

SPECIAL REPORT

Learning to Communicate with a SIDS Establishment that Denies the Cause of Sudden Infant Deaths

HANNES KAPUSTE Dr. Med.

Institute for Research in Professional Education, Hufnagelstr. 1, München D-80686, Germany

Abstract

A high incidence, and most of the features, of 'cot deaths' were described 50 years ago. While avoidance of prone sleeping led to a significant reduction of SIDS incidence it remained the most frequent post-perinatal death. The consensus of the establishment is that the cause(s) is/are not understood. The toxic gas theory by Richardson, however, renders this consensus incorrect. While it (1) was based on definite experimental evidence, (2) could explain practically all of the features associated with SIDS, and (3) was the basis for the first significant decline of SIDS incidence in England and Wales, the establishment did not accept the evidence, in particular after two Expert Groups established by the British Department of Health concluded in their Final Report of May 1998, that the toxic gas theory was unsubstantiated. Richardson's Comments, however, submitted in June 1998 which refuted these conclusions have been suppressed. The impact of this neglect has been tremendous. T. J. Sprott in New Zealand meanwhile has provided conclusive evidence that children protected by polythene covers of their mattresses will not die from SIDS. At present the SIDS establishment are launching an improved definition and diagnosis of SIDS to provide a better framework for investigations and put an end to "the literature that is beset by contradictions and unsubstantiated conclusions". Looking forward now to how long it may take until an effective means of SIDS prevention will be accepted, a look into Kuhn's The Structure of Scientific Revolutions may help to begin effective professional communication that can bring about the necessary change to save babies' lives. To improve the tedious expert-expert interaction in professional journals one may use the modern means of communication, coordinate reader-reader and reader-author interaction by email to gain control of the biased establishment. Experience shows that individual efforts cannot overcome their powerful defence. In conclusion, it will take a coordinated activity of motivated readers who are well established in their local communities using the means of modern communication to promote effective national and international SIDS prevention.

Keywords: SIDS, sudden infant death, causation, toxic gases, mattress, prevention, British Government, coordinated activity via email communication.

INTRODUCTION

Fifty years have elapsed since an increasing incidence of unexplained infant deaths, "every year at least 600 babies die in England and Wales", prompted A. M. Barrett to propose that *unexpected deaths in sleeping quarters of apparently healthy infants* should be described as *cot deaths* [1]. It has been 35 years since J. B. Beckwith at the Second International

Conference on Causes of Sudden Death in Infants in 1969 proposed the name and definition of the *Sudden Infant Death Syndrome* or *SIDS* [2] which was then adopted as code 798 (now R95) of the International Classification of Disease, the only context in which a pathologist can give a certifiable cause of death in the absence of finding an explanation, pathological or otherwise. The term played an important role by providing support to grieving families, diminishing the guilt and blame characteristic of these deaths, and focusing attention on a major category of post-neonatal infant death.

The efforts of researchers to find the cause(s) and the means of preventing these tragic deaths increased steadily from 39 references in 1970 to a peak of 390 in 1992 (correlating with the increase of studies on risk factors), levelling since at 300–350 references in PubMed per year. Incidentally, the entry *Sudden Infant Death Syndrome* yielded only 74% of more than 8100 relevant references found by entering a more complex combination of terms [3]. In addition to these journal articles, more than a dozen books have been published, and reports of international conferences on sudden death in infants are published now every year. These efforts have formed a large international scientific community with many of their members having spent or continuing to spend a substantial part of their professional life on SID-research, with an established cohesive elite, comprising e.g. 42 outstanding members [4] who form a powerful consensus about what is known in medicine about the pathology, aetiology, epidemiology and possible prevention of sudden infant death.

The consensus is briefly:

- (1) As there are no positive pathological signs, SIDS has remained a diagnosis of exclusion.
- (2) There are several ‘risk factors’, the avoidance of which decreases the incidence, the most important being prone sleeping position, hyperthermia and parental smoking.
- (3) While through avoidance of risk factors there was a substantial decrease in incidence, SIDS has remained the most frequent cause of death in the post-perinatal period.
- (4) The cause(s) of death in SIDS-victims and the contribution of risk factors are not understood.

Readers of this journal who know B. A. Richardson (BAR) and his toxic gas theory since his presentation at the BSAENM/AAEM Symposium in 1990 in Buxton [5], from one of his earlier publications [6–8], from his address at the BSAENM Meeting in November 2001 and/or from his publication in this journal [9] have good reason to disagree with point 4 above, and may already be convinced that the consensus of the SIDS establishment is wrong. A good example of this is Dr Myhill’s article for parents about how “the Richardson Report into cot death elegantly explains all the facts of the cot death epidemic” [10]. T. J. Sprott’s report in this issue about the outcome of his intervention campaign in New Zealand which has put BAR’s recommendations for mattress wrapping to avoid toxic gases effectively into practice will add a new dimension to the discussion of BAR’s theory. Since the consensus of the SIDS establishment has been so far that BAR’s toxic gas hypothesis has been disproved, this establishment will now be facing a scientific revolution. As the readers of this journal are well informed, we can move on from the discussion of theory to the discussion of strategy and ask ourselves, “How can we deal with the SIDS establishment’s failure to recognize the cause of sudden infant deaths?”

THE TOXIC GAS THEORY AND THE SIDS ESTABLISHMENT

Let us review for a moment the state of SIDS research in 1989, when BAR informed the Foundation for the Study of Infant Deaths (FSID) and their Scientific Advisory Committee of the progress of his investigations, and Members of Parliament in a briefing paper, published by the Parliamentary Office for Science and Technology, and submitted a

preliminary paper to the *British Medical Journal (BMJ)*, hoping to encourage cot death researchers and pathologists to take his findings into account.

