996/01 <24mnd	1999 <10mnd
Primary prone sleeping	108	222	74	136	2528
Placed on the side	78%	69%	24%	15%	8%
Duvet-use last night	13%	11%	20%	14%	8%*
Pillow use last night	-	73%	55%	38%	18%
M smoked after pregnancy	4%	-	1%	15%	2%
F smoked after pregnancy	44%	41%	40%	40%	20%
At least one parent smokes	51%	-	44%	48%	32%
Used sedative for child	-	55%	68%	60%	39%
None of these factors	-	8%	5%	1%	-
* 1-3 months	-	3%	10%	18%	-

* 1-3 months

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An analysis of SIDS risk factors by CHAID

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Objective:

To determine whether analysis of the New Zealand Cot Death Study by Chi-squared Automatic Interaction Detector would have helped to determine interaction between SIDS risk factors that were not immediately apparent from standard logistic regression techniques.

Methods and Design:

The Answer tree module of SPSS was used to analyse data from the New Zealand Cot Death Study. The New Zealand Cot Death Study contains information on 393 cases and 1592 controls and took place between November 1987 and October 1990. Variables contained in the main analysis of the data which identified 4 modifiable risk factors1 are used in the analysis.

Results:

The first split and hence that resulting in the optimal split of groups was carried out on maternal smoking. The odds ratio for smoking was 4.06. Following this both the smoking and non-smoking groups had sleep position as the next optimal split, the split in both cases grouping back and side together and separate from prone. The Odds ratio for prone sleep position was 3.47 amongst the smoking group, and 4.81 amongst the non-smoking group. Splits beyond this point occurred on a number of different variables and though not in the same order there was some consistency in the variables on which subgroups were split.

Conclusions:

CHAID analysis is an additional tool that may be of particular use in case-control studies to detect relationships and interactions between risk factors. In this case it has confirmed that smoking and sleep position are the two most important risk factors for Cot Death in this dataset.

1. Mitchell EA et al. Four modifiable and other major risk factors for cot death: The New Zealand Study. J Paediatr. Child Health 1992: Suppl. 1, S3-8.

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Co-sleeping and sudden unexpected infant death

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Debate continues as to the possible risks and benefits of co-sleeping with an infant. Some studies have reported that this practice carries no increased risk as long as the mother does not smoke. However, many health care professionals are concerned that the parental bed may not be the ideal place for a baby to sleep.

Objective:

To examine the possibility that co-sleeping with an infant is associated with an increased risk of sudden infant death syndrome.

Method Design:

As part of four-year case-control study of sudden unexpected infant deaths in Scotland we examined the sleep environment of 131 SIDS cases and 278 age-matched controls. Home visits were made to complete a questionnaire within 28 days of the index case's death. Information was collected on both normal sleep practices and those of the day/night of death.

Results:

On the night of death, 65/129 (50%) index cases co-slept with parents compared with 55/278 (20%) control infants OR 4.12 (95%CI 2.55, 6.66). For mothers who smoked 51/99 (52%) index cases co-slept as did 16/93 (17%) control infants OR 5.11 (95%CI 2,50, 10.56). For mothers who did not smoke 14/30 (47%) index cases co-slept as did 39/185 (21%) controls OR 3.28 (1.37, 7.83). There was no significant interaction between co-sleeping and maternal smoking.

Conclusion:

In the Scottish population, co-sleeping is a risk factor for sudden infant death, even for mothers who do not smoke. There may be other factors responsible for the increased risk of SIDS for babies co-sleeping with parents. This paper will discuss hypotheses generated by this case control study.

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A case controlled study of SIDS in the republic of Ireland: Factors relating to the infant's sleeping environment

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Objective:

Maternal smoking and use of the prone position are the most consistent risk factors associated with SIDS whilst there has been conflicting evidence in the literature regarding the role of co-sleeping and/or bed-sharing, having being reported either to increase the risk or to have no effect. Differences between studies are likely due to ethnic or social differences, variability in the prevalence of other risks factors for SIDS or differences in the statistical methods used to analyse data. The objective of this study was to examine the role of co-sleeping and other factors relating to the infant´s sleep environment as risk factors for SIDS using data collected from a case control study conducted in the Republic of Ireland between 1994 and 2001.

Methods:

The study utilised data on every infant who died from SIDS between 1st January 1994 and the 31st December 2001. Parental interviews were conducted for each infant who died (n = 332) and for controls matched for age and geographical location (n = 1137). Details were obtained on socio-demographics, parenting practices, precise sleeping arrangements and details of events occurring at the time of the infant´s last sleep. A corresponding reference sleep period was used for controls. Conditional logistic regression was used to investigate differences between cases and controls using STATA version 6. Odds ratios and 95% confidence interval levels were used for comparisons. Co-sleeping was defined as any shared sleeping arrangement of an infant with a parent(s)/relative in or on a bed/sofa/armchair.

Results:

Co-sleeping was found to increase the risk of SIDS by a factor of 15 (OR 15.31; 95% CI 9.10 - 23.77). Only 6% of controls were co-sleeping during their last sleep period compared to 43% of cases. At the time of their last sleep, 28% of cases were unaccustomed to this practice. The associated risk varied with duration; infants who were bed-sharing for the entire sleep period being most at risk (OR 13.37, 95% CI 8.07 - 22.17). However, we found no significant risk associated with bed sharing for a period of <1 hr (OR 0.78, 95% CI 0.35 - 1.77). On multivariate analysis, bed sharing became non-significant for infants >= 16 weeks of age. The risk associated with co-sleeping was only slightly reduced when maternal smoking habits were controlled for (OR 12.42; 95% CI 6.96 - 22.17). Other factors which emerged significant as in a multivariate analysis were use of the prone position and absence of routine soother use whilst use of pillows, duvets and bed coverings of tog >=10 became non significant.

Conclusions:

These results indicate that infants who co-sleep are at an increased risk of SIDS. This risk is influenced by maternal smoking habits and increases with duration of co-sleeping. With no significant risk associated with bed sharing for a period of less than one hour/night. The precise circumstances and complex interactions involved in co-sleeping require further investigation/clarification before it can be advocated as a safe practice. Use of the prone position remains a strong risk factor along with absence of routine soother use. All risk factors identified in this study are amenable to change.

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Smoking and SIDS over 8 years: Current Irish epidemiology

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Objective:

To examine the effect of infant cigarette smoke exposure on the risk of Sudden Infant Death Syndrome in a contemporary Irish epidemiological database ranging from 1994 to 2001.

Method:

An eight -year prospective population based case control study was conducted using data for infants dying from SIDS from the 1st January 1994 to 31st December 2001 inclusive. The diagnosis of SIDS was accepted when used on the death certificate after a thorough post-mortem examination. Control families were picked randomly from the birth register with matching for the date of birth and geographical location of the SIDS case. Four control infants were picked for each SIDS case. Eighty one per cent of the SIDS families and 77% of the control families agreed to participate in the study. Conditional logistic regression was used to investigate differences between cases and controls using the statistical package STATA version 6. Multivariate analysis included devising a social disadvantage index. Simultaneous testing was applied to investigate the variables that were significant in the univariate and subsequent multivariate analysis at 5% level of significance.

Results:

In total, there were 332 SIDS cases and 1137 control infants involved in this study. The data shows that 74% of SIDS mothers smoked during pregnancy compared to 26% of control mothers giving an odds ratio, of increased risk of SIDS in smokers, of 8.08 (CI 5.68-11.49 $p<0.001$). The data also shows a dose response effect with an odds ratio of 5.72 for those smoking 1-10 cigarettes per day (CI 3.82-8.56), rising to 14.1 for those smoking more than 10 cigarettes per day (CI 8.98-22.13). Other data shows that 67% of fathers of SIDS cases smoked during pregnancy compared to 28% of fathers in the control group. The paternal data also shows a dose response effect with odds ratios of 4.00 (CI 2.60-6.16) for the 1-10 cigarettes per day group, rising to an odds ratio of 6.08 (CI 4.14-8.92), for the group smoking more than 10. Other results shows that more other household members of SIDS infants smoked, 26% compared to 7% in the control group. The risk of SIDS increases significantly where both parents smoke compared to only the mother smoking (OR 3.7 rising to 11.3 $p<0.001$).

Conclusion:

Cigarette smoke exposure is a powerful SIDS risk factor increasing the risk of SIDS in Irish infants almost fourfold and also in a dose dependant fashion with the risk increasing with the number of cigarettes smoked. Evidence based information is necessary about smoking but it is also imperative that we seriously address the issue of smoking cessation in order to realistically empower parents to discontinue smoking.

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To be or not to be after ALTE

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Objective:

To investigate why some infants with ALTE (apparent life threatening event) die and others survive.

Method design:

Retrospective epidemiological study based on questionnaires answered by the parents. Fiftyeight consecutive ALTE infants and 56 sex and age matched ALTE control infants as well as 244 SIDS cases and 868 SIDS controls were included. All ALTE underwent a thorough medical examination at the local hospital during a three-day observation period following emergency care. During this period other possible causes of the event such as an infectious, neurologic, cardiac or metabolic disorder were excluded.

Results:

The ALTE infants did not differ from their matched controls with regard to gender (n.s.), sleeping position (n.s.), breast feeding (n.s.), and use of dummy (n.s.). The mothers had similar ages (n.s.), education (n.s.) and incidence of smoking during pregnancy (n.s.).

The age and seasonal distribution when ALTE and SIDS occured were similar.

However, the ALTE infants slept more often in supine position than the SIDS victims when found ($p=0.002$). The ALTE infants were also significantly more often breast fed ($p<0.001$) than the SIDS infants. Nearly 60 % of the SIDS mothers smoked versus about 20 % of the ALTE mothers ($p<0.001$). The mothers of the ALTE infants were also older ($p<0.005$) and had a higher educational level ($p<0.001$),

The incidence of ALTE was found to be 1,9% among SIDS controls, but 7,4% among infants who later on died of SIDS. In the ALTE group survivors had the combination of prone sleeping and maternal smoking in 13.3%, while the corresponding figure of those who became SIDS victims was 33.3%.

Conclusion:

Our results suggest that ALTE is essentially the same entity as SIDS with regard to age and seasonal distribution etc., but due to less nicotine exposure, supine position and higher parental education their risk of dying in SIDS seems to be reduced.

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Multi-agency protocols to coordinate a thorough investigation when a baby dies

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Objective:

Responding when a baby dies requires a multi-agency approach 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 non-natural deaths. The Foundation for the Study of Infant Deaths (FSID) has been campaigning since 2000 for multi-agency protocols in England and Wales.

The recommendations are that:

- Within 24 hours of a baby's death, each bereaved family should be visited at home by a paediatrician to take a full history of events and offer support to the family.
- Every baby should receive a thorough post mortem examination by a pathologist who has special training and follows paediatric protocol.
- A discussion is held after each death with the professionals involved.

Method Design:

FSID regional staff has been visiting coroners, paediatricians, senior police staff, child protection teams and casualty departments to promote the campaign and offer guidelines for multi-agency protocols. FSID staff has initiated meetings with the key professionals to discuss the possibility of working towards a protocol. FSID has also held one conference in December 2001 to promote joint agency working and will host a second conference in July 2003. The second conference will offer guidance on how to get started, bridging the gaps between health, social services and police training, audit and evaluation.

Results and Conclusions:

As at 31 January 2003 13 areas have established multi-agency protocols in England and Wales. A further 12 areas are having meetings to discuss protocols.

The presentation will expand on the way in which areas are working towards multi-agency protocols, the professionals who are taking the lead and some of the pitfalls encountered.

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Parents experiences related to participation in voluntary death scene investigation

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To evaluate parents' experiences related to participation in voluntary death scene investigation. Voluntary death scene investigation after sudden infant death has been implemented as a research program in southeast Norway by the Institute of Forensic Medicine, University of Oslo. The operative phase of the project started in September 1st 2001. The evaluation study started 2 months later. Parents were interviewed in their homes by a clinical psychologist from Senter of Crisis Psychology in Bergen. The interview focused on the parents' reactions to being asked to participate in the investigation, their evaluation of the investigation and how it impacted them afterwards. A total number of 18 mothers and 17 fathers have been interviewed. Almost all the participants have evaluated the participation in the death scene investigation as positive; some of the parents did also find it psychologically helpful. If asked for advice they would recommend other parents to participate in similar projects. The paper addresses the question how a death scene investigation can be psychologically helpful. Questions regarding the parents' reactions to information regarding autopsy will also be discussed.

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The forensic pathologist at the death scene – experiences from the German case-control death scene investigation study on SIDS

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Objective:

Today, investigation of the death scene is widely accepted as an integral part of the SIDS definition. The Institute of Legal Medicine in Hamburg was a participating member of the German case-control death scene investigation study group on SIDS. Primary goal of the study was to obtain statistical data regarding possible risk factors in the sleeping environment of SIDS victims and healthy infants (controls). Exact methodology and epidemiological results will be presented elsewhere on this congress. Apart from this, we report about personal experiences from the forensic pathologist's viewpoint.

