CONSENSUS ON DIAGNOSTIC CRITERIA FOR THE EXCLUSION OF SIDS

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The great discrepancy with regard to SIDS rate throughout the world, may be due to different definitions and diagnostic criteria for sudden unexpected death in infancy. Experts from Europe, North America and Australia have met in Oslo to address the issue.

Consensus was reached for cardiac pathology, lung pathology and for investigations of genetic/metabolic disorders. Consensus with regard to cerebral pathology and a common international autopsy protocol is under way. More work is needed to operationalise observations from the death scene and information from the family history and circumstances of death.

In November 2003 forensic and paediatric pathologists as well as other experts met at Soria Moria in Oslo to discuss diagnostic criteria for sudden unexpected infant death. The aim of the conference was to reach consensus with regard to exclusion criteria for sudden infant death syndrome (SIDS). Another aim was to build a bridge between the Middle European way of stratifying sudden unexpected death in infancy (SUDI) and the Anglo American/Nordic way of using the diagnosis SIDS.

Previous efforts and failure to standardise the diagnostic approach

In 1992 forensic pathologists from the Nordic countries were able to agree upon common diagnostic criteria for SIDS (1). This approach led to standardisation of the diagnostic practises in the Nordic countries (2,3). This diagnostic harmony within the Nordic countries was in contrast to the rest of Europe and the world where the stratification of SUDI caused serious problems (4,5)(Table 1).

Table 1. Proportion of SUDI diagnosed as explained death (non-SIDS) by different authors

Helweg-Larsen et al, 1992(6):	70%
Arneil et al, 1985(7):	45%
Rajs, 1993(8):	26%
Mitchell et al, 2000(9):	25%
Knowelden at al, 1984(10):	17%
Imbert et al, 1992(11):	17%
Taylor et al, 1990(12):	17%
Byard et al, 1994(13):	9%
Cheron et al, 1993(14):	4.5%
F Hatton et al, 1995(4):	2.5%

Discrepancy in the way of diagnosing SUDI/SIDS may in fact be one important reason for the large variation in SIDS-rates throughout Europe and the rest of the world (Fig 1)

In Middle Europe sudden unexpected death in infancy has been diagnosed as explained death, partly explained death and unexplained death (15,16). However, for practical purposes:

unexplained death= SIDS and partly explained death=borderline-SIDS

Decrease in SIDS-rates – increase in diagnostic problems!

Around 1990 most western countries experienced a dramatic drop in the SIDS-rates (3). Genuine SIDS cases decreased in number whereas the remaining SUDIs did not change to a similar degree (Fig 2). However, the relative proportion of non-SIDS cases has increased after 1989 (Fig 3).

As a result of this development the relative proportion of deaths due to poor parenthood, neglect and abuse has increased (17). Thus for the pathologist who diagnoses SUDI, it appears as if the so called grey zone cases (Fig 4) has increased.

A Standard diagnostic practise - premise for research and for legal protection

Although SIDS is most likely a heterogeneous entity, the syndrome has been significant in the fight against sudden unexpected deaths in infancy. The term does not necessarily refer to a specific disease process, but to a lethal situation in which and infant, who may

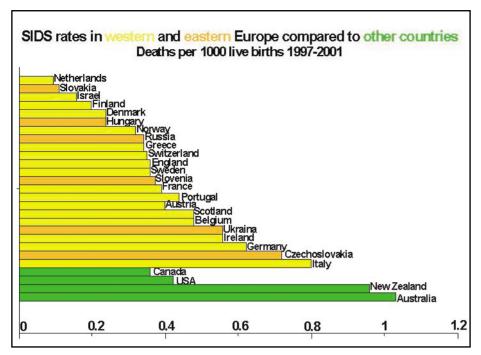


Figure 1. SIDS rates in western and eastern Europe compared to some other countries. Deaths per 1000 live births 1997-2001 (WHO)

The large variation may partly be due to different ways of diagnosing SIDS.