At that time the list of proposed and discredited causes of unexpected deaths of apparently healthy infants was already very long: maternal overlaying, accidental mechanical suffocation, post mortem evidence of acute ‘fulminating’ infection, internal suffocation by an enlarged thymus, hypogammaglobulinemia, hypersensitivity to cow’s milk, overwhelming viremia, hypoparathyroidism, poisoning by common household medications, apnea and laryngospasm. Thus commonly held etiologic theories had been buried, among them allergic, traumatic, endocrine, and toxic causes. Common cold viruses were generally recognized as playing some role in the majority of cases [2]. Despite the consensus among pathologists on protocols for autopsies, the frequent findings of elevated hypoxanthine levels in the vitreous humour, intrathoracic petechiae having been established, and abnormalities in brainstem and other tissue markers that suggested hypoxia and hypoxemia as part of what has been termed “the final common pathway” of SIDS, pathologists could not put forward even an idea about how all of this came about. Thus since about 1985 there has been an increasing effort by epidemiologists to determine the relevance of circumstances i.e. *risk factors*, the term introduced into the MeSH database of PubMed in 1988. Besides the relationship to sleep, which is part of the definition, the impressive distribution of age [12], male sex [12–13], season at incidence [14], preceding minor infection [2], the predominance of bottle feeding [2] and the prone position when found [2, 14–16] (which Barrett had already documented in 1953 [1]), hyperthermia [17, 18], younger maternal age [13], short intervals between pregnancies [13], gestational age of less than 40 weeks [13], prematurity [12], low birth weight [12, 13], lower socioeconomic status [13], maternal smoking [12, 13] and other risk factors had been well established by 1989. But there was no explanation offered of how all of these factors could explain sudden infant death.

Knowing that the entire scientific medical community, having spent years and thousands of attempts, could not find a cause of sudden infant deaths, T. J. Sprott, a forensic scientist consultant like Richardson, was prompted to think of a ‘non-medical’ cause. In 1986 he suggested in a New Zealand nation-wide newspaper that “cot deaths could be caused by infants being exposed to a very poisonous gas, one which does not have a very strong smell but has the effect of stopping breathing and which the infants were not so likely to come in contact with in earlier days”. Even though Sprott could not identify the gas(es), he suggested that the gas “was generated by microbiological activity on chemicals in the baby’s cot” [11]. The insidious nature of the toxic gas was finally identified by BAR, as being produced by an otherwise harmless fungus, not normally known in microbiology, and not known to be toxic by toxicologists but capable of consuming antimony, phosphorus and/or arsenic to form bio-methylated gases. The gases resulting from this interaction killed infants who slept in their beds, without leaving any sign which could be seen by pathologists. So it should not have embarrassed the medical elite that finally it was not one of them but forensic scientists (one of whom happened to know this particular fungus quite well from his specialty ‘biodeterioration’) who identified this insidious gas as the cause of SIDS.

Given the state of the art in 1989 described above, the SIDS establishment should have accepted the toxic gas theory immediately, because (1) BAR presented experimental evidence proving that *Scopulariopsis brevicaulis* was found on all of 50 mattresses on which children had died from SIDS, (2) all of the incubated samples of the infected materials produced toxic tryhydride or trialkyl gases, (3) this evidence explained practically all of the established risk factors, factors which are very difficult to explain by any other hypothesis, and (4) the explanation provided a method for preventing SIDS, either by using a new mattress for each baby or by covering the mattress with polythene sheeting (a proposition which is easy to understand, based on the toxic gas explanation).

But the SIDS establishment, for whatever reasons, did not accept the toxic gas theory, the FSID decided that since the toxic gas hypothesis was 'unproven' there was no need for parents to take any action, and the *BMJ* took no action at all. While the SIDS establishment did not react, many parents reacted to BAR's recommendations and adopted his mattress precautions when they were disseminated by the media throughout Britain in early June 1989. This was followed by a sharp decrease in the SIDS rate, the first reduction to occur [8, 9].

In this journal we need not discuss in detail what ensued in the British professional scene: a highly controversial discussion in the *Lancet*, beginning in 1990 [6], the appointment of the Turner Committee by the British Department of Health in 1990, the negative report of this committee, the 1994 Cook Report TV programme 'The Cot Death Poisonings' the appointment of the Expert Group to Investigate Cot Death Theories, known as the Limerick Committee, BAR's detailed report to the Expert Group in December 1994 [8], their first report a year later [21] with the editor of the *Lancet* shaming BAR by describing him as: "An overzealous proponent of a pet theory and a media crusader do not make a good pairing" [20], the extremely negative conclusions of the 365-page Final Report published in May 1988 [22] its short version [23], and Richardson's reaction that:

Both groups ignored my recommendations for investigations that I considered most appropriate, apparently because they feared that my hypothesis might be correct and embarrassing to the government departments involved in infant mattress controls [9].

But it should be said that in the professional discussion there is no mention of the Comments to the Final Report which BAR submitted to the Department of Health in June 1998 [19]. The result of the total neglect of BAR's Comments to the Final Report is serious, because, as I wrote in a registered letter to the Chief Medical Officer in June 2004 [19]:

Much of the alleged credibility of the Final Report is based on the allegation that is stated throughout, and in particular pages 49ff, that the Expert Group replicated Richardson's original experiments, and moreover that this "work... was conducted with his cooperation and his presence at key stages ..." that e.g. "Richardson agreed that the experiments ... in Bristol, followed his procedures" ... and that "By replicating and extending Richardson's work, it was demonstrated that his interpretation of his findings was incorrect."

In his Comments of June 1998, however, Richardson states in detail with regard to most of these statements that they are not true and he explains the manner in which many of the procedures followed by the Expert Group would result in non-detection of the true causes of SIDS, i.e. toxic gases in particular.

Meanwhile, the medical establishment has accepted the conclusion of the Limerick Report that there was no evidence to show that toxic gases are the cause of SIDS. There is, however, no other cause or causes of SIDS available which could explain, as the Toxic Gas Theory does, all the epidemiology and so called "risk factors" related to it. In particular, it explains the most important prediction that babies will not die from SIDS if they are protected from toxic gases either by sleeping on mattresses wrapped in a gas-impermeable diaphragm (e.g. polythene) or mattresses free from phosphorus, arsenic and antimony. ...