Method design:

From April 1999 to October 2001 15 (suspected) SIDS cases and 26 controls have been visited at the scene in Hamburg. Ideally, the examination took place only 1 to 3 hours after the infant was found dead.

Results:

Classification of the cases in 'classical' SIDS cases, 'borderline-cases', natural deaths from other causes and homicides was often facilitated by the observations made at the scene. More than any report written by the police, the examiner's own impression helped to distinguish typical from atypical SIDS scenes and to identify dangerous sleeping environments. In 2 cases the results of the scene investigation led to the suspicion of homicide, later confirmed by autopsy. Selected SIDS and Non-SIDS cases will be presented.

Parents' reactions to the investigator's activities ranged from cooperation to unconcealed hostility in one case. However, in retrospect, most parents felt comfortable with the presence of the forensic expert, which gave them the opportunity to ask questions and convinced them, that every possible measure was taken to clarify the unexpected death of their child. Vice versa, it helped the pathologist to establish contact with the parents as early as possible.

Conclusions:

A death scene investigation leads to a better understanding of individual cases. In many cases it facilitates contact to the parents.

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First results from the German case-control death-scene investigation study on SIDS

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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 observations/measurements in parents' homes, in cases shortly after death. The study protocol included: temperatures; dimensions of room and cot; type, dimensions and weight of all bedclothes; type, dimensions and softness of the mattress; 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) grams 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). Despite the limitation of low numbers, our data suggest that use of a pillow and heavy bedding may be risk factors for SIDS. More results from this study will be presented.

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Better investigation in cases of sudden unexpected death in infants and small children

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Objective:

In Norway death scene investigations are normally not performed in cases of sudden death of infants and small children. This is a problem when diagnosing SIDS since knowledge of the circumstances are required, and best gathered by visiting the death scene and interviewing the parents. In Southeast Norway a research programme, including voluntary death scene investigation, a thorough scientific autopsy and an expert conference in each case, has been implemented. The aim is to improve the legal protection of infants and small children, look for new risk factors for SIDS and ensure a right diagnosis as a basis for research into death mechanisms for sudden infant death.

Method:

The project started April 1st 2001, and during the first phase all hospitals and police districts in the region were visited and given information about the project. The operative phase of the project started September 1st 2001. Information about new cases is immediately forwarded to the Institute of Forensic Medicine, University of Oslo. Autopsies are performed at the institute, preferably within 24 hours, according to the demands of the Nordic SIDS protocol and the diagnosis made according to the Nordic criteria. As soon as possible, in most cases immediately after the autopsy is finished, a death scene investigation is performed by a police expert employed by the institute and by a forensic pathologist. Informed

consent is necessary, and the family may withdraw from the project at any time without giving reasons. All scenes of death are videotaped, and the situation in which the baby was put to bed and how it was found dead is reconstructed by means of a doll of appropriate size. When all investigations are finished an expert conference is held. A psychologist evaluating the impact of the death scene investigation has visited the first 20 families included in the project.

All parents are also asked to fill in a questionnaire. Three live controls matched for sex, age and place of living are picked out by the hospital where the dead infant was borne, and these parents also receive a questionnaire with questions relating to a reference day corresponding to the last day of the dead child.

The project period is 3 years and the estimated number of deaths among infants and small children is approximately 90.

Preliminary results:

Until March 4th there have been 42 deaths. Death scene investigation has been performed in 35 cases, whereas 26 expert conferences have taken place.

Of the 30 deaths diagnosed, 17 have been diagnosed as SIDS, seven as disease and there has been one accident with a pram tipping over. Among the remaining five cases there were two traffic accidents, one of malpractice and two cases were homebirths (infanticide?). The questionnaires have not yet been evaluated, however seven out of 17 of the SIDS cases occurred while in bed with one or two adults.

Conclusion:

In the last 18 months we have realized the importance of seeing the death scene. We are looking forward to evaluating the impact of this new approach by comparing the distribution of diagnoses before and after the death scene investigation was implemented.

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Sleep environment in sudden unexpected death in infancy and early childhood

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- Preliminary results

Objective:

To examine the effect of sleep environment on sudden unexpected death in infants and small children (SUDI) compared to a control population in Southeast Norway, September 2001- February 2003.

Method:

In a 3 year study that started 1st September 2001, all cases of sudden unexpected death between the ages of 0 and 3 years in Southeast Norway are included in the prospective study "Better investigation in cases of sudden death in infants and small children". The study includes death scene investigation with video recording of all cases, as well as a questionnaire to all cases and 3 controls matched for age, sex and place of birth. In this preliminary study 27 SUDI cases were compared with 27 controls based on questionnaire data and analysis of video recording (cases only) for variables connected with sleeping environment on the last sleep (SUDI)/reference sleep (controls); sleeping position when found, co-sleeping, layer of clothing, firmness of mattress, type of duvet used and room temperature. The data for each variable was then scored according to a presumed increased risk of a warm sleeping environment, a score of one giving no presumed risk and a maximum score of four indicating high risk; 20 being the combined worst case score. The data was analysed using X2 -test, multiple logistic regression for calculating odds ratios (OR) with 95% confidence interval (CI) and students T-test for comparing final score between cases and controls.

Results:

The risk for SUDI was higher for prone sleeping, especially with the face straight down in the mattress (OR 21.4, CI 1.4-315.1, p=0.03), sleeping on a soft mattress (OR 12.8, CI 1.5-112.7,

p=0.02) and sleeping in two or more layers of clothing (OR 9.8 CI 1.2-77.4, p=0.03). There were also more SUDI cases co-sleeping, sleeping in a warm room and using a warm duvet than controls, but these differences were not statistically significant in the multivariate analysis. The total score differed significantly between cases (mean score 10.1, CI 9.3-10.9) and controls (mean score 6.6, CI 6.0-7.1) ($p<0.01$).

Conclusion:

These preliminary results indicate that sleeping prone on a soft mattress in a warm sleeping environment increase the risk of sudden unexpected death in infants and small children. Whether this risk differs between different diagnoses in the SUDI group remains to be investigated. We have not split the cases in different diagnostic groups as a few cases still lack results of ancillary tests.

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Used infant mattresses, parity and sudden infant death syndrome

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Multivariate analysis (which included parity) in two case-control studies in Scotland found a valid statistical association between used infant mattress and Sudden Infant Death Syndrome: OR 2.51 (95%CI 1.39, 4.52) in the first study (BMJ 1997; 314: 1516-20) and OR 3.07 (1.51, 6.22) in the second study (BMJ 2002; 325:1007-9). However, it has been suggested that in the multivariate model used parity may still have confounded this association. Clarification of this issue is important as 33% of babies in Scotland sleep on a used infant mattress.

Objective:

To explore the possibility that the observed association is a result of the confounding effect of parity and not due to used infant mattress at all. We used case-control data from the above two studies in Scotland which covered the period January 1992 to May 2000.

Method:

Questionnaires were completed on 278/317 (88%) cases notified in time for an interview within 28 days of death and 554/634 (87%) controls. Parity was determined by asking how many live births has mother had, including this one? Exposure to a used infant mattress was assessed by asking parents about routine sleeping place for both cases and controls and ascertaining the mattress status (new for this baby or not). Routine bed sharers were excluded (35 cases and 33 controls). Analysis of the pooled data was carried out, stratified by families of different sizes.

Results:

The observed association between used infant mattress and SIDS remained, OR 2.26 (1.40, 3.63).

Of more interest is the alternative hypothesis that the observed association between parity and SIDS is in fact due to the confounding effect of used infant mattress and not due to parity at all. The multivariate fixed effects analysis in the second of our two studies inferred this conclusion as parity became non-significant only when used infant mattress was added to the model. Using stratified analysis we confirm that the increase in the risk of SIDS associated with each one unit increase in parity, OR 1.49 (1.28, 1.73), almost disappears and becomes non significant, OR 1.20 (0.95, 1.50), when controlled for used infant mattress and the matched nature of the data.

Conclusion:

Parity does not confound the association between used infant mattress and SIDS; on the contrary, used infant mattress may explain the observed association between parity and SIDS in Scotland.

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Smoking during pregnancy and sudden infant death

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Smoking during pregnancy has been identified by many epidemiologic studies worldwide to be a risk factor for sudden infant death (SID). Recently a dosage-effect-correlation could be described: The risk for SID increases with increasing daily cigarette consumption. The information about the cigarette consumption in most epidemiologic studies has been obtained by parents interview some days or weeks after death. Because the parents are informed about the main risk factors prior to the interview, we suspect that some of the answers were biased by this knowledge.

In 155 SID cases from the German SID study (GeSID) cotinine and nicotine levels were determined in hair, pericardial and cerebrospinal liquid using a specially adapted GC-MS method in SIM-mode following solid phase extraction.

The following conclusions can be drawn:

1. By comparison of the cigarette consumption of case and control mothers the risk for SID caused by smoking is calculated depending on the dosage. 2. Based on the chemically determined nicotine and cotinine concentrations, “normal” values for infants of non-smokers could be defined. 3. The cotinine and nicotine concentrations in the body fluids of infants from smoking mothers showed no clear correlation to the stated cigarette consumption. 4. In cases showing high cotinine levels in the cerebrospinal fluid brain oedema could be found. The pathophysiological significance of this finding remains unclear up to now.

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Changing risk-patterns for Sudden Infant Death Syndrome, Norway 1967-1998

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The effects of several established risk factors for Sudden Infant Death Syndrome (SIDS) have been reported to increase with time along with the decrease in total incidence (Daltveit et al., 1997). We have revisited these changing risk-patterns through analyzes of data from the Medical Birth Registry of Norway for the period 1967-1998. In the previous study we focused on maternal age, parity and marital status. We have in the present study particularly looked at the effect of maternal education on the risk for SIDS. Also, in Norway we observe an increase in pregnancies with a second partner, and we therefore look at the risk for SIDS after a change of partner.

We analyzed all data in Norway for the period 1967-98, covering more than 1.8 million singleton births. The data were linked into units of sibships, and we studied the risk for SIDS in 2nd born children, in families with the same parents in 1st and 2nd pregnancies and in families with a new father in the 2nd pregnancy.

For the first years with low incidence of SIDS, high-educated mothers (University degree) actually had slightly higher risk for SIDS than the other mothers (OR=0.82).

In the period with high incidence (1985-89) the risk was doubled in the low education group, and for the last years (1990-98) the risk was five fold elevated in this group.

In recent years we have seen an increase of pregnancies to mothers with a new partner. The risk for SIDS in infants for these mothers are likewise elevated with an OR=2.0 compared to women with same partner in both pregnancies. The likelihood of a new partner is higher in groups of women with low education.

These ‘new’ risk factors add to the factors we previously have reported, and all go in a direction of stronger socioeconomic mechanisms in the risk for SIDS.

Daltveit AK, Øyen N, Skjærven R, Irgens LM: The epidemic of SIDS in Norway 1967-93: changing effects of risk factors, Arch Dis Child, 1997, 77, 1, 23-27

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Speaking about the unspeakable

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When a big blow hits, it sometimes is difficult to find words, and if they are found to speak them.

In such sensitive moments the professionals are in a delicate situation; they must take part, but can easily contribute to making the difficulties become bigger.

One leading question can help the professionals in such situations;

WHO can speak with WHOM about WHAT issue in WHICH way WHEN?

Video-examples.

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Application of ESPID recommendations for the classification of SID cases a ten years' experience in Styria (Austria)

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Objective:
At the Second ESPID Congress held in Luebeck/Travemuende in 1992, a proposal for the classification of SID cases was established by ESPID members. Although this proposal has never been published as an official document, it was applied in our study since 1993 (R.Kerbl et al., Wien Klin Wochenschr 107/8, 237-241, 1995).

Method:
55 consecutive SID cases observed between 1993 and 2002 in Styria, the southeastern province of Austria, were analysed by a multidisciplinary team of health professionals. The study group consisted of pediatricians, forensic pathologists, pathologists, psychologists, nurses, members of the parents' association and health authorities.
SID cases were analysed with respect to potential risk factors during pregnancy and early life, the death scene and post-mortem findings. From the latter, every SID was classified either as 1) classical SIDS, 2) borderline SIDS, 3) non-autopsied SID or 4) explained death.

Results:
Of the 55 infant deaths primarily classified as "SIDS", 21 cases were assigned to category 1, 19 to category 2, four to category 3, and in 11 cases death could be explained by major post-mortem findings. For 15/21 cases of category 1 and 10/19 cases of category 2 the death scene investigation showed the typical "risk profile" of bedding and/or environmental conditions. In four cases, child abuse or infanticide was considered as possible, but could not be proven despite careful autopsy. In the last years, SID incidence in Styria decreased to a level of about 1/10,000 live born infants, and the few deaths still occurring mainly presented with the typical risk profile.

