MATTRESS BIODETERIORATION AND TOXIC GAS GENERATION: A POSSIBLE CAUSE OF SUDDEN INFANT DEATH

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ABSTRACT

Cot mattress materials become naturally infected by the fungus Scopulariopsis brevicaulis in normal use. This fungus is able to convert any phosphorus, arsenic or antimony compounds in the materials into the toxic trihydride gases phosphine, arsine, and stibine. Generation of these very toxic gases through biodeterioration of cot mattress materials obviously represents a serious threat to health, but whether death, illness or simple irritability results depends on other factors. Increased exposure to these heavier-than-air gases by sleeping in the prone or face down position and by overwrapping also causing hyperthermia and increased gas generation are two such factors. *SIDS, cot death, toxic gas, infant mortality, biodeterioration*

INTRODUCTION

Unexplained sudden infant death (cot or crib death) which accounts for a significant proportion of post neonatal mortalities in some countries, is usually attributed to sudden infant death syndrome or SIDS, as the causes remain unknown, despite extensive research. SIDS accounts for about two deaths per 1,000 live births in the United Kingdom, two to four generally in Europe and North America; and about six in New Zealand, but only about 0.3 in Hong Kong and less in Russia, China, India and Africa, and amongst infants of African or Asian origin in England and Wales.[1-6] The high death rate in New Zealand is due to an exceptional rate of about 11.5 amongst Maori infants; the rate for Caucasian infants at about 1.6, being lower than most western countries.[7] In Australia, very high rates have been reported amongst aborigines, but enquiries amongst SIDS researchers have indicated that exceptional mortality rates may be associated with locality rather than race.

Many recent studies of SIDS have concentrated on epidemiological interpretations of the circumstances of death. It was noted that there was an increase in the Netherlands from .46 per 1,000 for 1969-71 to about 1,31 since 1978.[8] The obvious explanation is that SIDS is now more widely recognized, but this increase was attributed instead to adoption of a prone or face-down sleeping position following recommendations during a pediatric conference in 1971. This hypothesis was publicized and it was recommended that the prone sleeping position should be avoided. Many parents responded to this warning and there was a decrease in sudden infant deaths as a result.[9] Hyperthermia due to overwrapping in relation to the temperature of the environment, particularly in cold winter, weather, has also been suggested as a cause of SIDS,[10-12] A recent controlled population study has confirmed that both die prone position and overwrapping are associated with SIDS.[13] It has been suggested that sleeping in the prone position presents more risk of obstruction of the upper respiratory tract.[9,14,15] Viral and bacterial infections have

also been reported, and may be associated with the prone sleeping position, but sleeping in the supine position may avoid these Problems but create others.[16-18] Infants that are subject to prolonged apnea may be particularly at risk.[16,19,20] Exposure to spores and mites from fungal infections on bedding following earlier sensitization may cause fatal asthma attacks.[21-28] However, none of these hypotheses are convincing as they are not common to all or even to a large portion of SIDS cases.

One possible explanation is that the prone position and overwrapping are exposing the infant to poisoning by accumulations of a heavier-than-air gas, the overwrapping and resulting hyperthermia perhaps increasing gas generation. Investigations by Penarth Research International Limited (PRIL) in 1988 into biodeterioration of plasticizers in industrial reinforced polyvinyl chloride (PVC) fabric had indicated that the toxic gas arsine might be generated through biodeterioration of an arsenical biocide 10,10'-oxybisphenoxyarsine (OBPA) by an arsenic-tolerant fungus or bacterium, and it was considered that arsine might be similarly generated from PVC cot mattress coverings. Scopulariopsis brevicaulis, formerly known as Penicillium brevicaule, was the most likely organism to be involved as it is a common infection in the domestic environment or substrates containing protein such as meat, cheese and milk, as well as damp wool and leather, and it is also well known that it can generate the trihydrides ammonia and arsine from nitrogen and arsenic compounds; mystery illnesses in adults and deaths in children were attributed in the past to arsine generated through infections on damp wallpaper printed with arsenical pigments such as Scheele's green and Paris green, or fixed with horse-hoof size containing white arsenic (arsenious oxide) as a rodent repellent.[29-41]