It is very surprising to me that I cannot find any mention of Richardson's Comments in the medical literature ... Now I would like to ask you definitely:

- (1) Have the Comments by Barry A. Richardson ... been distributed to the members of the Expert Group ...?
- (2) Is there an adequate reply to his Comments available?

In September I received an answer from Dr Maynard of the Department of Health:

I should first explain that the Expert Group was an ad hoc Committee established in 1994 by Sir Kenneth Calman, the CMO at the time. It was charged with producing a report on the toxic gas hypothesis. A very comprehensive final report was produced in May 1998 which was, in general, well received by the scientific community and those involved with cot death. The Committee then ceased to exist. We thus did not circulate Mr Richardson's comments of June 1998 to the Expert Group. [19]

The negative impact of the neglect of BAR's Comments on SIDS-research and -prevention has been tremendous, not so much in Great Britain, where mattress manufacturers and many parents know about the possibility of toxic gases in infant beds, but all over the world. A search through PubMed for *Sudden Infant Death AND toxic gas theory* reveals four references to a controversial discussion in the *New Zealand Medical Journal*, 1998, ending with Sylvia Limerick *et al.* presenting the claims of the "Chief Medical Officer's Expert Group to Investigate Cot Death Theories" [24] but no reference to probably the only critical comment to these claims published in peer reviewed journals, by Fitzpatrick [25]. Searching for *Sudden Infant Death AND bedding AND toxic gases* reveals two references, one to the article of Warnock *et al.* (which is refuted by BAR's Comments) already mentioned here [21] and the other to the exceptionally positive evaluation of BAR's and Spratt's research in the USA [26]. Searching PubMed comprehensively for BAR's theory will lead to about 100 references and a rather time-consuming evaluation with controversial results [27], not of the kind, however, which Lady Limerick likes to claim, that "The Expert Group investigated the toxic gas hypothesis very thoroughly and found no evidence to substantiate it as a cause of SIDS" [28].

But the international SIDS establishment chose either to accept the conclusions of the Expert Group without any independent evaluation, as Byard in Australia and Krous in the USA [29], Kurz, Kenner and Kerbl in Austria [30], and Poets and Jorch in Germany [31], or not to mention the toxic gas theory at all, as Rognum in Norway and the authors at the Third SIDS International Conference [32], the authors at the 10th Congress of ESPID, the European Society for the Study and Prevention of Infant Death in Oslo 2003 [33] and the authors at the 8th SIDS International Conference in Edmonton 2004 [34]. So when we consider the scientific revolution ahead of us, we are dealing with the extraordinary influence which the vested interests of the British Government had on this powerful consensus of the SIDS establishment not to recognize the cause of sudden infant deaths, i.e. toxic gases.

THE NEW INITIATIVE TOWARDS AN IMPROVED DEFINITION AND DIAGNOSIS OF SUDDEN INFANT DEATHS

There is a new development in SIDS-research to be observed. Based on a new initiative of J. B. Beckwith in March 2003 to improve the definition of SIDS [35] and the invited critiques of six renowned SIDS-researchers [36], a meeting of an invited panel of experts was held in January 2004 in San Diego, including paediatric and forensic pathologists, and paediatricians, all of whom had extensive experience with sudden infant deaths. For administrative and vital statistics purposes the panel developed a new general definition of SIDS as the "sudden unexpected death of an infant < 1 year of age, with onset of the fatal episode apparently occurring during sleep, that remains unexplained after thorough investigation including performance of a complete autopsy and review of the circumstances of death and the clinical history, and then stratified it into *Category IA SIDS* (completely documented), *Category I B SIDS* (incompletely documented), *Category II SIDS* (certain Category I-criteria missing), *Unclassified Sudden Infant Death* and *Postresuscitation Cases*

to facilitate research into sudden infant death. It is anticipated that these new definitions will be modified in the future to accommodate a new understanding of SIDS and sudden infant death [37]. In July 2004 members of the panel continued their efforts in promoting the new 'SIDS Definition and Diagnostic Criteria' at the 8th SIDS International Conference in Edmonton, Canada [34], so the new development seems to be well on its way.

This was, however, not the first initiative of Beckwith to improve the 1969 definition, of which he recently observed: "If a prize were offered for the poorest definition of a disease or disorder in the scientific literature, this one would be a strong contender" [35]. At the 1969 meeting he had argued that a narrower age distribution should be part of the definition and for the inclusion of apparent or presumed onset of the lethal event during sleep. In 1989, Beckwith proposed this again, including the distinction between typical and non-typical SIDS cases for the purpose of enhancing the quality of research reports. He was "profoundly disappointed that so little improvement in the definition had resulted from 20 years of intensive research" under the original definition [35]. At the 1992 SIDS International Meeting in Sydney he again proposed stratification of the definition to enable separation of cases in typical and atypical groups. The proposal was not accepted at the time, although others subsequently supported sub-classification. Thus Beckwith was not alone during the last decade in his promotion of a better definition of sudden infant death. One can find a number of contributions of prominent members of the SIDS establishment supporting his view in various journals [38], including that of Byard and Krous who summarized this development:

The diagnosis of causes of sudden infant death is an often complex and difficult process. Variable standards of autopsy practice and the use of different definitions for entities such as sudden infant death syndrome (SIDS) have also contributed to confusion and discrepancies. For example, the term SIDS has been used when the requirements of standard definitions have not been fulfilled. In an attempt to correct this situation recent initiatives have been undertaken to stratify cases of unexpected infant death and to institute protocols that provide frameworks for investigations. However, if research is to be meaningful, researchers must be scrupulous in assessing how extensively cases have been investigated and how closely cases fit with internationally recognized definitions and standards. Unless this approach is adopted, evaluation of research findings in SIDS will be difficult and the literature will continue to be beset by contradictions and unsubstantiated conclusions. [39]