Conclusion:
Our results demonstrate that an extensive analysis of SID events is a prerequisite for reliable and comparable SID statistics. The ESPID recommendation of 1992 appears to be very useful for this purpose and the application of this classification may therefore be recommended.

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Neuropathology of nonaccidental head injury in early childhood. Differential diagnosis of shaken baby syndrome versus sudden infant death

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Objective:

Child abuse has to be seriously considered in cases of severe head trauma. The traumatic origin is not necessarily reflected in the neuropathology of the shaken baby syndrome, which is characterized by retinal, subdural and subarachnoid hemorrhages, and few or no signs of external craniofacial trauma.

Method design:

The study population comprised 17 cases of children with fatal head injury collected at the Institute of Legal Medicine of the Free University Berlin, in which the perpetrator had confessed or nonaccidental head injury had been established by conviction in a criminal court. Age ranged from 6 weeks to 48 months. The brains and spinal cords were investigated macro- and microscopically using immunocytochemistry as well as both silver and routine staining. We concentrated on the ten infants under one year to elucidate the differential diagnosis of shaken baby syndrome versus sudden infant death.

Results:

Nine of the ten infants showed no signs of skull bruising and were assumed to have only been shaken. The longest survival time was three weeks. Subdural and subarachnoid hemorrhages were thin and not space occupying. Epidural bleeding around the upper cervical cord in three cases indicated whiplash injury. Microglial activation, astroglial gliosis and diffuse axonal injury in the brain and spinal cord were observed in most cases. Severe global cerebral edema with hypoxic-ischemic encephalopathy was the most prominent feature in our study.

Conclusion:

Cervical cord and low brainstem injury, probably due to hyperextension, may lead to apnea preceding the hypoxic-ischemic changes seen at necropsy. Only microscopic examination demonstrated the high degree of diffuse axonal injury that enabled us to identify cases of shaken baby syndrome previously masked by the false diagnosis of sudden infant death syndrome.

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Alveolar maturation arrest in two siblings: An extreme and lethal form of pulmonary hypoplasia

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Alveolar maturation arrest is an extremely rare primary maldevelopment of lungs resulting in primary pulmonary hypoplasia. Up to now only a few cases have been reported and neither a certain underlying cause, nor a genetic etiology has been documented. We report two new cases of two term siblings presented with severe progressive respiratory distress shortly after birth, both delivered uneventfully after uncomplicated pregnancies with an interval of 2 years. The clinical courses were almost identical: progressive respiratory failure and pulmonary hypertension unresponsive to surfactant, nitric oxide treatments and conventional and high frequency oscillatory ventilation modalities, leading to death in a few weeks. Pulmonary biopsy revealed in both cases alveolar maturation arrest at a gestational age of 24 weeks, with normal bronchial and vascular structure of lungs (alveolar dysplasia). We speculate that a genetic predisposition may be responsible for this extremely rare lethal developmental defect.

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Do metabolic diseases play a role in SIDS?

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Background:

It has been suggested that a small percentage of sudden infant death syndrome (SIDS) cases could have an unsuspected inborn metabolic disorder. One of these might be mitochondrial myopathy. We have previously shown an increased number of substitutions in the D-loop of mitochondrial DNA in SIDS cases. Furthermore, we have shown that a significant proportion of the SIDS cases die after periods of hypoxia. Many SIDS victims are also more quiet and less active than controls. A disease, which affects muscles like the diaphragm, can result in reduced ventilation and eventually coma and death.

Objectives:

To examine whether muscle fibres in SIDS victims differ from those in controls.

Material and method:

Frozen sections of muscle from the diaphragm and thigh from 26 SIDS cases, 10 cases of infectious death and 7 cases of accidental death were examined with 12 different staining methods. The area of 50 fibres from the different specimens were also measured and compared.

Results:

There is a tendency towards more demonstrable, large motor endplates in thigh muscle in SIDS than in accidental deaths. This finding does not seem to correlate to age. There is no difference between SIDS cases and infectious deaths. The mean area of the muscle fibres in thigh muscle is greater in the cases of accidents than in cases of SIDS and infectious death. However, cases of accidental deaths are older than the two former groups and the area of the muscle fibre in the thigh muscle increases with age. The variation in fibre size is greater in the SIDS cases than in the two other groups, however not statistically significant.

Discussion:

The finding of large motor end plates especially in thigh muscle is suggestive of regeneration of muscle fibres. The significance of this finding is still uncertain. Among 38 cases that presented as sudden unexpected death there were two cases of primary metabolic myopathy. Without routinely examination of skeletal muscle, these cases would have been missed.

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Post-mortem radiography after unexpected death in infancy and early childhood

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Objective:

The aim of this study is to review the post-mortem radiographic examinations of children 3 years or younger in cases of unexpected death, and correlate to autopsy findings.

Method Design:

Consecutive post-mortem skeletal surveys were performed during a 5-year period (1998-2002). The radiographic examinations were undertaken before the autopsies (except in 1 case). Totally 123 cases were included.

Pediatric radiologists interpreted all the films, which routinely consisted of anterioposterior views of the appendicular and axial skeleton and thorax/abdomen, lateral views of the axial skeleton and thorax, and 2 oblique views of the ribs. A few examinations from other hospitals had only anterioposterior projections and in some additional CT-studies were included.

Imaging results were organised by pathologic findings and correlated to autopsy results.

Results:

Sixty-four cases were considered normal while 59 cases revealed pathological findings in the skeleton and soft tissue.

Conclusion:

Post-mortem radiography should be performed before autopsy to detect any skeletal and soft tissue finding that may help to distinguish cases of SIDS, borderline SIDS, natural, accidental and undetermined causes of death from those of child abuse.

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Sudden death in children, acute aspiration of gastric contents, and resuscitation attempts

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Objective:

Significance of massive gastric contents aspiration found in some sudden child death autopsies and its role in the cause of death is controversial among paediatric pathologists, but they usually deny it as a cause of death. Several explanations for such aspiration are put forward: agonal event, migration by gravity from the stomach into the pharynx and then down the airways during body's movements, or forcing of gastric material in airways by attempts at resuscitation.

Material and Methods:

We reviewed the results of 125 sudden and unexpected deaths in infants (n = 115) and children (n = 10) investigated in our centre between Nov. 94 and Dec. 2002. They were sub-divided in 2 groups according to attempts at resuscitation

(n = 90) or not (n = 35) when the body was found. Acute massive aspiration was defined as presence within both lungs of gastric material within over 2/3 of the bronchioles and flooding of gastric material within the alveoli associated with oedema and haemorrhage (vital signs). When gastric material was found only in trachea and/or large bronchi, or in a small number of bronchioles, the case was classified within the "no aspiration" group.

Results:

Resuscitation attempts: Yes (n = 90) No (n = 35)

No aspiration	**(n = 87)**	**n = 63**	**n = 24**
Massive aspiration	**(n = 38)**	**n = 27 (30%)**	**n = 11 (31.4%)**

Conclusion:

Near the same proportion of vital aspiration cases was found in both groups "with" and "without" attempts at resuscitation. Thus, we conclude that massive gastric contents aspiration is not linked to attempts at resuscitation.

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Detection of viral genome in myocardial samples from SIDS victims and children with unnatural deaths

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Introduction:

Epidemiological aspects and recent investigations indicate an inflammatory process in cases of suspected sudden infant death syndrome. Virus-induced myocardial inflammation can explain sudden death but still there are no studies detecting viral genome within the myocardium with molecular pathological techniques (RT-PCR, PCR) while immunohistochemical methods have improved the diagnosis of myocarditis.

Methods:

Eight myocardial samples were taken from each of 65 SIDS autopsy cases. Control groups: 11 cases aged 1 to 11 m (control group I) and 25 cases 1 to 10 y (control group II) from children with sudden unnatural death (homicides, accidents). Molecular pathological techniques were performed: PCR for PVB19, Epstein-Barr-Virus (EVB), adenoviruses (AV) and semi-nested reverse transcription-polymerase chain reaction (RT-PCR) for enteroviruses (EV) including CVB3 to investigate myocardial samples and those from liver and spleen. Additionally immunohistochemical methods with antibodies against infiltrating leucocytes (LCA), macrophages (CD68), T-lymphocytes (CD3) and

the MHC-class-II molecules were applied as well as antibodies against VP1-capsid protein of enteroviruses.

Results:

Viral genome was found in the myocardial samples from SIDS-victims: 16xEV (5xCVB3), 2xAV, 3xEBV, 7x PVB19 and 1xHHSV-6. There was not one virus-positive myocardial sample in control-group I and only 3 PVB19-positive cases in control-group II. In part immunohistochemical findings point to a virus-induced myocarditis while conventional histology according to the Dallas-criteria was without significant pathological findings.

Conclusion:

Myocardial samples from suspected SIDS-cases without any pathological findings using conventional stainings should be investigated with molecular pathological techniques to detect viral genome, especially including EV, EBV, AV and also influenza a+b as well as cytomegalovirus. Additional immunohistochemical stainings together with molecular pathological detection of cardio tropic viruses can point to virus-induced myocarditis as probable cause of sudden death of natural cause in infants.

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Chemo receptor responses in infants with apnea of infancy

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Objective:

The central and peripheral chemoreceptors play a crucial role in the control of breathing and in the defence against hypoxia and hypercapnia during sleep. Contradicting findings in earlier studies suggested the poor sensitivity to O2 and CO2 as factor contributing to sleep apneas in very preterm born infants and in adults. Objective: to study whether a deteriorated O2/CO2 sensitivity is associated with apnea of infancy (AOI) in full term infants.

Methods:

Seventy- infants: 17 healthy controls and 53 with AOI (26 with periodic breathing, PB and 27 with recurrent apnoeas, RA lasting >6 sec.) were recruited. Tests were performed during deep sleep as a part of polysomnographic over-night recordings. "Ventilation" was calculated as the product of the amplitude and the frequency of respiratory movements recorded with inductance plethysmography. PCO2 and PO2 were monitored by transcutaneous (Tc) electrodes. The one-minute "ventilation" that directly preceded the CO2/O2 test was used as the control condition. The "ventilatory" response to 4 – 6% CO2 in air during 6-8 minutes, was expressed as the slope of the relationships between the change in "ventilation", expressed in percent of the control "ventilation" and the increase in TcPCO2 on the breath-by-breath basis. The response to O2 (100% O2 for 60 sec) was estimated as mean value of the four lowest consecutive breaths expressed in percent of the control "ventilation". The response time was calculated as the number of breaths from initiation of the CO2/O2 breathing until appearance of significant changes in "ventilation" in four consecutive breaths.

Results:

The response to CO2 was similar in infants with PUB and RA (104% and 193% increase in ventilation/kappa TcCO2 respectively) and was significantly lower than in the control group (311% change in "ventilation"/ kappa, p=0.009). No such difference was found in the response to O2 (p=0.26). However, the response time to both CO2 and O2 was significantly longer in infants with AOI than in the control group (CO2 test 16.1 and 9.1 breaths respectively, p=0.02; O2 test: 13 and 3.5 respectively, p=0.002). The strength of the ventilatory response to CO2 was negatively correlated to the response time ($p<0.0001$).

Conclusion:

The weaker and delayed response to CO2 and prolonged reaction time to changes in O2 in infants with AOI point at a defective chemoception as a contributing factor to AOI.

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Arousals and cerebral blood volume in preterm infants

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Objective:

The aim of the study was to evaluate whether arousals in preterm infants are accompanied by a change in cerebral blood volume.

Method:

Polygraphic recordings were performed in 7 stable preterm infants with a mean postconceptional age of 34.4 ±1.2 weeks. 52 arousals were scored according to the guidelines of the Paediatric wake-up Club (Bruxelles 2002). Changes in cerebral blood volume (CBV) were measured by near infrared spectroscopy. Values of interest were assessed during the arousal period and 30 seconds before and thereafter. These periods were subdivided into 10-second intervals and compared to the arousal period.

Results:

The mean duration of arousal was 7 ±3 seconds. Respiratory rate was higher (35 ±15 bpm) during the arousal when compared to the period preceding (29 ±14 bpm; $p < 0.001$) and following (29 ±11 bpm; $p < 0.001$) the arousal. Heart rate decreased during the arousal (131 ±30 bpm) when compared to the 10 second period preceding (140 ±14 bpm; $p < 0.05$). Cerebral blood volume slightly decreased during the arousal when compared to the period preceding (-0.05 ±0.24 ml/100g brain) and to the period following (-0.05 ±0.25 ml/100g brain) the arousal.