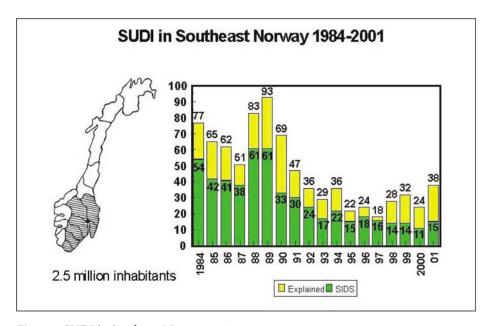


Figure 2. SUDI in Southeast Norway 1984-2001

be physiologically compromised in poorly understood ways, is subjected to additive effects of a number of risk factors at a particularly vulnerable time of life (18). Several authors have independently proposed models for the understanding of SIDS as a triple sequence event (19-21). It may well be that different infants may have different predisposing factors e.g. aquired or inherited abnormalities. Recently, Carl Hunt in an interview with Science claimed: "There are multiple genetic risk factor for SIDS" (22). Others (di Maio) deny any genetic inheritance in SIDS,

claiming that: One sudden unexplained death in a family is tragic but innocent, two is suspicious, three is murder. The latter way of thinking has led to conviction of mothers who have lost several infants suddenly and unexpectedly. Conversely the assumption of multiple genetic risk factors for SIDS has led to acquittal in such cases (22).

It seems more important than ever to make the diagnosis of SIDS based on commonly accepted diagnostic (exclusion) criteria. Risk factors for SIDS should not be introduced as causes of death. Moreover, the diagnostic exclusion criteria must be based on observations made in infants who have died from clear cut disease, accidents or maltreatment.

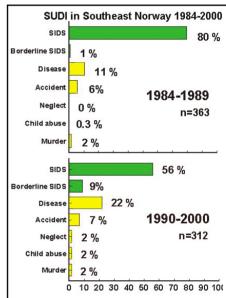


Figure 3. SUDI in Southeast Norway 1984-1989 and 1990-2000. The proportion of SIDS cases has decreased whereas the proportion of non-SIDS cases has increased. Cases of neglect and abuse were almost lacking in the first period.

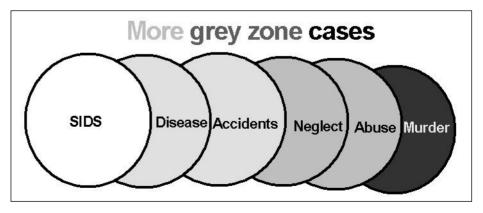


Figure 4. Increased awareness of grey zone cases

Most pathologists who deal with SUDI recognise that they need to consult different types of experts to ensure reliable diagnoses. In many countries case conferences have been implemented. In such conferences experts like biologists, microbiologists, genetesists, neuropathologists, paediatricians, toxicologists and police experts play an important role (17).

AIMS OF THE CONSENSUS **MEETING**

The experts have tried to reach consensus on interpretation of the following diagnostic steps:

- Death scene investigation
- ÍÍ) Family history, injuries and inflicted disease and other circumstansces
- III) Cerebral changes
- IV) Respiratory system examination
- Heart examination
- VÍ) Metabolic/genetic investigation
- Autopsy protocols for diagnostic purposes, extensiveness of sampling and interpretation of findings.

RESULTS AND RECOMMENDATIONS

I) Significance of death scene investigaton

L Sveum, P Sidebotham, M Schlaud, P Blair, TO Rognum

Techniques for videotaping of death scenes were demonstrated and discussed, and a scoring system for evaluation of risk factors was proposed (Table 2).

A preliminary study of 33 SUDI and 33 controls showed a clear cut difference in the risk score profile (Fig 5).

Discussion and further development The need for more detailed studies of

the death scene was agreed upon. It is possible that qualitative studies might be performed to generate new hypothesis concerning risk factors.

A risk score system based on the results of the Avon study was proposed for implementation in the future (Table 3).

Table 2. Scoring system applied in a preliminiary study Variables connected with sleeping environment scores given for:

	Scores	Variation of scores		
Sleeping position when found Firmness of mattress Layers of clothing Type of duvet Room temperature Co-sleeping	1 - 4 1 - 4 1 - 4 1 - 4 1 - 2 1 - 2	1 (supine) - 4 (face straight down) 1 (firm) - 4 (very soft) 1 (one) - 4 (≤2) 1 (light) - 4 (warm, thick and heavy) 1 (≤22°C) - 2 (>22°C) 1 (own bed) - 2 (with adult)		
Range of scores	6 - 20			

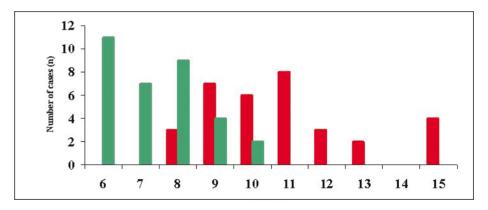


Figure 5. Risk score for SUDI ■ and controls ■. As expected the SIDS cases had higher risk scores than the controls.