It took the eminent J. B. Beckwith 35 years to convince his fellow pathologists to begin to adopt a better definition and stratification of their diagnoses of sudden infant deaths, so that the SIDS-epidemiologists may begin to collect better data. Looking forward from today, how many infant deaths will it take until the combined elites in the fields of paediatric pathology and epidemiology are able to look beyond what they presently recognize to be relevant risk factors, to finally be able to identify the cause[s] of sudden infant deaths is already enough to make one despair of the communicational quality in professional medicine. The despair in this perspective is much increased, however, when we consider four additional problems that will come into play:

- (1) The members of the SIDS establishment will not find what they are looking for: the undiscovered cause(s) of SIDS.
- (2) The causes of SIDS and the effective means of prevention have been discovered long ago but disregarded by the elite.
- (3) The people who understand the cause of SIDS and have devised a means of prevention are not the respected insiders.

- (4) To find the cause of SIDS they will necessarily have to abandon their consent-orientation and pass through a phase of deliberate dissent.

To analyze these tremendous problems further let us take a look into the history of clinical ecology and the structure of scientific revolutions.

CLINICAL ECOLOGY AND THE STRUCTURE OF SCIENTIFIC REVOLUTIONS

Clinical ecologists know from their own history that it may easily take longer than a lifetime until mainstream medicine accepts a reasonable concept, which is not, for one reason or another, within their usual frame of reference. From Hare (1905) until Brostoff and Challacombe (2002), one can easily list 25 good examples of this [40], many more that resemble e.g. J. B. Beckwith's disappointing experience described above, and much experience with the fact that a medical expert who is an established member of a cohesive group will perceive and experience great difficulties when beginning to deviate from the consensus. Clinical ecologists will also understand very well that while SIDS is definitely an environmental problem it is out of the reach of the members of their societies. This is because these children die before any physician, much less an environmental one, may be called to their attention—and the paediatricians and forensic pathologists who finally will come into play and determine the state of the art regarding SIDS in medicine are not members of their societies and are difficult to reach.

So the question arises as to how members of the societies for environmental medicine who wish to take an interest in the prevention of sudden infant deaths can help individual members of the SIDS establishment to understand that, despite their basically reasonable new initiative to develop an improved definitional and diagnostic approach to sudden infant deaths, they are nevertheless about to enter a very long dead-end road if they do not recognize toxic gases to be the cause of SIDS. It may be helpful if I divert to the philosophy of science perspective on the structure of scientific revolutions, as described by Thomas Kuhn [41].

The fact that the aetiology and the cause of sudden infant deaths have not been discovered for so many years in Kuhn's terminology must be seen as a *continuous crisis*. The prevailing conviction that there is no single cause for all of the instances of sudden unexpected deaths in infancy, and the belief that BAR's toxic gas explanation has been disproved, will be regarded as the *basic paradigm* on which *normal research* by the *scientific community* concerned with SIDS has been undertaken until now. Solving the problems which lead to the continuous crisis by *changing the basic paradigm* now to one cause of death, i.e. toxic gases, that can both explain the cause of death and prevent all of these deaths easily, will be termed a *scientific revolution*.

From the historical findings of Thomas Kuhn the reaction of the SIDS establishment we have described could have been expected:

Scientists tend not to accept a new scientific paradigm if they had a prominent role in supporting the former one. There will be some scientists who can be persuaded to change their minds, more likely those who are younger and new to the field ... Probably the single most prevalent claim advanced by the proponents of a new paradigm is that they can solve the problems that led the old one to a crisis. ... particularly persuasive arguments can be developed if the new paradigm permits the prediction of phenomena that had been entirely unsuspected while the old one prevailed ... because scientists are reasonable people, one or another argument will ultimately persuade many of them. ... what occurs is an increasing shift in the distribution of professional allegiances, ... At the start a new candidate for paradigm may have few supporters, ... if they are competent, they will improve it, ... the number and strength of the persuasive arguments in its favor will increase.

More scientists will then be converted, ... will adopt the new mode of practicing normal science, until at last only a few elderly hold-outs remain. [41]

One must bring about a relevant change in the SIDS establishment in order to save babies' lives. Let us look back again at our experience so far: since Hare's time there have been about 100 years of unsuccessful attempts to have the treatment of food allergy accepted by mainstream medicine; since Barrett there have been 50 years of pathologists not coming to grips with the definition of what they call 'cot death'; since Beckwith there have been 35 years until his proposals of definition and diagnosis have been accepted; 16 years since Richardson's unsuccessful attempts to get mattress precautions accepted by the SIDS establishment as an obligatory preventive suggestion to parents; about 7 years that the definite and significant success of T. J. Sprott's prevention campaign in New Zealand has been denied. There is also the general historical fact reported by Thomas Kuhn that a scientific revolution, as he terms it, is difficult to achieve and in fact may take rather a long time to resolve. So there are not very good prospects for progress with cot death, unless, as I say above, one can bring about a relevant change.

Before we begin to discuss this option let us look at two examples, so as not to suggest that we are dealing with general human fate or experience uncharacteristic only for communication in science and in professional medicine. If we compare, for example, the progress made in the prevention of cot death in medicine with that in the photocopy industry during the last 50 years, it immediately becomes evident that there must be a significant difference between these two areas of human enterprise. This is certainly not in this case because the prevention of cot death by professional medicine is more difficult to achieve than the progress which industrial engineering has provided for us when we copy, for example, the latest article of Byard and Krous in a public library. The other comparison is with the military. How long would it take, for example, from the day the US government was told that the most dangerous terrorist was present in a particular house far away from the US until this house was destroyed? Compare this to the government being told that the most dangerous combination of fungi and fire retardants was present in the majority of babies' cots in the US until the government would announce that parents had better cover the mattresses of their babies with a sheet of polythene. As in the first case there would probably be only a short discussion, in the latter, however, a very long one. We are not dealing here with problems typical of human nature but with decisional problems in particular areas of human enterprise. So let us discuss how to begin an effective professional communication in medicine 'for baby's sake'.