Conclusion:

Arousals in preterm infants are associated with a slight decrease in cerebral blood volume. The mechanisms leading to reduced cerebral blood volume during arousals remain to be clarified.

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Breast fed infants are more easily aroused from active sleep

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Objective:

Although all medical research has demonstrated that breastfeeding is best for infant health, there is no consistent evidence that it reduces the risk for Sudden Infant Death Syndrome (SIDS). Arousal from sleep is believed to be an important survival mechanism that may be impaired in victims of SIDS. Previously others and we have shown that arousability from sleep is impaired by the major risk factors for SIDS. In this study we examined whether arousal responses from sleep were altered by method of feeding, and hypothesised that breastfed infants would have lower arousal thresholds.

Method Design:

Forty-three infants (24f/19m) were studied between September 1997 and October 2001. All infants were born at term between 38-42 weeks gestation (39.8±0.2 weeks; mean±sem), with normal birth weights, mean 3534±63g, (range 2700-4725g). Apgar scores were 3-10 (median 9) at 1 min and 6-10 (median 9) at 5 min. Infants were studied on 3 occasions: Study 1 (n=42): 2-4 weeks after birth, mean age 18±1d, (range 8-32d); Study 2 (n=39): 2-3 months post-term, mean age 73±1d (range 63-84d) and Study 3 (n=36): 5-6 months post-term, mean age 173±2d, (range 155-213d). All infants were studied using daytime polysomnography on 3 occasions: 2-3 weeks post-term, 2-3 months post-term, and 5-6 months post-term. Multiple measurements of arousal threshold (cm H2O) in response to air-jet stimulation applied alternately to the nares were made in both active sleep (AS) and quiet sleep (QS) whilst infants slept supine. Arousal thresholds and sleep epoch lengths were compared between formula fed and breast fed infants at each age with a student's t-test. Data are presented as mean ± sem and were considered statistically significant at the $p<0.05$ level.

Results:

There was no significant difference between the two groups in either infant age or weight at any of the three ages studied. Arousal thresholds were not different between breast-fed and formula fed infants in QS at any of the three ages studied. However, in AS breast fed infants were significantly more arousable (i.e. had lower arousal thresholds) than formula fed infants at 2-3 months of age ($p<0.01$). Arousal thresholds were significantly elevated in QS compared to AS in both groups of infants at all three ages studied ($p<0.001$), with the exception of the formula fed group at 2-4 weeks of age. When infants were grouped into male and female there was no significant difference in arousability in either sleep state at any of the three ages studied. There was no difference between groups of infants when sleep epoch length was compared at any study. However, at 5-6 months of age breast fed infants had significantly longer sleep cycles ($p<0.01$).

Conclusions:

We have demonstrated that breast fed infants are more easily aroused from AS at 2-3 months of age than formula fed infants. This age coincides with the peak incidence of SIDS. Differences in sleeping patterns between the two groups could not explain these findings. This study supports breast-feeding of infants during the critical risk period for SIDS (2-4 months) as reduced arousal particularly in AS could impair the ability of an infant to respond appropriately to a life threatening situation.

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Physiological relationship between autonomic reactions and arousals in infancy

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Introduction:

Future SIDS victims showed both a dysregulation of cardiac autonomic controls and fewer cortical arousals. Environmental risk factors for SIDS, such as sleeping prone, prenatal smoking or high environmental temperature enhance a deficiency in autonomic and arousal controls.

Objective:

Changes in blood pressure (BP) were measured following auditory stimuli in the prone and the supine position to study the correlation between arousal from sleep and autonomic responses.

Method:

Two newborns born at term, 8 infants and 4 children were recorded polygraphically during one night, while sleeping in the prone and the supine position. They were exposed to white noises of increasing intensities during both REM and NREM sleep in each position. BP changes were measured by Finapress in the children and by Pulse Transient Time (PTT) in the infants and newborns.

Results:

Basal systolic BP pressures were lower ($p < .001$) and PTT were higher ($p = .012$) in the prone than in the supine position in children and in infants. Following the auditory stimulations, the increases in systolic BP (p: .024) and the decreases in PTT ($p < .001$) were smaller in prone than in supine position. During cortical arousals, the same findings were found, independently of sleep stages.

Conclusion:

Compared when they are sleeping supine, children and infants sleeping prone had lower basal BP and higher PTT and smaller changes in BP and PTT after auditory stimulation. Reduced blood-pressure changes to stimuli could be implicated in the increased arousal thresholds in prone position.

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Effects of sleep position and sleep state on postnatal development of heart rate responses at arousal

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Objective:

Maturational factors have been implicated in SIDS aetiology, suggesting that alterations in autonomic nervous system function may be involved. Our aim was to characterise the maturation of cardiovascular autonomic control by examining heart rate responses to an arousing stimulus in healthy term infants at 2-3wk, 2-3mo and 5-6mo postnatal age.

Method Design:

24 infants (15F/9M), born at term (38-42wks) with normal birth weights (mean 3540±76g, range 2765-4109g) were studied on 3 occasions: 2-3 wk (mean 14±1d, range 8-19d), 2-3mo (mean 75±1d, range 63-84d) and 5-6mo (mean 182±4d, range 155-213d). All infants were studied in the Melbourne Children's Sleep Unit, with daytime polysomnography. Air-jet stimulation delivered alternately to the nostrils of infants was used to induce arousal in both active sleep (AS) and quiet sleep (QS). Heart rate (HR), EEG, submental EMG, respiratory movements, oxygen saturation, and visual observations of behavioural pattern were recorded. Infants slept both prone and supine at each study; the initial position was randomised. Baseline heart rate (BHR) data were collected over 20 beats prior to each stimulus presentation that induced arousal. Data for 30 beats after stimulus onset were analysed for maximum heart rate (MaxHR). MaxHR was the average of 3 beats prior to and three beats after peak heart rate. The change in heart rate (DHR%) at arousal was calculated as the difference between MaxHR and BHR normalized for BHR. Comparisons between sleep states; sleep positions and postnatal age were made with 2-way ANOVA for repeated measures. 11 infants in AS and 15 infants in QS had complete data sets which could be analysed for maturational effects. Results are expressed as mean ± SEM and significance is at the $p<0.05$ level.

Results:

Heart rate responses to arousing stimuli (DHR%) were significantly elevated in the supine compared to the prone position in AS at both 2-3mo ($p<0.01$) and 5-6mo ($p<0.05$); this failed to reach significance at 2-3 wks. In QS, DHR% was only significantly affected by position at 2-3wk ($p<0.05$). DHR% was significantly elevated in AS compared to QS in both supine ($p<0.05$) and prone ($p<0.001$) positions at 2-3wk. At 2-3mo the difference between sleep states was only apparent when infants slept supine ($p<0.001$). DHR% was significantly increased from 2-3wk to 2-3mo in QS when infants slept prone ($p<0.05$). Arousal thresholds were elevated in the prone position compared to the supine position at 2-3 wk and 2-3 mo of age ($p<0.001$). DHR% was not correlated with arousal threshold in either AS or QS in either position at 2-3 wk. At 2-3 mo DHR% was negatively correlated with arousal threshold in AS when infants slept prone and at 5-6 mo this became a positive association.

Conclusion:

The finding that changes in HR at arousal were most affected by sleep position in AS across all ages suggests that autonomic control of HR is not impaired in this sleep state. However, our findings that in QS, HR increases at arousal were only affected by sleeping position at 2-3wk of age when incidence of SIDS is low may be of significance of SIDS.

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Skin conductance activity in infants born by smoking and non-smoking mothers during the first 10 weeks of life

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Objectives:

Skin conductance activity (SCA) reflects the activity in the sympathetic nervous system, associated to arousal, which innervates glands located in the palmar side of the hand and the plantar side of

the foot. Increased CNS sympathetic activation, gives an increased firing in the postganglionic cholinergic fibers, which in turn increases the number and amplitude of the waves, as well as the basal level. The basal level, the number of waves with high amplitude, the number of waves per second, as well as the amplitude of the waves all increased during the first 10 weeks of life.

Smoking may alter the development of the sympathetic nervous system, associated to arousal. 10 healthy infants born to term by non-smoking mothers, as well as 10 healthy infants born to term by smoking mothers were measured during their first and third day of life and after 3 and 10 weeks of life. During the spontaneous measurements the basal level, the number of waves per second, as well as the amplitude of the waves were measured in a prone and supine posision. During the stimulated measurements, the percentage that respond, the amplitude of the response as well as any habituation pattern was recorded.

Results:

During the spontaneous measurements the number of waves per second, recorded in a supine position increased in both groups of infants, but a significant delay were demonstrated in the group of infants where the mothers were smoking ($p<0,02$) The basal level and the amplitude of the waves all increased, but no difference between the two groups were detected. ($p<0,45$) ($p<0,71$) During the stimulated measurements the percentage that responded as well as the amplitude all increased. But no differences between the two groups were detected. ($p<0,74$) ($p<0,52$)

Conclusion:

In conclusion the different development rate in the number of waves per second may indicate that the sympathetic nervous system, associated to arousal, may be effected by the mother smoking habits.

Information regarding the mothers smoking habits were obtained solely by questioning the mother, and may be incorrect and thereby may affected our results. A new project, where we measured nicotine in the infant's urine sample during the first day of life may confirm whether the mother has been smoking or not.

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Mother's need for support and confirmation when loosing an infant

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Objectives:

To study the support received and needed by mothers when they have lost an infant and what bring appeasement after the loss.

Method design:

The target group was members of an association in Sweden for parents who had lost a child before, during or after labour. Only the mothers were addressed. A questionnaire was constructed, both with structured and open-ended questions. Altogether, 164 subjects (77 %) responded. A mixed method, triangulation, was used in analysing.

Results and Conclusion:

Preliminary analysis gave the following trends. A miscarriage, stillborn or the death of a baby is often not acknowledged by society as a real loss. Still mothers stressed the importance of the recognition of the dead child as a family member, and being able to talk about their child, even though bringing up the subject of a dead child often provoke uneasiness.

Bereaved mothers spoke about the importance of recognising death as a part of life, no matter how young the life might be, illustrating integrative and transformational grief. Mothers seek significance in coping through the mechanisms of conversation and transformation. For recovery it was valuable to have a chance to communicate the experience through talking and writing. The respondents also expressed a need to keep the memory of the dead child through various symbols, or simple by lighting a candle. Photos, hand- and footprints from the child were also signifying a great deal.

Health care professionals have powerful, wise information and empathy to offer, but some women experienced social isolation and did not receive the support they needed from partners, relatives, friends or health professionals. The grief mothers suffer must be recognised by acknowledging the mother's loss of a special precious child and her need to grieve, and the potentially life-long effect the loss may have.

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Grief in a cultural context: Coping strategies after sudden death in Norway versus Tigray, Ethiopia

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Objective:
The paper explores socio-cultural influences on coping-strategies after sudden death, by comparing data collected from samples in two different cultural settings.

Method Design:
Twenty Norwegian families bereaved by SIDS or their children's accidents, and 14 Tigrinian-Ethiopian families bereaved by war events, were in in-depth interviewed about their post-loss coping strategies.

Results:
The material reveals fundamental differences in descriptions of post-loss coping-strategies between the two samples. Norwegian informants more frequently describe strategies referring to openness and emotional ventilation, while Tigrinian-Ethiopian informants more frequently describe strategies referring to repression and emotional control.

Conclusion:
The differences can be understood in terms of ethno-psychology, in the sense that they reflect different dominant cultural beliefs and norms in the two respective contexts. The paper argues for a highly context-based understanding of peoples' needs after a sudden death episode.

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Micro-sociological analysis of social support following traumatic bereavement: Unhelpful and avoidant responses from the community

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This article explores why parts of the social support to survivors of traumatic deaths fail. The data is based on in-depth interviews with 69 parents who lost their child by suicide, SIDS or accident between 1.07.97-31.12.98. Despite considerable positive support from the social network, an overall majority of the survivors also experienced unhelpful encounters or lack of anticipated support. The difficulties for network members to communicate support in a way that is helpful, is termed social ineptitude. The ineptitude of networks is explained by lack of norms to guide this infrequent encounter. The bereaved parents claim that openness and frankness about their situation is their best empowering strategy to meet this social ineptitude. These concepts, elaborated from survivors accounts are discussed within a communicational and interactional perspective. A micro-sociologic model of communication is the frame of reference for the discussion.

Key words: unhelpful social support, survivors of traumatic deaths, social ineptitude, openness, and communicative competence.

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Help to grieve - a description of a parent-support-group in Sweden

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In 1995 it was not widely acknowledged that perinatal death and infant death was difficult for parents to cope with and often required guidance. Bereaved parents though, faced the death of their baby as a highly stressful life event that had serious consequences both in the short and long-term perspective. The parents were often placed in a position of having to justify their feelings in the face of denial and silence from friends, families, their workplaces, and the medical community.