INVESTIGATIONS

Preliminary experiments

Small samples of new PVC cot mattress coverings were placed on Petri dish malt agar plates inoculated with S. brevicaulis. Growth spread from the point of inoculation in the normal pink hyphal form associated with fungus but then spread rapidly round the PVC sample. The fungus did not spread over the PVC surface but it was observed that the edges of the samples were distorted and that the fungal growth altered around the samples to a slime form. Pieces of the affected PVC were stained in picro-aniline blue but only the distorted edges of the sample retained the stain, indicating that they had been invaded by the fungus, although few strands or structural details were visible.

When growth was well developed, small pieces of mercuric bromide (or chloride) paper were introduced over the edges of the dishes. This paper develops a yellow or orange coloration in the presence of arsine, but no color change was observed during these initial experiments; subsequent analysis of the PVC by solvent extraction and atomic absorption spectroscopy confirmed that organic arsenic cornpounds such as OBPA were not present. However, it was observed when working on these cultures that technicians suffered headache, an early Symptom of arsine poisoning. It is well known that S. brevicaulis is able to convert nitrogen and arsenic compounds into the trihydrides ammonia and arsine, and it was realized, although not previously reported, that the fungus might also be able to convert compounds of the other Group V/Vb elements phosphorous and antimony into the trihydrides phosphine and stibine which also produce headaches. These gases also complex with mercury but form white compounds so that they cannot be detected, and the experiments were therefore repeated using both silver nitrate and mercuric bromide papers. Silver nitrate paper darkens through the formation of reduced silver when exposed to reducing gases, including phosphine, arsine and stibine, **but before darkening phosphine produces a yellow color, and arsine and stibine produce a pinkish brown color; these latter gases can then be distinguished using mercuric bromide paper which will produce a yellow or orange reaction only with arsine.** All new PVC cot mattress coverings produced a strong stibine reaction, subsequent analysis of the fabrics identifying the source as antimony trioxide is included as a fire retardant, and many also produced a phosphine reaction from the phosphate plasticizers that are preferred when fire retardant properties are required. S. brevicaulis is usually described as protein tolerant and found on protein substrates, but growth is encouraged by the

presence of compounds of any of the Group V/Vb elements nitrogen, phosphorous, arsenic and antimony. Filter papers treated with the arsenical biocide OBPA at a series of concentrations encouraged growth until a threshold was reached at which the fungicidal properies began to dominate and suppress growth.

Samples of PVC fabric from the first cot mattresses received from SIDS incidents were placed on a Petri dish malt agar plates. Various domestic spoilage organisms developed but, in all cases where the samples were taken from areas affected by warmth and perspiration, a rim of buff colored slime developed around the samples that was characteristic of the slime form of S. brevicaulis. The organism seemed reluctant to spread far from the samples, but it was found that it was more active on plates prepared from malt with added nitrogen compounds, the most convenient medium for growth being 5% malt and 5% soya flour agar, a medium that was used in all subsequent experiments on naturally infected materials as it encouraged S. brevicaulis to grow away from the samples so that the presence of this fungus could be clearly identified.

Experiments on mattresses from SIDS incidents

Fifty mattresses from 45 SIDS incidents were tested between March and September 1989 by placing small samples on malt/soya Petri dish plates. Small strips of silver nitrate and mercuric bromide papers were clipped over the edges of the dishes when growth was well established to detect and identify any generated phosphine, arsine or stibine gas. Two mattresses were involved in five incidents; The mattresses included one cotton covered, 26 PVC covered, 15 PVC covered with exposed foam at one or both ends, and 8 exposed foam; exposed foam mattresses, sometimes described as 'safety' or 'vented' mattresses, are usually covered by cotton or polyester open weave fabric or net. There were 25 small mattresses from carry cots or Moses baskets and 25 larger mattresses from dropside cots; it is not known whether this distribution is normal or whether it indicates that a particular type of cot is most likely to be involved. All the mattresses, whether PVC or cotton covered or exposed foam, were found to be infected by the fungus Scopuiariopsis brevicaulis and all generated phosphine, arsine, or stibine, or mixtures of these gases, depending on their composition, from the area affected by warmth and perspiration of sleeping infants.