THE CONTROL OF EXPERT-BIAS AND WASTE OF TIME IN PROFESSIONAL MEDICAL COMMUNICATION

While discussing the problems of professional medical communication one must keep in mind the immediate goal of parents: preventing the sudden death of their baby by applying mattress precautions. While BAR's recommendations were well on their way to becoming public opinion, Great Britain mounted an 'Expert' intervention that had great influence on the SIDS establishment and public opinion all over the world. One must beware of a counterproductive emphasis on placing blame on individuals "who had a prominent role in supporting" this intervention. Kuhn's history of scientific revolutions should remind us to get away from the tedious expert-expert interaction in professional journals by taking a shortcut using the modern means of communication to coordinate professional reader-reader and reader-author interaction to better control the establishment.

The preliminary proposal must be that:

- (1) Professional readers select articles which indicate that their author(s) fail(s) to realize the cause of SIDS.
- (2) Use reader–author-email interaction to remind authors of the cause of SIDS and find experts who appear to be sincere.
- (3) Coordinate professional reader–reader-email interaction to select a number of authors to be approached.
- (4) Use multiple reader–author-email interaction to persuade the selected SIDS-experts to accept the toxic gas theory.
- (5) Coordinate our efforts to initiate public information by the media, professional information of individual parents and new research on the toxic gas theory of sudden infant death.

Returning now to the general problem of expert-bias in professional medical communication, one must realize that certain means, e.g. privileged communication, co-option to professional societies, alienation of individuals and influence of funding organizations, can add up to the establishment of a consensus that is contrary to truth. And when looking into how the wrong consensus about the toxic gas theory has come about and how the communication in the SIDS establishment is presently operating, one can see that communication is actually working in this way. From this understanding one should be able to derive an optimal way to participate in this communication in order to speed up the process of changing the basic paradigm on the cause of SIDS along the lines of the resolution of revolutions as described by Thomas Kuhn.

There is no doubt that the faulty consensus on the cause(s) of SIDS was initiated in the UK by two committees established and funded by the Department of Health, when it was clear that the British Government had a vested interest in establishing that there was no evidence for Richardson's toxic gas theory: in 1990 the Turner Committee and in 1994 the Limerick Committee. Their reports and their most prominent members, namely Lady Limerick and P. J. Fleming, then had a decisive influence on publications, on new research and on the consensus of national and international societies [42], in particular ESPID [33, 43] and SIDS [34, 44–45]. The international consensus spread into the medical journals [46] determining the decisions of their editors and reviewers. The influential SIDS-societies usually accept new members only by co-option, e.g. ESPID [33]. That makes it difficult to participate in this privileged communication if one has deviant views. Attempts to communicate individually with members of the SIDS establishment are typically met with alienation [47]. The SIDS establishment appears to be a closed society that has developed an optimal means of defence and will not allow attempts to approach it directly with views that deviate from their basic consensus. But while this does constitute a very powerful influence in a false direction, one must not believe that the resulting tragedy, the death of many thousands of healthy children, has been intended by anyone. Psychology does not work like that, as "the path to hell may be paved with good intentions". The more a minor scientific error is based on the best of motives, e.g. "fire retardants will save a few lives", the more terrible may be its consequences, because the personal need for a good conscience in the actor may prevent him from recognizing the error. And if the consequences of a minor error—as in this case—add up to such a tragic dimension that the innocent actor cannot face them and will, by not facing them, lose his innocence more and more, one had probably better not try to intrude into the explosive atmosphere of a meeting of partly innocent believers and partly intelligent people acting finally only to save face.

So the intention must be to decrease the extremely negative impact of these international organizations by selecting those of their members who appear to be honest and able to face the truth, and approach them individually, for example when they come out in the open as they periodically must with their publications, offering their email addresses for personal

correspondence. The best way to be successful with this approach has been offered by Thomas Kuhn: "Particularly persuasive arguments can be developed if the new paradigm permits the prediction of phenomena that had been entirely unsuspected while the old one prevailed." There are a few good examples for this, not understood by the SIDS establishment, that can be explained by the toxic gas theory: the higher incidence of SIDS in child care settings [48–50], in the unaccustomed or secondary prone position [51–53] and the failing autoresuscitation of gasping [54–55]. But these are only examples and as PubMed offers presently 300–350 new articles on SIDS per year [3] there will be many occasions to remind an author or an editor of the disregarded cause of SIDS, be it a member of the establishment [4] or one of the major journals [45] or not, in order to motivate them, one by one, to support Richardson's mattress precautions. As Kuhn said:

There will be some scientists who can be persuaded to change their minds, more likely those who are younger and new to the field ... because scientists are reasonable people, one or another argument will ultimately persuade many of them. ... what occurs is an increasing shift in the distribution of professional allegiances.

The situation in the SIDS parent organizations will be similar to that in the scientific establishment in that there will be emotional problems which should be recognized. For more than ten years parent organizations have not received or distributed the best information about how to prevent sudden infant deaths. In addition parents have not conducted research but have trusted their scientific advisers. It may be more emotional for parents to realize they have lost their baby because they did not know or believe in mattress precautions. So again one should not arouse emotions by discussing this in their meetings, but instead attempt to approach them sensibly when coming into individual contact with them. Contrary to the outcome for SIDS researchers, the activity of parent organizations will be much more meaningful and effective after they accept the toxic gas theory. For example, imagine if, instead of funding questionable research by authors who disregard toxic gases as the cause of SIDS, they funded the free distribution of mattress covers in a given area and published the results themselves, not only in scientific journals but also in the public media and on their homepages in the internet. There are quite a few meaningful research projects that do not need much funding or professional medical participation. They could be done at the high school level, for example presenting the official British statistics on live births and sudden infant deaths by marital status, parity and type of registration [56], or bereaved parents collecting retrospective information on the relative occurrence of cot deaths on used mattresses and discuss them, as the established researchers would not do, in relation to the toxic gas theory [57–58].