II) Family history, injuries, inflicted disease, and other circumstances

M Gregersen, R W Byard, A Borthne, J Grøgaard, L Sveum, T O Rognum.

The most difficult area in the Soria Moria meeting involved developing a scoring system for the evaluation of grey zone cases (accidents, poor parenthood, neglect, abuse) (Table 4).

Cases were presented and a proposed scoring system was discussed.

The case of an 8-month-old boy was presented. He had fallen down between his mother's bed and a cradle and was suffocated with his face being pressed against the side of the mattress

The case received a score of 0 (accident). Another case where the parents

The Avon	Clinicopa	thologic	cal classif	fication o	f SUDI.	
Name: Date of death			udy Number. ate of Birth	•••••	•••••	
Hospital				ımber		
Place of Postmor		11 D	ospitai umit m sthologist*	 	*********	
Paediatrician*				• • • • • • • • • • • • • • • • • • • •		
HV*			r `		• • • • • • • • • • • • • • • • • • • •	
Child Protection				at LCD meetin		
Child I Totection	officer					
(*note if present at Local Case Discussion meeting)						
Classification**	0	I A	I B	II A	II B	TIT
Contributory or	Information	No factors				III
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potentially	not	identified	not likely	may have	certainly	and
"causal" Factors	collected		to have	contributed	contributed	provides a
	1	1	contributed	to ill health,	to ill	complete
	1	1	to ill health	or possibly	health, and	and
	1	1	or to death.	to death	probably	sufficient
	1	1			contributed	cause of
					to the death	death
Social factors						
Non-accidental						
injury/ evidence						
of abuse or harm						
Past Medical						
history						
Family history						
History of final						<u> </u>
events						
Death-scene	<u></u>					
examination						
Radiology						
Toxicology						
Microbiology /						
Virology						
Gross pathology						
Histology	1					
Biochemistry		 				1
Metabolic	·	1				
investigations						
Special						
investigations						
(e.g.	1	1				İ
histochemistry)						
Other (specify)						
Overall						
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Table 4. Standardised observations in the grey zone cases poor parenthood & neglect.

Fatal Event	Score
Accident	0
Poor parenthood	1
Neglect Abuse	2
Abuse	3

had left their 2 ½- month-old girl for 18 hours in a warm environment resulted in death from overheating and dehydration. This case received a score of 2 (neglect).

Discussion and future development

Sections II and VII were characterised by their multi-disciplinary discussion. A paediatrician, a radiologist, forensic pathologists and a police expert contributed with invited presentations on Munchausen syndrome by proxy, bone injuries, failure to thrive, exposure of discrepancies between history and injuries as well as the technique of interviewing parents. However, the discussion is far from being finished. The different disciplines need each

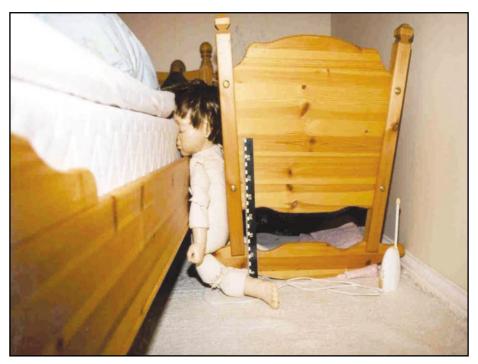


Figure 6. A 8-month-old boy suffocated between his mother's bed and the side of a cradle. His face was pressed against the side of the mattress.

other to broad the difficult area between the health system and the legal system. This important field should be subjected to more research.

III) Cerebral changes

G Stoltenburg-Didinger, K Skullerud, HF Krous

The following criteria for the classification of cerebral changes were recommended for use in the Nordic study of sudden infant death by a project group at the Nordic SIDS meeting in Copenhagen, November 22-23, 1991.