A group of parents who recently had lost their infants felt that they wanted to support each other and met to talk about their losses. In 1997, a non-profit-making association was established, the "Parental Association of Little Angels". Today the association has about 350 members all over Sweden. About 260 of the members have lost one or more infants, the other members are "support members", grandparents to a dead child, or health care professionals.

The association's objectives are:

- to support families who have lost a child before, during or after delivery
- to share experiences with others and make suggestions for how to assist and support parents in need
- to distribute information about the association and what it means to loose a child
- to distribute a newsletter four times a year
- to cooperate with hospitals and health care professionals

The association helps bereaved parents to get into contact with another parent that has been in the same situation. Groups of grieving parents are organised locally and meet regularly. These meetings offer parents a forum where they can find acceptance and validation of their feelings.

The strength of the association lies in the personal experiences of the members, which give them the needed insight to help bereaved parents and to work for a better support system from society.

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Training volunteers to take the FSID Helpline from home

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Objectives:
To look at the training needs of 28 volunteers who since August 2002 have taken over the FSID Helpline from 6pm - 11pm every night of the year. The volunteers divert the telephone line from FSID head office to their own home telephone line and each volunteer is on call one night every four weeks. The FSID helpline receive calls from bereaved families, professionals working with bereaved families and parents and professionals who need infant care advice.

Method Design:
The volunteers have been selected from FSID Befrienders who have completed the Befriender Preparation training and regularly attend Befriender meetings. This will have ensured that they have a fundamental knowledge of the procedures following a sudden and unexpected infant death, an understanding of the grief emotions experienced and knowledge of the information available.
An initial package of information has been developed which includes:

- Guidelines to cover diverting the telephone line, examples of different types of calls relating to bereavement and to infant care advice, informa tion relating to different callers, and confidentiality of the calls.
- Six-month rota with volunteers' names and telephone numbers
- Copies of press releases and special statements giving FSID position on various issues
- Personal support for volunteers

Training meetings are organised to discuss specific issues for taking the calls in isolation and in the volunteer's own home and to focus on specific calls.

Results and Conclusions:
Six months after the scheme was started the first training meeting has identified several needs when home-based volunteers are used for the helpline.

- How to maintain an efficient way of keeping 28 volunteers up to date with information
- How to continue to support volunteers effectively
- How volunteers can feel that they have answered the call in the most supportive and appropriate way.

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Problems on sudden infant death (SID) cases in Japan

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We cite three points of problems with SID cases in Japan. They are diagnostic problems, troubles with SID cases, and problems on risk factors and prevention.

1) Diagnostic problems
Japanese SIDS Research Society issued diagnostic criteria for SID cases in 2000. SID is classified into five types in these criteria:

Type I; SIDS (typical SIDS),
Type II; possible SIDS,
Type III; endogenous death with obvious findings,
Type IV; exogenous death,
Type V; unexplained cause of death or not classifiable.

There are obscure points of diagnosing, and it leads to increase in number that classified into type V.

The idea that deal with SIDS cases divide into two groups in Japan. One is predominantly comprised of neonatologists and anaesthesiologists, who regard most of SID as SIDS, that is endogenous death resulted from abnormality of respiratory system. Another side is predominantly comprised of forensic pathologists and emergency medical doctors, who regard most of SID as exogenous death such as asphyxia or abuse. This situation results from the fact that they cannot achieve a consensus of opinion about autopsy findings and circumstances. The Autopsy rate increased from 43.9% to 75.3% in Osaka pref. from 1990 to 2001. But it is still low where administrative autopsy is conducted.

2) Troubles with SID cases
a) SIDS or Asphyxia? ; About 40 SID cases that occurred in hospitals or nurseries are going to court now. Most of the bereaved family advocates that the cause of death is asphyxia, and the other protests that it is SIDS.
b) SIDS or Abuse? ; The number of child abuse case is enormously increasing, recently. We experienced a case that turned from possible SIDS to victim case in a nursery, last year. The case was reported seriously all over the nation.

3) Problems on risk factors and prevention
Most of Japanese babies used to be laid on its back historically. Japanese Health, Labour and Welfare Ministry finally recognized the correlation between SIDS and prone sleep, and SIDS prevention campaign was carried out in 1998. The campaign was focused on three points. They are back to the sleep, no smoking around infants, and try the breast-feeding. Japanese government announce the number of infants who usually sleep on prone position decrease markedly. But we recognize the infants who usually sleep on prone position are still predominant in nursery, now. We should consider how to treat the baby who sleeps only on prone position, and who cannot be breast-fed.

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Problems around the world: France

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A governmental circular of the French Ministry of Health was published in 1986 to promote and facilitate investigations of sudden unexpected death in infants (SUDI). Indeed at that time only 30% of SUDI were investigated because of the current SIDS definition (no need of an autopsy because SIDS means nothing to find).

This circular instituted 30 hospital « referral centers » and imposed the costs of body's transport and autopsy to be supported by the hospital. This allowed the autopsies' number to increase over 50%.

Investigations of SUDI in France are made through 2 different ways according to immediate suspicion of child abuse (forensic way) or not (medical way):

- Forensic way: suspicion based upon circumstances, previous history and/or clinical exam. Advantage = no need for parents' agreement. Limitations are due to the French organization: very few forensic pathologists specialized in pediatrics; forensic pathologists work alone without any multidisciplinary approach (=> loss of data); knowing if the death is of « natural » or « non natural » cause is the only thing the Justice is interested in and there is no money to further investigate the « natural » causes of death.

- Medical way: this route is a privileged one for reaching a diagnosis. Advantages: multidisciplinary approach; use of all the hospital technologies; costs supported by the hospital; allows research and long-term follow-up of parents. Limitations: needs both parents' agreement; the death certificate is signed right after death and before autopsy and is not corrected according to the autopsy's findings. Thus, all SUDI investigated in the medical way are recorded as SIDS while there may be a diagnosis after PM.

Ideally, there should be a tight cooperation between forensic investigators and pediatricians to reach a diagnosis for parents' benefit. Actually, communication between these 2 ways is difficult.

Thus, when no obvious violence sign is present, everything is done to keep the case in the medical way.

Therefore we have been asking for years a change of the definition*:

1- Keep the term SIDS for parents and administrative purposes = « clinical SIDS »

2- Add the PM findings for medical and research purposes = « SIDS with » e.g. myocarditis, disseminated bacterial infection.

*Rambaud C, Guilleminault C, Campbell PE. Definition of the sudden infant death syndrome.
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Is the SIDS approach changing in Latin American countries?

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Introduction: SIDS is not considered a health problem in Latin America because infant mortality by other causes is still very high (see table). Many countries in the world, in particular those countries of the Latin region, have low levels of awareness of SIDS and the strategies for its prevention. SIDS incidence is unknown in Latin America and in most of the countries of the region SIDS is not included in the official rates of infant mortality (1). I am fully persuaded that the prevention interventions addressing SIDS risk factors can also produce health benefits far beyond the prevention of SIDS.

Latin America: the representatives of SIDS International, The SIDS Global Strategy Task Force and Who proposed a survey with the aim to determine the prevalence of different sleeping positions and other risk factors for SIDS and to identify the advice given to parents in several maternity units in Latin America and the Caribbean through a specially-designed questionnaire. Two hundred thirteen hospitals from 16 different countries answered the survey (2). The preferred sleeping position for healthy infants was prone in 8.5% of the hospitals, side in 48.8% and supine in 25.2%. In 17.5 % of the hospitals no position was preferred or the answers were inconsistent. Concerning the recommendations given to parents on the best position for their infants at home, 25.7% recommended the supine position, 7.6% the prone position and 59% the side position. Recommendations to avoid prone position have been endorsed by the Latin American countries during the Simposio Latino Americano in Italy in 2002 and published in the side page of the Asociación Latinoamericana de Pediatría (www.alape.org), which included 26 countries of the region. Infant mortality rates show the effects of socio –economic inequalities regarding the population ´s health.

Infant Mortality

South America

Country:	Population*:	Mortality**:
Argentina	37.488,0	17.6
Bolivia	8.705,0	67.0
Brazil	172.236,0	33.1
Chile	15.402,0	10.1
Colombia	43.074,0	13.5
Ecuador	12.880,0	30.0
Peru	26.093,0	37.0
Uruguay	3.361,0	14.1
Paraguay	5.636,0	19.4
Venezuela	24.632,0	19.2
Suriname	419,0	16.9
French Guayana	153,0	20.0

The Caribbean region

Cuba	11.239,0	7.2
Haiti	8.359,0	80.3
Puerto Rico	3.899,0	10.6

Central America

El Salvador	6.390,0	35.0
Guatemala	11.687,0	49.0
Honduras	6.656,0	42.0
Nicaragua	5.215,0	45.2
Panama	2.899,0	16.8

Source: Pan American Health Organization (2.000)
* Thousands / ** Number per 1000 birth.

Argentina:
The International Child Care Practices Study (ICCPS) collected information on childcare practices associated with SIDS risk from 21 centres in 17 countries. One participating centre (Buenos Aires, Argentina) noted a relatively high rate of prone sleeping in the study population together with a significant incidence of SIDS in the community (3). With WHO regional office support, a successful "Link" programme between New Zealand and Argentina was launched to raise SIDS awareness and advice "boca arriba" (face up)(4). The "Tarjeta Cuna" (cot card) has been one of the several strategies implemented. This card carries key messages for SIDS prevention and infant health about safe sleeping positions, breastfeeding and non-smoking habits. The cot cards have been displayed on every newborn cot in Maternity Hospitals throughout Argentina. The cot cards are of international significance because they are a low-cost intervention promoting excellent education

practice with extensive outreach. Through discussion between families and staff, trust in new information can be supported and the importance of established information strengthened. A sharp decline in the use of the prone position was observed in a prospective study that we have conducted to assess secular trends in use of prone infant sleep position from 1997 through 2001 among infants attending for vaccination at the Hospital Italiano de Buenos Aires (5). Argentina has not got a national autopsy law regulation concerning the sudden unexpected death victims yet; therefore the Argentine annual SIDS rate is questionable.

Uruguay:
Ivon Rubio carried out a prospective study of SUDI in Montevideo, Uruguay. In all cases of SUDI a thorough autopsy was performed: including total body X-ray, toxicological screening, the death scene investigation and a case conference with the experts (health system and judicial system). The study included 268 cases. 60 % of the cases had an accurate diagnosis, 21 % were diagnosed SIDS and 19 % entered in the grey zone (6).

Conclusions:
Our education challenge is to improved protection from SIDS for all children of the world. It is possible to make significant improvements in children health not only by promoting economic growth but also by reducing income gaps within country.

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SIDS – problems in Australia

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Great advances have been made in Australia in reducing the death rate from SIDS due to the 'Reduce the Risks' campaign. SIDS deaths have fallen from over 500 annually in 1988 to less than 150 in recent years. This makes accurate investigation of the remaining infants even more imperative to determine why the remaining infants are dying despite the generally reduced death rate. Unfortunately, when we examine the investigation of infant deaths in Australia we find that there are significant problems.

In rural areas of Northern Australia cases of unexpected infant deaths have been designated as SIDS deaths without autopsies, radiographs, toxicology screening or death scene examinations. Diagnoses of SIDS have been accepted in recent years in both Australia and New Zealand based on autopsies that were not performed by pathologists.

Even in larger centres there are inconsistent practices, with pathologists using different definitions and criteria. For example, the unexplained death during sleep of a previously healthy 2-year-old toddler would be deemed 'undetermined' in one Australian state, and 'SIDS' if the death occurred 5 kilometres away over the border into another state. Cases of accidental asphyxia from wedging have also been classified as 'SIDS for parents'. Perusal of databases reveals cases of 'SIDS' with facial petechiae.

There are many reasons why we need a consistent approach to this problem. Parents are receiving different diagnoses based on location, not findings.

Researchers are publishing results based on cases that do not fulfil the basic criteria for SIDS. Epidemiological data may be skewed by the higher percentage of non-SIDS cases that are now found (25% in one recent Australian study). Unless a consistent approach is established to these issues national data on SIDS deaths in Australia will have to be treated circumspectly.

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The continuing challenge of SIDS in the United States

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The scourge of SIDS continues to challenge not only the United States of America (USA), but also the entire world as exemplified by this panel of international speakers. Given the size and diversity of the USA, these challenges vary from region to region, however, many are shared with other countries.