Groups and individuals with a vested interest will prevent a change of opinion in their meetings by peer pressure, privileged communication and alienation of intruders. The consensus is well established in the media, among politicians, and in the departments of health which often finance SIDS-research. So individual attempts to promote the introduction of mattress precautions by writing to experts in SIDS-research and to members of parent organizations, by writing articles for medical journals presenting the evidence provided by Richardson and Sprott [59], and by approaching the media and the departments of health with 'news' about the toxic gas theory and the effectiveness of mattress wrapping, are disregarded easily on the advice of the SIDS establishment.

It appears, therefore, that it will take more than individual efforts to promote effective SIDS prevention. So it is suggested that individual readers of professional journals who are motivated to help in preventing the tragedy of sudden infant death begin using the new media to develop a team and network approach to overcome the expert-bias that prevails in the SIDS establishment. Finding papers that demonstrate the failure of their authors to recognize toxic gases as a possible explanation of findings, the reader may begin a correspondence with the author(s) and/or the editor of the journal about SIDS prevention.

His/her evaluation of this *reader–author* and/or *reader–editor* interaction may then be shared with others, either on a private level or by use of an internet domain designed to coordinate *reader–reader* interaction as a means to help the SIDS establishment recognize the cause of SIDS and promote more reliable means of SIDS prevention.

NOTES AND REFERENCES

- [1] Barrett AM. Sudden death in infancy. In: Gairdner D, editor. *Recent advances in pediatrics*. London, 1954:301–20.
- [2] Bergman AG, Beckwith JB, Ray CG, (eds.). *Sudden Infant Death Syndrome*. Seattle and London, Univ. of Washington Press, 1970:14–22.
- [3] Web search: 20 August 2004 – 6,063 references for ‘Sudden Infant Death Syndrome or SIDS’, 8,109 references for ‘Sudden death and Infant or Sudden Infant Death or SIDS or SID or Cot Death or Crib death or Death Cot or Death Crib or sudden unexpected death infancy’.
- [4] SIDS experts: Bajanowski T, Barness LA, Beal S, Beckwith JB, Becroft DM, Bentele KH, Bergman AB, Berry PJ, Blair P, Brooke H, Byard RW, Corey T, Cutz E, de Jonge, Emery JL, Fleming P, Guntheroth WG, Haas JE, Hanzlick R, Hauck F, Helweg-Larsen K, Houstek J, Huber J, Hunt CE, Irgens LM, Kahn A, Keens TG, Kenner T, Kerbl R, Krous HF, Kurz R, Limerick S, Mitchell EA, Nishida H, Peterson DR, Poets CF, Rambaud C, Rognum TO, Taylor E, Thach B, Tonkin S, Valdes-Dapena M.
- [5] Richardson, B. Cot mattress biodeterioration and the Sudden Infant Death Syndrome. British Society for Allergy and Environmental Medicine, British Society for Nutritional Medicine, American Academy of Environmental Medicine. Third International Symposium: Food and Environmental Factors in Human Disease, Buxton, UK, 3–6 July 1990.
- [6] Richardson BA. Cot mattress biodeterioration and SIDS. *Lancet* 1990; 335(8690): 670.
- [7] Richardson BA. Mattress biodeterioration and toxic gas generation: a possible cause of sudden infant death. *Envir Med* 1991; 8: 1–9.
- [8] Richardson BA. Cot mattress biodeterioration and toxic gas generation: A possible cause of Sudden Infant Death Syndrome. December 1994. In: Limerick S (Chairman). *Final Report of the Expert Group to investigate Cot Death Theories: Toxic Gas Hypothesis*. Department of Health, London, England; May 1998 (ISBN 1851839 874 6, out of print).
- [9] Richardson BA. Progress in reducing cot deaths since 1988. *J Nutr Envir Med* 2002; 12: 113–15.
- [10] Myhill S. Cot death – what every parent needs to know. <http://www.drmyhill.co.uk/article.cfm?id=198>.
- [11] Sprott J. Cot death theory worthy of airing. *Sunday Star*, 20 April 1986, Auckland, New Zealand.
- [12] Krous HF. Sudden infant death syndrome: Pathology and pathophysiology. *Pathol Annu* 1984; 19 Pt 1: 1–14.
- [13] Lewak N, van den Berg BJ, Beckwith JB. Sudden infant death syndrome risk factors. Prospective data review. *Clin Pediatr* 1979; 18: 404–11.
- [14] Tonkin SL. Epidemiology of cot deaths in Auckland. *N Z Med J* 1986; 99(801): 324–6.
- [15] Senecal J, Roussey M, Defawe G, Delahaye M, Piquemal B. [Prone position and unexpected sudden infant death] *Arch Fr Pediatr* 1987; 44: 131–6. [In French].
- [16] Engelberts AC, de Jonge GA. Sleeping position and cot death. *Lancet* 1988; 2(8616): 899–900.
- [17] Stanton AN, Scott DJ, Downham MA. Is overheating a factor in some unexpected infant deaths? *Lancet* 1980; 1(8177): 1054–7.
- [18] Stanton AN. Sudden infant death. Overheating and cot death. *Lancet* 1984; 2(8413): 1199–201.
- [19] Richardson BA. Comments to the Final Report of the Expert Group to Investigate Cot Death Theories: Toxic Gas Hypothesis. St Peter Port, Guernsey. Penarth Research International Limited, June 1998:1–10; Correspondence between Dr Kapuste and the Department of Health, 28 June–10 September 2004; for copies email to: hannes.kapuste@t-online.de.
- [20] Editorial. SIDS theory: from hype to reality. *Lancet* 1995; 346: 1503.
- [21] Warnock DW, Delves HT, Campell CK *et al*. Toxic gas generation from plastic mattresses and sudden infant death syndrome. *Lancet* 1995; 346(8989): 1516–20.
- [22] Limerick S (Chairman). *Final Report of the Expert Group to investigate Cot Death Theories: Toxic Gas Hypothesis*. Department of Health, London, England; May 1998 (ISBN 1851839 874 6, out of print).
- [23] Expert Group to Investigate Cot Death Theories: Toxic Gas Hypothesis (Chairman: Lady Limerick). Chairman’s Foreword, Executive Summary, Abstracts, Conclusions and Recommendations, Terms of Reference and Members of the Group, May 1998. http://sids-network.org/experts/expert_group_to_investigate_cot_.htm.
- [24] Mitchell EA, Fitzpatrick MG, Waters J. SIDS and the toxic gas theory revisited. *N Z Med*