Neuropathology and SIDS

There is no general agreement about the significance of the cerebral abnormalities found in cases of SIDS. Scientific reports have focused on loss of neurons in the hypoglossal nuclei, gliosis in the brain stem, retarded myelination, and hypoxic/ischaemic brain damage (23-28). In addition a careful neuropathological examination is necessary to exclude other cerebral causes of death in all cases of sudden infant death.

Suggested criteria

- 1. Brains with definite lethal lesions, e.g., bacterial meningitis, encephalitis, microscopical and macroscopical brain abscesses and traumatic cerebral lesions (diffuse axonal injury).
- 2. Brains with potentially lethal lesions, e.g., malformations, metabolic and degenerative disorders, perinatal and later postnatal hypoxic/ ischaemic brain damage (intraventricular haemorrhages, periventricular leukomalacia, infarcts in the cerebral grey matter, selective neuron necrosis, cardiac arrest encephalopathy). Disturbances in the cerebrospinal fluid circulation (e.g. hydrocephalus slit ventricle).
- 3. Structurally normal brains and brains with non-specific changes (SIDS cases), e.g., hyperaemia, moderate brain oedema.

Recommended guidelines for the neuropathological examination

The brain should be fixed in 10% formalin for at least 6 days. Bilateral sections are taken from the hippocampus. Unilateral sections from the frontal lobe, the parietal lobe, including the periventricular white matter, the corpus striatum, thalamus as well as at least one section from the cerebellum, mesencephalon, pons, medulla oblongata, and the cervical part of the medullary cord, are needed (Figures 7-9)(29).

With regard to staining methods, haematoxylin and eosin should be used for all sections. Other staining methods are needed in some cases, including staining for

- myelin (e.g. Klüver-Barrera, Luxol, immunohistochemistry for myelin basic protein),
- gliosis (e.g. immunohistochemical

- detection of glial fibrillary acidic protein [GFAP])
- nerve cells and axons (e.g. Bodian,
- Bielschowsky or immunohistochemical methods, including amyloid precursor protein (APP)),
- mircroglial cells (e.g. immunohistochemical analysis with CD68).

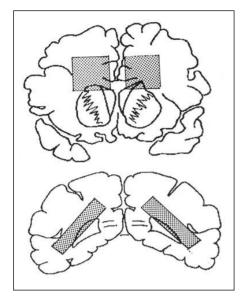
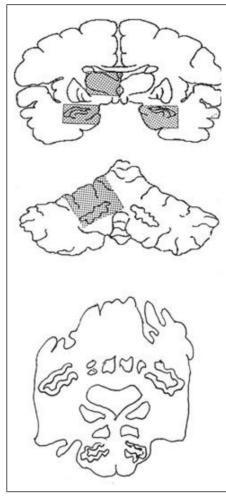


Figure 7. Recommended sections for the diagnosis of periventricular leukomalacia.

Figure 8. Recommended sections from the thalamus, hippocampus and cerebellar cortex to disclose hypoxic neuron necrosis at any age. An additional section from the pons is needed to detect a perinatal selective neuron necrosis. Alternatively the sections may be taken from the cerebellar peduncles



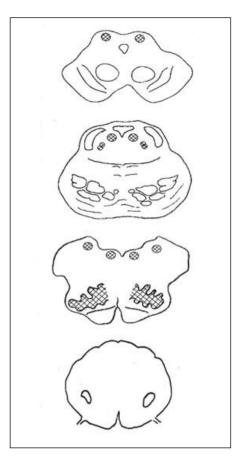


Figure 9. Nuclei affected by cardiac arrest encephalopathy: diffuse neuronal necrosis or infarcts.

IV) Lesions in the respiratory system

C V Isaksen, M Gregersen, T Bajanowski

The criteria for SIDS, borderline-SIDS and explained death remains unchanged as to the lung findings, but a revision also include criteria for interstitial changes.

There were 3 presentations:

- Markil Gregersen presented the Nordic Diagnostic criteria for exclusion of SIDS in the respiratory system (30).
- 2) Thomas Bajanowksi presented diagnostic criteria for explained SUDI in the respiratory system according to the German protocol.
- Christina Vogt Isaksen summarized the presentations under the heading The significance of respiratory system lesions – revised Nordic criteria.