Before delineating these challenges, it is important to recognize important successes in the struggle against SIDS. Our Back to Sleep campaign that began in 1994 and was patterned completely after earlier and highly successful public education campaigns in Australia, New Zealand, and Western Europe has resulted in similar reductions of the SIDS incidence from approximately 1.5 per 1000 live births to approximately 0.7 deaths per 1000 live births. Use of standardized protocols in investigation of death scenes1 and post-mortem examinations2 has produced data for productive research ranging from the safety of sleep environments3-5 to attempts at explaining mechanisms of death6, 7 to molecular neurobiology that may enhance vulnerability to SIDS.8, 9

The struggle is not over. Infants in the USA are still dying of SIDS. Two recent studies make clear that the Back to Sleep campaign has not reached all sectors of American society. SIDS rates remain higher among infants who are African American, reside in Mid-Atlantic States, are less than 8 weeks of age, and have mothers with previously born children.10 A subsequent study by this group revealed a decline in prone placement for sleep from 44% to 17% among whites compared to 53% to 32% for African Americans between 1994 and 1998.11 Eighty six percent of caregivers placing infants prone received the opposite recommendation from only one source. In contrast, recommendations from multiple sources increased the probability for supine sleep position (OR, 6.01 [95% CI, 4.57-7.90]).

What remains to be done? The Back to Sleep campaign must be modified and intensified to reach all segments of the diverse American population. In California, brochures that explain safe sleeping position and environments for infants are now produced in greater than 10 languages. But that is not enough in a state with greater than 140 languages! Health care providers must be ever diligent in educating new parents about infant care that reduces the risk of SIDS. Physicians are the most effective in getting the Back to Sleep message across to new parents.11

Efforts to achieve universal use of the standardized death scene and autopsy protocols must continue. They help ensure comprehensive evaluations and provide the data that allows careful case analysis for diagnosis as well as effective reviews by consulting physicians when requested. The data in the completed protocols also provides research opportunities including clarification of the role of risk factors, the possible contributions of infections and immunological responses, and the identification of subtle pathologic abnormalities that render infants vulnerable to the cataclysmic event known as SIDS.

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Modifiable risk factors for SIDS in Germany

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The incidence of Sudden Infant Death Syndrome has been falling in Germany over the last decade. However, little is known about the prevalence and the importance of well-known risk factors in Germany since the prevention campaign in the early 90s.

Design

A three year, population based, case control study was conducted in half of Germany, consisting of 373 cases. All sudden and unexpected deaths in infancy, if they fitted the inclusion criteria, were included in the study. Parental interview was carried out soon after the death (mean: 39 days) and 3 living control infants, matched for age, gender, region and sleep time were recruited. Major risk factors for SIDS in Germany are presented in this paper.

Results

The risk for SIDS in Germany increases when the infant is put down prone for sleeping (OR: 12.5), maternal smoking during pregnancy increases the risk (OR: 3.6), exclusively bottle feeding (OR: 2.8), co-sleeping with an adult (OR: 1.2) and young maternal age (OR: 8.1). Using a pacifier during the last sleep is protective (OR: 0.4) in our analysis.

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Combined effects of prone position and tobacco exposure on arousals in infants

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Objective:

To evaluate the combined effects of body position and tobacco exposure on auditory arousal thresholds in healthy infants.

Method:

Thirty-two infants with a median age of 9 weeks were studied with polygraphic recordings for 1 night: 16 were born to mothers who did not smoke and 16 were born to mothers who smoked (> 9 cigarettes per day) and continued to smoke after birth. The infants were exposed in REM sleep to white noises of increasing intensities while sleeping successively in the prone and supine positions, or vice versa. Arousal thresholds were defined by the auditory stimuli needed to induce polygraphic cortical arousals. Cortical arousal was scored in the presence of EEG changes, accompanied by presence of body movement and changes in heart rate.

Results:

More intense auditory stimuli were needed to induce arousals in infants of smokers than in infants of non-smokers in supine position ($p < 0.001$) and in prone position ($p < 0.01$). Auditory arousal thresholds were higher in infants sleeping in the prone position than those in the supine body position in both groups of infants respectively for non-smokers ($p = 0.02$) and for smokers ($p < 0.01$).

Conclusion:

Tobacco smoking could potentate the effects of prone position on arousals from sleep.

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Could swaddling decrease cot death? Facts and findings on swaddling

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Swaddling is a worldwide used infant care practice. One fifth of the children around the world are swaddled in their first months of life. The sleeping position is always supine when swaddled. This presentation comprises a historical review, physiologic findings and recent Dutch data on swaddling.

In the Netherlands from 1996 to 1999 about 8 percent of all infants less than 9 months old are still placed to sleep prone. Half of this group is described as restless and/or crying excessively. It is well known that some infants sleep poorly when placed supine. Recently Thach et al. demonstrated that swaddling has a significant inhibitory effect on arousal. Swaddling seems to promote sleep and thus might prevent caregivers placing infants prone.

In a randomised controlled trial (RCT) "The efficacy of swaddling in infants who cry excessively" swaddling is compared to offering regularity and stimulus reduction in 400 infants. Preliminary data of this study will be presented.

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A comparison of efficacy between two natural exogenous surfactant preparations in premature infants with respiratory distress syndrome

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Objective:

Respiratory distress syndrome (RDS) is a major cause of mortality and morbidity due to lung immaturity associated with surfactant synthesis deficiency. The mortality and various morbidity rates have been substantially reduced by means of exogenous surfactant replacement, the cornerstone in the treatment of RDS. Although natural preparations have been determined to be superior to synthetic surfactant preparations in terms of effectiveness and side effects, there are not many studies investigating the clinical responses among various natural surfactant preparations.

The objective of this study is to compare two natural surfactant preparations (Alveofact, Survanta) in terms of effectiveness and side effects. A total of 50 infants with RDS were given surfactant due to RDS were taken into the scope of this study.

Results:

Survanta and Alveofact were administered to randomised infants with RDS and the results obtained during clinical observations were compared. Second hour mean FiO2, MAP and a/APO2 values showed changes in favour of Alveofact (n=25) group compared to the Survanta (n=25) group ($p<0.05$ for each parameter). However, this difference disappeared in the 6th hour. No statistical difference was established between the two groups with regard to side-effects (pneumothorax, sepsis, intraventricular hemorrhage, bronchopulmonary dysplasia), duration of mechanical ventilation in survivors, duration of hospitalisation in survivors and mortality before the 28th day).

Conclusion:

It was concluded that results obtained with different surfactant preparations having dissimilar compositions were not different in terms of final impacts and side effects.

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Candida septicemia as a problem in neonatal intensive care units

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Objective:

Background: Disseminated Candida infections are becoming increasingly common among infants in neonatal intensive care units (NICU).

Method:

We retrospectively reviewed the charts of 1011 patients admitted to two NICUs of closely located university hospitals in the same region of Turkey between 1999-2001 and analysed the data of 19 patients with fungemia.

Results:

Fungal sepsis ratio in NICUs was 1,87 per cent. Seventeen patients (89%) were prematurely born. Mean gestational age was 30,89 weeks (23-41), mean birth weight was 1558 grams (540-3400), mean fungal detection time 15,61 days (4 –53). All patients had more than one predisposing factors such as previous or ongoing antibiotic therapy, total parenteral nutrition given by central or peripheric venous catheter and invasive procedures. Candida(C) albicans (10), C.glabrata (1), C.krusei (1), C.tropicalis (1) and C.parapsilosis (5) were isolated from blood cultures. Two patients had candiduria along with candidemia and another patient had positive pleural and peritoneal fluid cultures for the same fungi. All infants were clinically symptomatic with septic appearance. The first clinical symptom was apnea in 10 patients (52,6%). Initial laboratory evaluations showed thrombocytopeia (68%), leucopenia or leucocytosis (26%), immature/total neutrophil ratio greater than 0,20 (63%) were the common findings.

Eighteen infants received antifungal therapy. One infant died before the initiating of antifungal therapy. Six infants were treated with Amphotericin B Lipid Complex (ABLC), four with Liposomal Amphotericin B, eight with Fluconazole. Six infants died (33,3 %) on 1,2,2,7,9,10 days of antifungal therapy. First control blood culture was performed between 7 to 10 days of treatment. Fungal eradication was achieved in twelve infants and mean eradication time was 7,91 days (7-14) in surviving patients. Mean duration of antifungal therapy was 18 days (13-28) for the survivors. None of the patients had severe side effects attributed to antifungal treatment. Mortality rates were similar in infants treated with fluconazole (37,5 %) and Amphotericine B compounds (30 %). The treatment cost for 21 days of antifungal therapy is 4000 and 2200 USA dollars for liposomal Amphotericin B and ABLC respectively. The cost was only 80 USA dollars for Fluconazole.

Conclusion:

Because of high mortality rate and high treatment cost of neonatal fungal infections, preventive measures seem to be important. Fluconazole seem to be cheaper and effective alternative to Amphotericine B in the treatment of selected cases.

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A rare cause of neonatal pneumonia: Chryseobacterium meningosepticum

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Chryseobacterium meningosepticum is a ubiquitous Gram-negative bacillus most-commonly associated with meningitis in premature neonates. The microorganism is highly pathogenic for the premature babies with their immature immune systems. These bacteria also cause pneumoniae, sepsis, soft tissue, and other infections in neonates and immuncompromised adult patients. A particular characteristic of these bacteria is its intrinsic resistance to antibiotics commonly used to manage serious infections. It has been a well-documented cause of outbreaks in neonatal and adult intensive care units.

We report a case of C. meningosepticum pneumonia in a newborn that has been receiving prophylactic ampicillin and netilmicin treatment for minimally respiratory distress syndrome. After a clinical and pulmonary aggravation, the quantitative culture of the endotracheal aspirate fluid revealed 8x104 cfu/ml of this microorganism. It was found to be resistant to all imipenem and cefotaxime, gentamicin MIC values, resistant to

trimethoprim-sulfamethoxazole (MIC values >=4), and susceptible to piperacillin-tazobactam (MIC values >=4), cefepime (MIC values >=0.24). To the best of our knowledge, this is the first reported case of infection due to C. meningosepticum, from Turkey. The patient was treated successfully with cephoperazon-sulbactam.

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Nonbronchoscopic bronchoalveolar lavage for diagnosing ventilator-associated pneumonia in ventilated newborns

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Objective:

This study was designed to determine the effectiveness of nonbronchoscopic bronchoalveolar lavages (NB-BAL) in diagnosing ventilator-associated pneumonia (VAP) in ventilated newborns.

Method:

Two hundred and -seven NB-BAL were obtained from 145 intubated neonates for microbiologic and cytologic evaluation of the distal airway. The NB-BAL samples were processed for microscopic quantification of the polymorphonuclear cells (PMN) containing intracellular bacteria (ICB) (positive threshold, 2%) and quantitative culture (positive threshold, 105cfu/mL) .VAP was defined as a new, progressive, or persistent (>24 hrs) infiltrate on the chest radiograph, with two or more of the following criteria: a) macroscopically purulent tracheal secretions; b) temperature of 38.5°C or <36.5°C; c) leukocytosis of 20,000 cells/muL or leukopenia of <4000 cells/muL; and d) worsening of respiratory status with a Pa O 2 /F IO2 ratio of <240. Colonization was defined as mechanical ventilation for more than 7 days, no signs of infection and isolation of the same bacteria species in two previously obtained NB-BAL secretions.

Results:

Out of 145 neonates, 40 (27.5%) were infected and 12 (8.3%) patients were colonized. Thirty-one patients (21 %) developed VAP according to diagnostic categories based on clinical, radiologic criteria. Twenty-eight newborn with VAP (90%) had positive NB-BAL culture Accordingly, the sensitivity of BAL fluid culture for VAP diagnosis was 90%, the specificity 90%, the positive predictive value 70% and the negative predictive value 97%. The percentage of ICB was significantly higher (14.6±4.7 versus 1.1±3.4 (<0.0001)) in newborn with VAP The presence of ICBs in 2% or more on the Giemsa stain corresponded to a sensitivity of 94 %, a specificity of 83%, a positive predictive value of 94 %, and a negative predictive value of 83 %. The sensitivity and specifity of combination of ICB and NB-BAL quantitative culture in sample for diagnosis were respectively 94% and 90%. The positive and negative predictive values were, respectively, 71% and 98 %.

Conclusion:

We conclude that NB-BAL lavage is well tolerated, and clinically useful in mechanically ventilated newborns. These results suggest that NB-BAL fluid microscobic examination and cultures can offer a sensitive and specific means to diagnose VAP in ventilated newborns and may provide relevant information about the causative pathogens.

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Tympan temperature loss during crying in premature infants

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Premature infants present a poor control of the reactions to the stimuli at birth, which subsequently improves slowly in time and later on causes life threatening events. Nonshivering thermo genesis is controlled by the preoptic hypothalamic nucleus.

Objective:

The study aimed to find when and how the premature infants responded by thermoregulation to pain and hunger stimuli.