- J 1998;111(1068):219–21; Fitzpatrick M and Joint Editors. 25 Sep;111(1074):371. Mitchell EA. 9 Oct;111(1075):395. Limerick S, Cooke M. 11 Dec;111(1079):482.
- [25] Fitzpatrick M. [no title]. *N Z Med J* 1998; 111(1079): 482–3.
- [26] Quinn JB. Baby's bedding: is it creating toxic nerve gases? *Midwifery Today Int Midwife* 2002 Spring; 61: 21–2.
- [27] Kapuste H. PubMed references to SIDS *and* toxic gases etc. For manuscript email to: hannes.kapuste@t-online.de
- [28] Limerick S. Personal communication, 28 June 2004. For copies email to: hannes.kapuste@t-online.de
- [29] Byard RW, Krous HF, eds. Sudden Infant Death Syndrome: Problems, progress and possibilities. London, Arnold Press, 2001:240–1.
- [30] Kurz R. Andere Krankheiten, pp.184–5. Kenner T, Kerbl T. Moegliche Ursachen fuer den ploetzlichen Saeglingstod. Historische und theoretische Ueberlegungen, pp.133–43. In: Kurz R, Kenner T, Poets C (Hrsg.). *Der ploetzliche Saeglingstod. Ein Ratgeber fuer Aerzte und Betroffene*. Wien, New York, Springer, 2000.
- [31] Poets CF, Jorch G. Stellungnahme zum Thema “vermeidbare Risikofaktoren fuer den ploetzlichen Saeglingstod.” *Monatsschrift Kinderheilkunde*, 2000; 11: 1065–6.
- [32] Rognum TO, ed. Sudden Infant Death Syndrome: New trends in the nineties. Oslo, Scandinavian University Press; 1995.
- [33] ESPID The European Society for the Study and Prevention of Infant Death. 10th Congress, Oslo, 27–31 May 2003. Congress Handbook. www.espid.net/events/
- [34] SIDS International Conference, Edmonton, Alberta, 2–6 July 2004. <http://www.sidsinternational.minerva.com.au/international2.htm>
- [35] Beckwith JB. Defining the sudden infant death syndrome. *Arch Pediatr Adolesc Med* 2003; 157: 286–90.
- [36] Haas JE, Krous HF, Becroft DM, Cutz E, Rognum TO, Berry PJ. *Arch Pediatr Adolesc Med* 2003; 157: 291–4.
- [37] Krous HF, Beckwith JB, Byard RW, Rognum TO, Bajanowski T, Corey T, Cutz E, Hanzlick R, Keens TG, Mitchell EA. Sudden infant death syndrome and unclassified sudden infant deaths: a definitional and diagnostic approach. *Pediatrics* 2004; 114: 234–8.
- [38] Vawter GF. *Pediatr Pathol* 1988; Emery JL. *BMJ* 1989; Cutz E *et al.* *Pediatr Pathol* 1993; Haas JE *et al.* *Pediatrics* 1993; Hanzlick R. *Arch Pathol Lab Med* 1994; Byard RW; Becker LE *et al.* *Am J Forensic Med Pathol* 1996; Rognum TO. *Acta Paediatr* 1996; Byard RW. *Med J Aust* 2001; Arnstad M *et al.* *Forensic Sci Int* 2002; Kerbl R, Einspieler C *et al.* *Wien Klin Wochenschr* 2003.
- [39] Byard R, Krous H. Research and sudden infant death syndrome: Definitions, diagnostic difficulties and discrepancies. *J Paediatr Child Health* 2004; 40: 419–21.
- [40] (1) Hare F. *The food factor in disease*. London, Longmans, vols I and II, 1905; (2) Von Pirquet C. *Allergie*. *Munch Med Wochenschr* 1906;53:1457; (3) Rowe, AH. *Food allergy*. Philadelphia: Lea & Febiger, 1931; (4) Coca AF. *Familial nonreaginic food allergy*. Springfield, Ill, Charles C Thomas, 1946; (5) Selye H. *Stress*. Montreal, 1950; (6) Williams RJ. *Biochemical individuality*. New York, 1956; (7) Rinkel HJ, Randolph TG, Zeller M: *Food allergy*. Springfield, Ill, Charles C Thomas, 1951; (8) Randolph, Theron G. *Human ecology and susceptibility to the chemical environment*. Springfield, Ill, Charles C Thomas, 1962; (9) Davies, A. *Let's get well*. London, 1966; (10) Williams RJ. *Nutrition against disease*. New York, 1971; (11) Miller JB. *Food allergy: Provocative testing and injection therapy*. Springfield, 1972; (12) Mackarness R. *Not all in the mind*. London, 1976; (13) Dickey, Lawrence D: *Clinical ecology*. Springfield, Ill, Charles C Thomas, 1976; (14) Williams, RJ, Kalita D, editors. *A physician's handbook on orthomolecular medicine*. New Canaan, CT, Keats Publ Inc., 1977; (15) Wright JV. *Dr. Wright's book of nutritional therapy*. Emmaus, 1979; (16) Randolph TG, Mos RW. *New new approach to allergies*. York, 1980; (17) Wright JV, Gaby AR. *Nutritional therapy in medical practice. Periodically taped seminars Baltimore, 1982 ... Carlisle, PA 2003*. Email: drgaby@earthlink.net; (18) Brostoff, J, Challacombe SJ. *Food allergy and intolerance*. London, Bailliere Tyn dall, 1987; (19) Davies S, Stewart A. *Nutritional medicine*. London, 1987; (20) Davies S, Downing D. *Nutritional medicine – a step in the right direction*. *J Nutr Med* 1990; 1: 3–7; (21) Rea, W. *Chemical sensitivity, IV vols*, Boca Raton, 1992–96; (22) Davies S, Downing D, Clarke S *et al.* *J Nutr Envir Med* 1995; 5: 3–4; (23) Anthony HM, Birtwistle S, Eaton KK, Maberly DJ. *Environmental medicine in medical practice*. Southampton, 1997; (24) Werbach MR *Textbook of nutritional medicine*. Tarzana, 1999; (25) Brostoff J,Challacombe SJ. *Food allergy and intolerance*. London, Bailliere Tyn dall, 2002.
- [41] Kuhn TS. *The structure of scientific revolutions*. Chicago, University of Chicago Press, 1962. [Library of Congress Catalog Card Number: 62-19621].
- [42] International SIDS Organisations: <http://www.sids.org.uk/fsid/internationalorgs.htm>
- [43] Fleming PJ. Positions and presentations at the ESPID Conference in Oslo 2003: (1) Member, International Scientific Committee; (2) Chairman, Workshop: Death Scene Investigation; (3) Author, Abstract 22, SIDS: A difficult differential diagnosis. The pediatric perspective.