Categories of Sudden Unexpected Death in Infancy (SUDI)

- SIDS
- Borderline SIDS
- Explained infant deaths

Classification of lung pathology

- Macroscopic evaluation of the lungs:
 - Weight (do the whole lungs and the sections float?)
 - Petechiae (none, scarce, abundant, dense)
- Tissue sampling:
 - Two tissue sections from each

lobe of the lung, one peripheral and one central, should be examined

• Microscopic evaluation of the lungs:

- Non-specific histologic lung findings
- Pleural petechiae
- Congestion
- Alveolar hemorrhage
- Septal hemorrhage/interstitial emphysema
- Pulmonary edema
- Alveolar macrophages
- Atelectasis and emphysema

Histologic findings in SIDS, Borderline SIDS and Explained infant death

The autopsy and clinical information do not reveal any cause of death

SIDS - lungs

Non-existant or minor changes:

Alveoli: Occasional neutrophils present, but fewer than ten alveoli contain ten cells

Interstitial tissue: Occasional lymphocytes scattered diffusely, or a few widely scattered, moderate-sized foci of infiltration (Fig 10).

Bronchi and peribronchial tissue: Mild or moderate peribronchial lymphoid infiltrates with insignificant neutrophil component; no pus cells in lumen.

Borderline SIDS

Pre-existing congenital disorders or clinical symptoms, and/or postmortem findings, which are not severe enough to explain the cause of death.

Mild changes are seen in many sections, and are present in bronchi and in peribronchial tissue, but even if prominent are not alone sufficient to explain death.

Borderline SIDS lung findings

Alveoli: Not markedly consolidated, but at least ten alveoli, each containing ten or more cells.

Interstitial changes: Moderate lymphoid infiltrates present in several sections.

Bronchi and peribronchial tissue: Neutrophil infiltration in wall, but insufficient pus in lumen to obstruct larger bronchi. Lymphoid cell infiltrates form heavy cuffs around bronchi or bronchioles in more than one section.

Explained infant death

(Explained SUDI = exclusion of SIDS)The cause of death is explained according to clinical information and/or the results of the post-mortem examination.

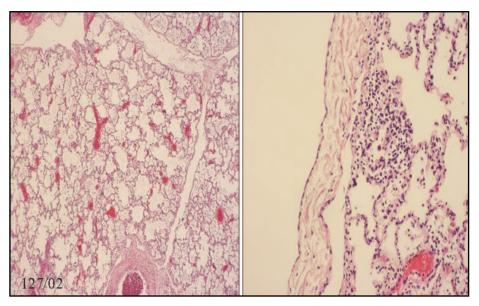


Figure 10. Pure SIDS. An 8-month-old boy found dead in the prone position, totally covered by an eiderdown duvet. Autopsy: no signs of disease or malformations.

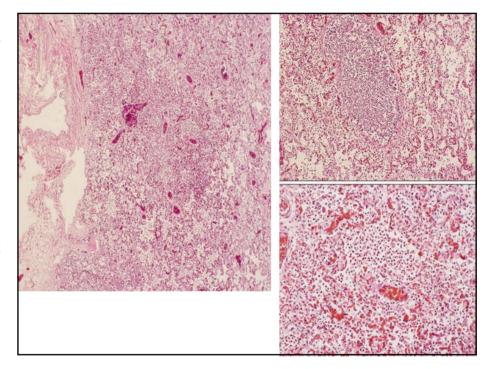


Figure 11. Not SIDS. 3 A 3-month-old girl found dead. Autopsy: severe pneumonia.

Explained infant death lung findings

Alveoli: Marked, obvious pulmonary consolidation (Fig 11).

Interstitial tissue: Widespread lymphoid infiltrates in the walls of the alveoli in all sections (interstitial pneuBronchi and peribronchial tissue: Purulent exudate fills lobar bronchi or larger branches of bronchial tree, atelectasis distant to obstruction.

In addition: Specific lung diseases that can cause death (congenital, allergic and inflammatory; also bronchiolitis if obstructive).

V) Heart lesions

Å Vege, P Råsten-Almqvist, HF Krous

The revised Nordic exclusion criteria for heart pathology in SIDS includes mandatory histological sections from the sino-atrial and atrioventricular nodes as well as frozen sections for further investigation if indicated. Family history of long QT-syndrome gives a borderline SIDS diagnosis. Heart weight twice the expected normal size is considered as a non-SIDS criterion.