Method:

The tympan temperature was measured as an index of the hypothalamic temperature in 70 premature infants in a stable cardio respiratory state

and enteraly fed: 45 were appropriate for gestational age (AGA) and 25 were small for gestational age (SGA) infants. The age of the infants was 31.4 ± 18 days in AGA infants and 22.8 ± 12 days in SGA infants (p=0.022) but the postmenstrual age was similar (p=0.56). Tympanic thermometry detects the infrared energy emitted from the tympan without contact with the membrane, it is not influenced by the ambient temperature, and the quantity of cerumen, respiration and feeding but it is affected by the emotional state. The temperature was measured for 3-5 subsequent days by the same person, using a thermistor thermometer (Metron TC 0.5) during crying caused by intramuscular injections, venipunctures or hunger. The second measurement was made after 30 minutes in a quiet state. All infants were examined in the daytime, in supine position and the thermometer inserted into the left auditory canal by a right-handed operator.

Results:

The tympan temperature was 35.6 ± 0.74°C during crying and it increased to 35.84 ± 0.78°C in the quiet state (p=0.002). In both situations, the tympan temperatures were inversely related with the age of the infants (r= -0.21). When the data were analysed in postmenstrual age groups, only premature infants over 34 weeks showed significantly lower (p=0.026) tympan temperatures during crying than during the quiet state.

Conclusion:

The premature infants are able to respond to pain and hunger stimuli at 35 weeks of gestation by lowering the tympan temperature and to induce thermo genesis by raising their body temperature.

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Is subdural hygroma in childhood a controversial lesion?

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Medical history:

A 9-month-old boy was found dead in his bed. Pregnancy and birth had been without complications; he was born at term weighting 3040 grams, and with a length at 49 cm. The head circumference was measured to 35 cm. At 3 months of age the growth of the head was expanding abnormally from normal (0 standard deviations (S.D.)) to 3S.D. at 7 month of age. Otherwise the boy had developed normally. There was no history of any traumas or head injuries.

Clinical diagnosis:

Because of the abnormal growth of the cranium, the boy was examined and the shape of the cranium and jaw was described as being wide, the backhead flat. As tentative diagnosis premature synostosis of the cranial sutures was suggested, which was confirmed by a computerised tomography, which also showed an external hydrocephalus. As differential diagnosis subdural hygroma was suggested.

Autopsy findings:

No syndrome like features were found, except for the above-described cranial shape. No fresh or older lesions or bone fractures were seen. The internal examination showed a thick gelatinous mass under the dura and pettechial bleedings in the pleura.

Neuropathology:

Based on this examination the following diagnosis were made: Hygroma and fibrosis of the leptomeningeum, recent haemorrhage of the falx cerebri and the dura of the anterior fossa cranii and oedema cerebri.

Conclusion:

The changes found in the brain may have contributed to the death even though no exact cause of death was found.

Discussion:

Based on this case the origin and pathogenesis of subdural hygroma is discussed. Is this controversial case a case of an overlooked shaken baby syndrome?

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Accidental suffocation from an anti-allergen quilt

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The relation between sudden infant death syndrome and the infant's sleeping environment seems to be well documented. One of risk factors is thought to be the use of quilts or duvets. The poster presents a case where an inappropriate use of an anti-allergen quilt caused death of an infant.

One morning a 14-month old girl was found dead in her bed. She was found with her anti-allergen quilt wrapped round her head. Attempt to resuscitate her failed.

The autopsy showed no signs of violence. There was slight acute tracheobronchitis and moderate to severe acute emphysema of the lungs.

The examination of the quilt showed that the surface was very smooth and soft. The quilt consisted of quadrants separated by welding in the ticking material, but most of the welds were ruptured making the quilt one-chambered. The quilt had been used without a cover.

Examination of the quilt ticking material at The Danish Technological Institute showed low air permeability, even lower than in material used for wind jackets and other windproof clothing's.

Using the quilt without a cover made close contact of the face to the smooth and almost air impermeable ticking material of the quilt possible. The defect quadrants displaced all the goose downs in the bottom of the quilt. It was therefore possible to wrap two or more layers of the quilt material round the head. It was concluded that the cause of death was suffocation due to the inappropriate use of an anti-allergen quilt.

The air permeability test results, a sample of the quilt material and the recommendations of The Danish Consumer Council regarding use of this type of quilts will be demonstrated.

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Oral nystatin propylaxis significantly reduces candidal colonization in vlbw infants

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Background:

Systemic candidal infections are responsible as many as 15 % of nosocomial infections among VLBW infants with a mortality rate of 25-60 % and a higher incidence of neurodevelopmental disabilities. Prolonged intubation, corticosteroids, parenteral nutrition, central line utilization, broad-spectrum antibiotics and teophylline are well known risk factors. However colonization with candidal species seems to be the most important predictor of invasive disease.

Objective:

To find out whether prophylactic oral nystatin results in a decreased incidence of candidal colonization in the VLBW infant

Subjects and methods:

Setting: A tertiary level Neonatal Intensive Care Unit (NICU) in Macedonio Melloni Maternity Hospital of Milan

Design:

Retrospective chart review of all VLBW infants admitted to NICU between January 1, 1999 and December 31,2001.

Methods:

Rectal, pharingeal, urine and blood cultures were performed in the 1st, 7th 14th and 28th day of life. Cultures were plated on SDA media for the isolation of candidal species. Propylactic oral nystatin has been introduced as a routine preventive measure for VLBW infants beginning from January 2000. Patients before the era of prophylaxis (1999) made up group 1 (n=49), while patients in the period of prophylaxis (2000, 2001) were considered as group 2 (n=91). Incidence of candidal colonization was compared between two groups.

Results:

A total number of 140 VLBW infants were enrolled in the study. The two groups were homogenous regarding demographic characteristics, risk factors, and treatment modalities. It has been found a dramatical decrease in the incidence of colonization from 27 % to 8.6 % as a result of the prophylaxis.

Conclusion:

Prophylactic administration of oral nystatin for VLBW infants decreases the colonization by candida species, which is the main contributor to fatal candidal sepsis. This study demonstrates that though simple and cheap, nystatin can still be effective in decreasing the VLBW mortality in due to candidal infections.

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Routine use of fentanyl infusions for pain and stress reduction in mechanically ventilated premature infant

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Objective:

For pain perception during the late gestational period. Pain perception and behavioural and neoruendocrinological responses against pain causes important clinical consequences in premature newborns. Fentanyl analgesia is an effective pharmacological measure against pain.

Method:

A total of 24 premature newborns are included in the study. The study cases are randomised into two groups and one group received fentanyl infusion at a dose of 1 µgr/kg/hour and the other group received placebo (%0, 9 NaCl). The characteristic features of the study (n=12) and placebo (n=12) group were similar. Serum glucose, cortisol, growth hormone and prolactin levels are measured at baseline, 24th, 48th ve 72nd hours in both groups and compared within and between groups.

Results:

Baseline level of mean serum cortisol (38,3, 27,9 g/dl) significantly decreased at 24th and 48th hours (12,412,8 and 10,17,1g/dl, respectively) when compared to placebo group ($p<0,05$). There were no statistically significant differences between groups in respect to serum glucose, prolactin and growth hormone levels. Behavioural sedation scores were less in fentanyl group ($p<0,05$ for 24th hour and $p<0,01$ for 48th and 72nd hours). When fentanyl and placebo groups were compared hemodynamically, it was seen that the mean blood pressure measurements during the first 24-hour were less in fentanyl group (597 mmHg) ($p<0,05$). The other hemodynamic parameters were similar. Comparisons according to the need for mechanical ventilation revealed that the requirement to positive inspiratory pressure at 24th and 72nd hours were less in fentnayl group (171 cmH2O in both times) ($p<0,05$ and $p<0,01$, respectively). The ratios of intraventricular hemorrhagia were 50 percent and 8 percent in placebo and fentanyl groups, respectively ($p<0,05$). There was 50 percent gastrointestinal dismotility in the fentanyl group and this was significantly higher than the placebo group ($p<0,05$).

Conclusion:

In conclusion, the application of fentanyl analgesia in premature newborns receiving mechanical ventilation is a safe and effective method which improves the behavioural and neuroendocrine responses to pain and which has positive influences on short-term clinical outcomes, with preventable side effects.

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Investigation of the il-10 gene in sudden infant death

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Cytokines are involved in regulation of the intensity and duration of the immune response, and the cytokine production is carefully regulated. Of special interest with regard to sudden infant death is IL-10, which is an immunregulatory cytokine that plays an important role in the development of infectious disease. The variability in IL-10 production has a hereditary component of approximately 75%, and this variation is mainly due to polymorphisms in the promotor region of the IL-10 gene. The best-described polymorphisms are three single nucleotide polymorphisms in bp –1082, -819 and –592, and two short tandem repeats (STR) named IL-10G and IL-10R. Previous studies have shown a possible association both between the –819/–592 TA allele and SIDS and the –819/–592 TA allele and infectious death. Several reports describe immune stimulation in SIDS, and elevated levels of several interleukins have been reported in SIDS victims.

Objective:

The purpose of the present study was to analyse the distribution of the different fragments of the two STR loci in the IL-10 gene promotor in SIDS cases and controls.

Method design:

The subjects consist of 180 cases of SIDS, 44 cases of borderline SIDS, 33 cases of infectious death and 79 living infant controls. The methods used were polymerase chain reaction and fragment analysis.

Results:

Four different R alleles and 10 different G alleles were detected. These alleles combined into 6 different R genotypes and 36 different G genotypes. There were no differences between the groups, neither with respect to IL-10R or IL-10G. This was true both when regarding allele and genotype distribution.

Conclusion:

The present study does not disclose any association between a specific IL-10R or IL-10G genotype and sudden infant death. However, based on previous findings, it cannot be ruled out that polymorphisms in the IL-10 gene contribute to a genetic make-up predisposing to SIDS. The mode of inheritance in SIDS is most likely multifactorial, and the genes involved in the immune system are assumed to be of great importance.

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Apoptosis in erythroid precursor cells in umbilical cord blood (CB)

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Background:

Programmed cell death (apoptosis) is a physiological mechanism by which organ genesis and homeostasis are regulated in fetal life. Newborns have high Hb levels at birth, but Hb falls, in term babies to physiological levels, in preterm infants to levels where anemia often induces symptoms and requires treatment. It is questioned if an "intrinsic" pre-programmed mechanism of apoptosis influence this development and if the erythroid precursors from preterm babies are more vulnerable to apoptosis than those in term babies.

Aim of the study:

To study the degree of apoptosis in erythroid precursor cells in CB in term versus preterm babies.

Material and methods:

We studied CB from healthy term babies, and so far one preterm baby. Mononuclear cells were separated using gradient separation (Lymphoprep). Erythroid precursor cells express glycophorin A (Gly A) on their surface and were separated from other mononuclear cells by magnetically labelled antibodies (Miltenyi Biotec) towards Gly A. Flowcytometric methods were used to estimate apoptosis (Annexin-Pi). We compared the degree of apoptosis in fresh and incubated cells from term and preterm babies.

Results:

CB from 10 term babies (mean GA 38,6 wk, BW 3572g) and one preterm baby (Ga of 26 wk, Bw 648g) have been analysed. The level of apoptosis is very low in Gly A positive cells from fresh CB (0,6%) in term babies. Gly A positive cells show a significant rise in apoptosis when studied after 18 hours of incubation. In the preterm baby, a similar very low degree of apoptosis was found (0,3%) in freshly isolated Gly A positive cells.

Conclusion:

In healthy term babies, and so far one preterm baby, the degree of apoptosis in erythroid precursor cells is very low. Further studies on preterm babies will be conducted.

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Urinary tract infection as a risk factor for sudden infant death syndrome

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Objective:

To examine UTI as a risk factor for Sudden Infant Death Syndrome

Method Design:

An eight-year case control study was conducted using data on infants who died from SIDS in the Republic of Ireland between the 1st January 1994 and the 31st December 2001. All sudden unexpected infant deaths in the Republic of Ireland are reported to the Irish Sudden Infant Death Association's National SIDS Register within 48 hours of the infant's death. Four controls per case are then selected matched by hospital and date of birth. There was an 81% response rate from the cases and a 77% response rate from the controls. Statistical analysis of the data collected was carried out using STATA version 6. All data was analysed by multiple conditional logistic regression. Differences between cases and controls were expressed using odds ratios and 95% confidence intervals. The variable for UTI was based on the mother's response (yes/no) when questioned as to whether she had suffered a urinary tract infection at any point during her pregnancy.

Results:

Twenty three percent of case mothers stated that they had contracted a urinary tract infection during pregnancy in comparison with only 6% of controls. In a univariate analysis occurrence of a UTI increased the baby's risk of SIDS by a factor of 4.82 (95% CI 2.78; 8.34). This was reduced to a factor of 2.67 when adjusted for the influence of maternal age, education, social disadvantage, smoking and taking antibiotics during pregnancy but remained statistically significant (95% CI 1.24; 5.75).