- [44] Limerick S. Presentations at the SIDS Conference in Edmonton 2004: (1) Future Research Directions: Introduction; (2) Historic texts: Opening address to the 1974 International symposium.
- [45] Fleming PJ. Position and presentations at the SIDS Conference in Edmonton 2004: (1) Member, International Advisory Committee; (2) The changing profile of SUDI – the AVON experience; (3) The Avon multi-agency approach to the investigation of sudden unexpected deaths in infancy and the care of bereaved families; (4) Perspectives from epidemiology; (5) SIDS: Sudden unexpected and unexplained... but what do we now know and how can we continue to learn and help prevent future deaths? (6) The changing profile of sudden unexpected deaths in infancy. The Avon Experience; (7) The Avon multi-agency approach to the investigation of sudden unexpected deaths in infancy and the care of bereaved families; (8) Perspectives from epidemiology.
- [46] Journals and number of articles on SIDS [For entry see ref. 3]: *Pediatrics* 368, *Arch Dis Child* 254, *Lancet* 244, *Br Med J/BMJ* 203, *J Pediatr* 164, *N Z Med J* 151, *Med J Aust* 130, *Am J Forensic Med Pathol* 104, *Med Hypotheses* 99, *N Engl J Med* 89, *JAMA* 84, *Forensic Sci Int* 83, *Acta Paediatr* 82, *Early Hum Dev* 82, *Eur J Pediatr* 82.
- [47] Limerick S, CBE, MA, Hon FRCP, Hon FRCPCH. Personal communication “There is no evidence from New Zealand to support the toxic gas hypothesis. Many doctors and scientists expert in this field in the UK now refuse to respond to Sprott’s emails”. Personal communication, 19 April 2004.
- [48] de Jonge GA, Lanting CI, Brand R, Ruys JH, Semmekrot BA, van Wouwe JP. Sudden infant death syndrome in child care settings in the Netherlands. *Arch Dis Child* 2004; 89: 427–30.
- [49] Daley KC. Update on sudden infant death syndrome. *Curr Opin Pediatr* 2004; 16: 227–32.
- [50] Moon RY, Patel KM, Shaefer SJ. Sudden infant death syndrome in child care settings. *Pediatrics* 2000; 106(2 Pt 1): 295–300.
- [51] Cote A, Gerez T, Brouillette RT, Laplante S. Circumstances leading to a change to prone sleeping in sudden infant death syndrome victims. *Pediatrics* 2000; 106(6): E86.
- [52] Mitchell EA, Thach BT, Thompson JM, Williams S. Changing infants’ sleep position increases risk of sudden infant death syndrome. New Zealand Cot Death Study. *Arch Pediatr Adolesc Med* 1999; 153: 1136–41.
- [53] De Jonge GA, Engelberts AC. Sudden infant death syndrome. Many infants move from position in which they are put to sleep. *BMJ* 1996; 313(7068): 1333; author reply 1333–4.
- [54] Sridhar R, Thach BT, Kelly DH, Henslee JA. Characterization of successful and failed autoresuscitation in human infants, including those dying of SIDS. *Pediatr Pulmonol* 2003; 36: 113–22.
- [55] Poets CF. Apparent life-threatening events and sudden infant death on a monitor. *Paediatr Respir Rev* 2004; 5 Suppl A: S383–6.
- [56] www.statistics.gov.uk/statbase/product.asp?vlnk=6725 Go to Health Statistics Quarterly, Autumn 1999 through 2004, search for “parity” and download Table 8.
- [57] Tappin D, Brooke H, Ecob R, Gibson A. Used infant mattresses and sudden infant death syndrome in Scotland: case-control study. *BMJ* 2002; 325(7371): 1007.
- [58] Tappin DM, Brooke H, Ecob R. Used infant mattresses, parity and sudden infant death syndrome. Abstract 15. ESPID.
- [59] Kapuste H, Sprott TJ, Richardson BA, Mitchell P. Giftige gase im kinderbett [Toxic gases in infant beds]. *Zeitschrift fuer Umweltmedizin* 10(1): 18–22, 2002.