In the heart session there were three presentations;

- 1. Petra Råsten-Almqvist presented exclusion criteria based on the Nordic SIDS criteria, and added new recommendations (31).
- 2. Henry Krous used the Dallas criteria (32) as a basis for his presentation and recommended that a diagnosis of myocarditis continue to require the presence of both inflammation and myocardial necrosis.
- 3. Åshild Vege summarized the two presentations, following which she moderated a discussion resulting in the following consensus regarding the gross heart description, tissues preservation, and microscopic slide selection and criteria.

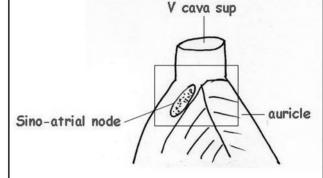


Figure 12. Serial sectioning from the right atrium with the sinoatrial node.

General processing of the heart:

A. Gross description:

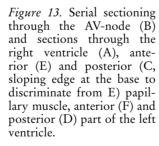
- Record body length, body weight and age
- Record heart weight
- Record ventricular thickness and valve circumferences
- Compare observed to expected values (see references)
- Describe cardiac and great vessel anatomy specifically noting abnormalities
- Freeze sample of left ventricle 70° C for immunohistochemistry, PCR, genetic testing etc if indicated.
- Fix remaining heart in 10% buffered formalin

B. Microscopic examinations

- Sections of SA and AV conducting system (Figs. 12 and 13)
- Sections according to the NORD SIDS criteria
- H&E staining
- Other staining if indicated

New criteria 1. Pure SIDS

Cases without any changes or cases with < 15 lymphocytes (10 HPF, using a x 40 objective) in one section in the absence of myocyte necrosis. Interstitial fibrosis, focal hemorrhages without inflammatory reaction and mild endocardial fibrosis are allowable.



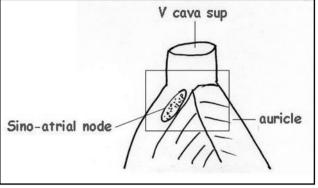
2. Borderline SIDS

Cases with changes that might be associated with functional derangements, but cannot explain death per se, are allowed. They are possible contributing factors, and should therefore be noted on the death certificate as such. Examples are the finding of < 15 lymphocytes in the myocardium – without myocyte necrosis - in one or several sections (Fig. 14a and b), focal hemorrhages with or without inflammatory reaction, patent ductus arteriosum (Botalli) after the first month, minor cardiovascular malformations such as a small VSD and ASD, and limited and uncertain changes mentioned under non-SIDS cases.

A family history of sudden death or lethal arrhythmias, such as long QT-syndrome and maternal collagen disease associated with congenital AV block III 'rd degree and/or myocarditis are also allowable.

3. Non-SIDS

Changes that undoubtedly could be fatal, or that in combination with alterations in other organs could substantially contribute to death. These include malformations, particularly obstructive left heart lesions such as



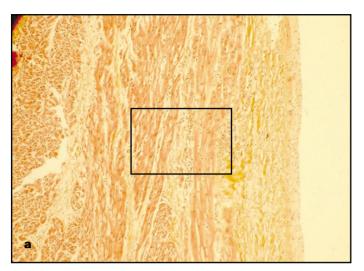
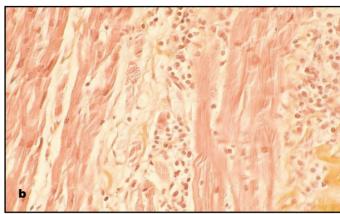


Figure 14 a,b. a) Inflammatory cells in the ventricular septum, with uncertain necrosis. No other areas with inflammation detected. (H&E with safranin x 100).



b) Higher magnification x 400.

mitral and aortic valve stenoses or atresia and coarctation of the aorta, VSD associated with ventricular hypertrophy and pulmonary hypertensive arteriopathy, myocardial tumors, myocarditis (Fig. 15), cardiomyopathies, Kawasaki disease, abnormalities of the coronary arteries, endocardial fibroelastosis, abnormalities of the conducting system, aneurysms of the membranous septum and metabolic diseases. Myocardial hypertrophy wherein the heart weight exceeds twice the expected normal weight is considered a cause of death absent other lethal lesions.