Conclusion:

The results highlight that urinary tract infection may be a significant risk factor of SIDS when account is taken of maternal age, education, social disadvantage, smoking and antibiotics during pregnancy

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Neonatal levels of 8-isoprostane, a marker of oxidative stress, in pregnancies associated with augmented risk for SIDS

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Objective:

Intrauterine conditions could be of importance for the vulnerability of an infant who dies from sudden infant death syndrome (SIDS). Epidemiological studies have shown that preeclampsia is associated with an increased risk for SIDS, and diabetic pregnancies are also at increased risk of perinatal deaths. Oxidative stress has been suggested to be involved in the development of both preeclampsia and SIDS.

8-isoprostane is a lipid peroxidation product, and a stable marker of oxidative stress. We wanted to evaluate the plasma levels of 8-isoprostane in

newborns from pregnancies complicated by diabetes mellitus or preeclampsia, compared to control pregnancies.

Methods design:

Women with preeclampsia, diabetes and normal controls, who were delivered by caeserean section, were included. Blood from the umbilical vein and arteries was collected immediately after cord clamping. In addition, amniotic fluid was collected. Total 8-isoprostane levels are analysed with GC/MS technique.

Results:

Laboratory analyses are still in progression, results will be presented at the meeting.

Conclusion:

8-isoprostane is a marker of oxidative stress that has been used in several clinical studies the last decade. This is a pilot study measuring 8-isoprostane levels in umbilical cord blood and in amniotic fluid from pregnancies complicated by preeclampsia or diabetes, pregnancies which are associated with maternal oxidative stress. A possible fetal oxidative stress in these fetuses could be of importance for the augmented risk for SIDS.

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Character of bacterial colonization of newborns in a neonatal intensive care unit and its relationship with nosocomial infections

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Objective:

We aimed to investigate the colonization of the neonates admitted in the neonatal intensive care unit of Cerrahpasa Medical School in a prospective study.

Method design:

During a period of 8 months, a surveillance study was conducted in 53 patients. Endotracheal, oropharyngeal, nasal, axillary and stool specimens were obtained during the first 24 hours of life, 4th, 7th day and then once a week during the rest of the hospitalisation. In case an infection was suspected, blood, CSF, urine, etc were also cultured. Environmental and staff hands specimens were also obtained.

Results:

A total of 1210 specimens were examined, and 599 bacteria species and 14 yeast species were isolated. K. pneumoniae was the most frequently isolated micro organism (42.7%) and coagulase negative staphylococci were the second (26.1%). No diphteroid rods, which are considered as the member of the normal skin flora, were cultured. In only 11.9% of cases, viridans streptococci were isolated in the oropharynx and in 5.9% of cases E. coli was cultured in the stool. High resistance was observed in Gram-negative bacilli against third generation cephalosporins and aminoglicosides, in staphylococci against methicillin and in streptococci against penicillin. Colonized strains were isolated from surfaces and hand cultures. The role of health care workers and technical equipments in transmissing the resistant Intensive Care Unit flora, was observed. Median beginning of colonization was 5.3±3.2 days. The rate of nosocomial pneumonia and the rate for "late onset sepsis" were 16,9%, 5.6%, respectively. The occurrence rate of infection with colonized strains was 70%. K. pneumoniae was the etiological agent of nosocomial infections in 38.1%, P. aeruginosa in 16.6% and C. albicans in 8.3% of cases.

Conclusion:

Colonization was strongly associated with beginning of oral feeding. Feeding with orogastric tube and prolonged hospitalisation were the major factors for colonization with high resistant pathogens. Colonization surveillance studies are important to perform in neonatal intensive care units in guiding the management decisions and in adapting the initial empirical antibiotic therapy.

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Sudden infant death syndrome, historical aspects of a still unsolved riddle - a vanishing epidemic?

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Objective:

Historical, sociological and medical aspects of SIDS

Method Design:

Review of the literature from antiquity until present times

Results:

The oldest documentation of crib death dates back to 500 BC. In 50 AD Soran from Ephesos warned against overlaying by negligent wet nurses and advised on sleeping position. During the middle ages the laws of the church influenced secular legislation with defined measures of punishment in cases of overlaying. The ecclesiastical right to punish was retained in all churches during the reformation and in the Protestant church the punishment became more severe. In medicine a publication warning against smothering of infants was published in 1609 and in 1677 case reports similar to modern reports were given. During the 18th century the prevention of overlaying was of great concern for many scientific academies and in 1780 and 1795 two textbooks discussed the existing knowledge of prevention of overlaying and smothering of sucklings. From the early part of the 19th century multiple theories have been proposed, beginning with the status thymo-lymphaticus and concluding today that after excluding natural causes, several trigger mechanisms may contribute to death. The campaign for return to sleeping in the supine position during the last ten years has lowered the death rate dramatically but has not solved the problem completely.

Conclusion:

The enigma of SIDS is not yet solved, though risk factors have been identified lowering the SIDS rate. The epidemiology has become more complicated and combinations of factors triggering a mechanism leading to death will continue to be discovered. Even if the epidemic is a vanishing one, a continuous search for an explanation is necessary, not forgetting the unimaginable pain these deaths cause the family involved.

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Epidemiology of apparent life threatening event (ALTE) in the Tyro

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Objective:

We investigated the epidemiology of ALTE in the Tyrol, a federal state in the western part of Austria. Aim of the study was to evaluate descriptive characteristics of ALTE and the importance of SIDS risk factors for ALTE infants.

Method design:

Since 1994 data on childcare practices for all infants born in the Tyrol were prospectively collected (participation rate 70%). Hospital records for all ALTE infants were reviewed according to a standardised protocol.

Results:

Between 1994 and 2001 a total of 162 infants with the diagnosis ALTE were enrolled in the study. Mean annual incidence was 2.6/1000 live births, incidence slightly decreased over the time period. Peak incidences for ALTE was at nine weeks of age, none of the infants who suffered ALTE subsequently died from SIDS. The feature most prominently associated with ALTE was parental reported apnoea in the first weeks of life. ALTE infants were more likely to sleep in the prone position than control infants but clearly less likely than SIDS infants. Further differences and similarities in the risk profile of ALTE and SIDS are described.

Conclusion:

In spite of some common features in SIDS and ALTE epidemiology, differences are more pronounced. Our results argue against common major pathomechanisms in both diseases.

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Subtle upper airways abnormalities in infants with apnea

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Objective:

This study examines the incidence of subtle airways lesions in infants with apnea and evaluates the contribution of these lesions to apnea episodes.

Study design:

Retrospective review of endoscopy evaluations conducted at a pediatric airways service between 1996 and 2001.

Methods:

Review of consecutive endoscopic findings and of clinical and laboratory data of 68 infants presented with apnea.

Results:

Seventy eight percent of these infants exhibited airways pathologies previously undiagnosed, with 52.9% demonstrating multiple synchronous lesions. The most common findings were dynamic pharyngeal wall collapse, laryngomalacia, adenoid hypertrophy, and tracheomalacia. Rare cases included supraglottic hemangioma, subglottic cyst and esophageal foreign body. Obstructive sleep apnea was diagnosed by polysomnography in 48.5%. Gastroesophageal reflux, failure to thrive and generalized hypotonia were common findings among these infants found in 41%, 55% and 80% respectively. In most cases, treatment of choice was conservative. Surgery was only performed when anatomical obstruction was found (35.5% of the infants). Although surgery relieved most symptoms in the early postoperative period, in 32% symptoms persisted and further treatment was required.

Conclusions:

Upper airways lesions in apnea, especially synchronous anatomical and dynamic appear to be under-diagnosed. Polysomnography and examination throughout the airway tract using flexible endoscopy are mandatory for the workup of apnea in infants.

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The effect on premature infants restrained in car seats of maintaining a neutral upper airway position

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Objective:

To test the hypothesis that an infant car seat modification that allows the infant to maintain a neutral upper airway position would prevent narrowing of the upper airway space due to head flexion. It would thus avoid oxygen desaturation during sleep in preterm infants restrained in car seats.

Method:

Seventeen preterm infants reaching 34-36 weeks gestation were studied at the week of discharge in a car seat for newborns, with, and without, a foam insert placed behind the body of the infant to allow the head to remain in a neutral position on the trunk. Infants were monitored in each position for 30 minutes with continuous pulse oxymetry and recording of respiratory, cardiac and nasal airflow activity. Respiration timed inspiratory radiographs, performed for assessment of upper airway dimensions, were taken in each position when the infants were quietly asleep.

Results:

Placement in an approved reclining car seat was associated with narrowing of the upper airway space compared to airway size when the inserts were in place (Mean SEM 3.6 +- .3 vs 5.2 +- .3mm, $p < 0.001$). No episodes of significant oxygen desaturation occurred while the inserts were in place. In contrast, some infants showed desaturation to SaO2 of 85% in the standard car seat without inserts ($p < 0.05$, Chi2). Desaturation was preceded by an obstructive breathing pattern on the polygraph.

Conclusion:

Preterm infants are well known to be at risk of oxygen desaturation when restrained in car seats. The present data strongly supports the hypothesis that this is due to flexion of the head leading to

posterior displacement of the mandible causing upper airways narrowing, sometimes to complete obstruction; and demonstrate that desaturation during sleep can be prevented by placement of a simple foam insert in the car seat that allows the infant to maintain its head in a neutral position on the trunk, and thus prevents this mandibular pressure.

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Low birth weight and sleep behaviour in two-month-old infants

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Objective:

The study aimed at evaluating a possible association between low birth weight (LBW) and certain behavioural features during sleep in 2-month-old infants.

Method Design:

Eighty-three infants (35 boys, 48 girls) who were born in St. Petersburg in 2000 to 2002 and met a conventional definition of LBW (< 2500 g at birth) entered the study. Of these, 66 (30 boys, 36 girls) were born both light and pre term (gestational age <= 36 weeks), and 17 (8 boys, 9 girls) were born light but at term. The control group consisted of 112 infants (51 boys, 61 girls), made up of healthy babies born at term and having appropriate birth weight, matched to case babies for gender distribution, date of birth, calendar age at study, and geographical distribution within a city as close as possible. The mothers were asked to fill in the questionnaire addressing infant, maternal and demographic major characteristics, as well as infant's major clinical signs and symptoms during day time and at night. As a part of interview, the mothers also completed a questionnaire addressing infant's behavioural features during sleep, which was based on Children's Sleep Habits Questionnaire.

Results and Conclusion:

LBW infants presented with longer average total sleep duration (difference in the means equal to one hour); principally due to longer night sleep. More often the parents rated LBW infants as sleeping too long and falling asleep in the transport; by the bed time, they were more often ready to fall asleep, but more frequently needed parents in the room and were put into parental bed before. No statistically significant difference was found between two groups in consideration for the time the baby usually spent wake during night, although LBW infants were more frequently brought to parental bed, if awake. These associations remained significant after adjustments have been made for major potential confounders. No statistically significant difference was found for any sleep characteristic in consideration between LBW infants who were born at term and pre term. LBW and the factors leading to it should be considered cautiously as possible predictors of certain infant behavioural features and parental interventions during infant's sleep.

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SIDS and LQTS: A genetic and epidemiological analysis of 41 SIDS victims.

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Background:

The congenital long QT syndrome (LQTS), which is caused by mutations in several cardiac ion channel genes, has been identified as the reason for ventricular tachyarrhythmias and sudden cardiac death. Prolongation of the QT has been associated with sudden infant death syndrome (SIDS), which is the leading cause of death among infants under one year of age. Although SIDS is a multifactorial disorder, the theory raises the possibility that congenital LQTS may contribute to a substantial part to premature sudden cardiac death. To date, genetic analysis of LQTS genes of randomly selected SIDS victims has only been performed in a single LQTS gene (SCN5A, LQTS 3).

Methods and Results:

Amutation screening with SSCP and sequencing techniques has been performed in 44 randomly selected SIDS cases from the Westfalian crib death study (1990-1994). Genetic analysis with SSCP (single strand conformation polymorphism) analysis and direct sequencing techniques of the complete coding sequences of the five LQTS genes (KCNQ1, KCNH2, SCN5A, KCNE1 and KCNE2) were performed. A family history for long QT syndrome was suspected in 2 of the 44 cases. In one SIDS case without a family history of LQTS a missense mutation in the KCNQ1 gene has been identified. Additionally several silent mutations, polymorphisms and intron mutations spreaded over LQT1 to LQTS5 have been identified.

Conclusion:

Genetic screening of randomly selected SIDS cases revealed in this study in approximately 2.5 % an identifiable LQTS channel defect. Mutations in cardiac ion channels as the basis for the development of lethal arrhythmias should be taken in consideration when searching for causes of sudden infant death.

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