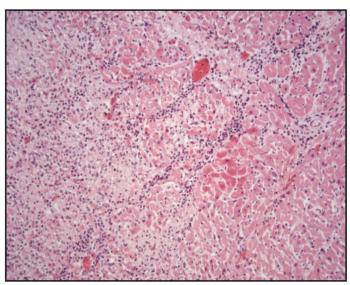


Figure 15. Myocarditis (H&E x 200).

VI) Metabolic and genetic investigation

M Arnestad, SH Opdal, RW Byard, Å Vege, JV Jørgensen, J Banner, T Bajanowski

Several sudden infant deaths in one family may either be due to infanticide or to an inherited predisposition for SIDS. This is a challenge to forensic and paediatric pathologists. New methods have made metabolic and genetic investigations easier. The new proposed criteria state that metabolic and genetic testing should be performed when indicated by history or autopsy findings, and that material should be routinely secured for further testing.

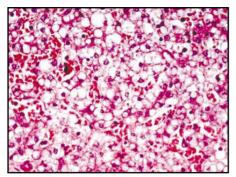
Metabolic and genetic disease can cause sudden unexpected death in infancy and early childhood (SUDI), but have previously not been part of the Nordic exclusion criteria for SIDS. Few positive diagnoses are expected through metabolic/genetic investigation of SUDI cases, also interpretation of results can be difficult. However, SIDS is a diagnosis of exclusion and

metabolic/genetic investigation can reveal the cause of death in a few cases. Metabolic disease is most often suspected from family history or autopsy findings such as fatty deposits in liver (Fig 16a,b). Genetic screening for inherited disorders, such as MCAD (Fig 17), should also be performed when indicated. However, most investigations of possible candidate genes looking for

genetic risk factors for SIDS, are still at the research stage.

In the session discussing metabolic and genetic findings seven presentations were given:

1. Roger Byard reviewed metabolic diseases and SUDI. A myriad of inborn errors of metabolism can cause sudden unexpected death in



H&I

Oil Red O

Figure 16 a, b. Liver tissue with fatty deposits suspicious of metabolic disease.
a) H & E stain. b) Oil Red O stain.

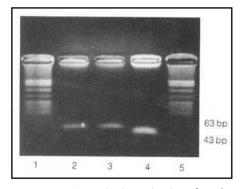


Figure 17. Genetic investigation for the MCAD A985G mutation. Lane 2 and 3 are controls without mutations. Lane 4 shows a patient who is homozygous for the A985G mutation.

infancy and childhood including fatty acid oxidation disorders, carbohydrate disorders, amino acid disorders, urea cycle disorders, organic acid disorders and others. Metabolic problems can be suspected from a family history of similar sudden death, cerebral swelling, dysmorphism, enlargement of the liver, spleen and/or heart, pallor of the liver, heart and/or muscles and fatty change in the liver, heart, muscle and/or kidney. The number of SUDIs caused by metabolic disease is thought to be low (1-2%).

- 2. Åshild Vege discussed the performance of histochemical staining of muscle. Primary metabolic myopathy can be detected when muscle is part of the routine investigation.
- 3. Jens Jørgensen presented possibilities and limitations of metabolic screening using Tandem Mass Spectrometry (MS/MS)(33). Amino, organic-, and fatty acid disorders as well as carnitine defects can be detected from blood spots.
- 4. Jytte Banner discussed post-mortem screening. One should always consider metabolic (34) and other genetic disorders in SUDI by looking for clues in the routine investigation, screen suspected cases and secure material for further testing. Their experience with post-mortem screening using achilles tendon biopsies (35) and blood spots is positive.
- 5. Siri Opdal described the state of current knowledge of genes investigated in SIDS. These include the genes MCAD (36), complement component C4, IL-10, serotonin transporter, HLA-DR and mitochondrial(mt) DNA (37), as well as genes involved in fatty acid oxidation (FAO) disor-

ders, glucose metabolism, thermal regulation, cardiac ion channels and thombosis (37). SIDS may be due to undiagnosed inherited disease and multiple "genetic risk factors" are probably involved. However no "SIDS gene" has so far been identified.

- 6. Thomas Bajanowski introduced the significance of the long QT Syndrome (LQTS) in SIDS. LQTS probably constitutes a factor in less than 5% of cases.
- 7. Marianne Arnestad summarised the presentations and moderated the discussion. A minimum requirement for investigation of metabolic and genetic disorders was presented and agreed upon.

Minimum requirements for metabolic and genetic investigation:

- 1. Detailed family history
- 2. Full autopsy including:
 - gross pathology and microscopic investigation of all internal organs
 - frozen section of liver, heart, muscle
 - neuropathology
- 3. Screening/further investigation if indicated
- 4. Secure material for further testing in each case:
 - blood/spleen (DNA)
 - fibroblast culture (chromosomal analysis etc,)
 - blood/bile spots (metabolic screening)

VII) Autopsy protocols for diagnostic purposes

Extensiveness of sampling - interpretation of findings

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The international autopsy protocol, the German protocol, the CESDI protocol as well as the Nordic protocol were

presented. A great challenge was to find the interface between extensiveness and applicability. All samples and tests necessary to establish a correct diagnosis must be included. Furthermore, samples that can be kept for

Table 5. Synopsis of the extensiveness of different protocols (no=n, yes=y).

	Nordic	Int	Gp	CESDI
Death scene	n	y	y	у
Photos	n	y	y	y
Bacteriology		varying	•	y
Virology		varying		y
Blood filter paper	y	у	y	n
X-ray		varying	•	y
Vitreous humour	n	у	n	y
Frozen sections	y	y	y	y
Toxicology	n	у	y	v

future investigations and research purposes should be included if possible. A synopsis of the four different protocols showed that the Nordic protocol was to a certain degree outdated since death scene investigation, photos and toxicology were not mandatory (Table 5).

The German protocol was possibly the most extensive one followed by the international autopsy protocol. The CESDI protocol was based on evidence emanating from the previous CESDI studies and in camparison to other protocols appears to be the least extensive but includes the major recommended ancillary investigationes. The importance of including investigations like bacteriology, virology and radiology was stressed by Arnestad who presented preliminary results from an ongoing study on the impact of different diagnostic tools. Bacteriology, virology and radiology were of diagnostic significance in 11 out of 35 cases of borderline-SIDS and non-SIDS cases (Fig 18 a,b).

Discussion and further development

The meeting decided to support the use of the international autopsy protocol. It has a modern design and is, in spite of its length, easy to use. The preliminary consensus document will be further discussed at the meeting in San Diego in January 2004.

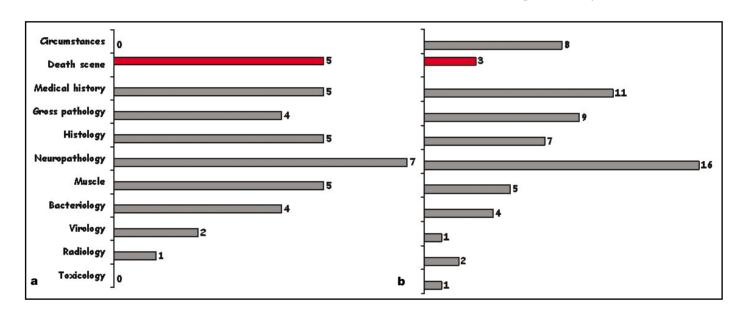


Figure 18 a,b. Diagnostic steps that contributed to the diagnosis. a) Borderline-SIDS cases n=15, b) Non-SIDS cases n=20.

FINAL CONCLUSION

The Soria Moria meeting of 2003 reached consensus concerning exclusion criteria for SIDS with regard to heart pathology, lung pathology as well as for genetic/metabolic investigations. The diagnostic exclusion criteria for the brain will hopefully be approved and a revised international autopsy protocol is around the corner. More work is needed for standardisation of observations during death scene investigation and concerning history and circumstances of death.

A meeting in San Diego in January 2004 will hopefully clarify issues regarding the definition (38-44) and autopsy protocol. A work shop during the SIDS International conference in Edmonton, Canada, July 2004 will address child abuse and other paediatric forensic issues. The Soria Moria meeting for 2004 is planned for October 2004. It will further address the development of standardisation of diagnostic criteria. Qualitative studies of death scenes may be an important issue to generate new hypotheses concerning risk factors.

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