Bed coverings were also sometimes examined; cotton sheets covering the mattresses were always saturated with perspiration if examined promptly, and gas, generation was detected in three cots which were examined shortly after death and which were still warm. Pink stain on mattresses was always associated with phosphine generation; phosphate causes pink pigmentation in S. brevicaulis, the mattress staining in two cases forming the shape of the sleeping infant. Although most of the mattresses had been used for previous infants, five had been purchased for use by the infant that had died. Two of these mattresses had PVC coverings which were brittle, pink stained and exposed foam, pink stained and generating phosphine from phosphate plasticizers, and another mattress was exposed foam, pink stained and generating phosphine from a phosphate fire retardant treatment, suggesting that deterioration and gas generation develops more rapidly with phosphates. Two children died at less than one month old, both of them sleeping for the first time on mattresses in current use by older children. A strange arsine reaction with mercuric bromide was only seen with one sample in this group; a PVC covered mattress which had been issued by the British Army to a serving family and which contained OBPA as a tropicalizing preservative. However, faint arsenic reactions were seen with many samples, apparently associated with arsenical impurities in antimony trioxide.

Phosphine, arsine and stibine generation in retation SIDS

Since September 1989 many more mattresses have been tested from SIDS incidents and from normal situations not associated with deaths. In all cases the results have been the same; the area affected by the warmth and perspiration of the sleeping infant has been naturally infected by S. brevicaulis, and phosphine, arsine or stibine, or mixtures of these gases, have been generated, depending on the composition of the mattress materials. Whilst the generation of phosphine, arsine and stibine from mattress materials is clearly a threat to health, there must be other factors involved in SIDS as

otherwise many more infants would die. These gases are heavier than air so that infants sleeping in the prone or face down position are most at risk. Overwrapping may trap generated gas,but gas generation may be accelerated by the higher mattress temperature caused by hyperthermia in the infant.

The rate of gas generation from PVC samples was therefore studied in relation to temperature. Well established plates were placed in a desiccator in a water bath and temperatures were varied between about 18 and 44ºC, allowing at least an hour for stabilization between each temperature change. Following stabilization, the dessicator was flushed with air, a test paper introduced, and the desiccator sealed. The rate of gas generation was then assessed by the rate of development of color change in the test paper. A special sensitive test paper was prepared by treating one end of wide range pH paper with mercuric chloride; phosphine, arsine and stibine react to produce hydrochloric acid and an acid reaction, the untreated part of the paper giving an alkaline reaction if there is any interference from ammonia generation but no ammonia was detected. Hyperthermia caused an increase in mattress temperature from 37 to about 42ºC and an increase in gas generation of 10 to 20 times.

Poisoning by phosphine, arsine and stibine in adults is usually associated with erythrocyte hemolysis but this is not observed in SIDS, apparently because death occurs through some other poisoning action before hemolysis develops. Blood samples from infants who died on mattresses found to he generating only stibine were analyzed by ICP-MS by Dr. N. Ward of the Trace Element Unit University of Surrey. Antimony levels of 2.8, 4.8 and 1.9 ng/ml were found; the 2.8 ng/ml result was confirmed by neutron activation analysis. Normal levels are 0.7 to 3.0 ng/ml in adults but less than 0.85 ng/ml in infants, so that exposure to stibine from mattress deterioration seems to cause an increase in blood antimony level of about 2 ng/ml. Analysis was limited to antimony because poisoning might increase arsenic and phosphorus levels in the blood by 1 and 6 ng/ml respectively, but such increases would be undetectable with normal arsenic levels of 2 to 5 ng/ml and much higher phosphorus levels.

SIDS mortality rates are very high in certain localities in Australia and New Zealand where lamb skins are often used for cot bedding. Arsenic is deposited in the wool of sheep eating grass contaminated by arsenical soil, and S. brevicaulis, a neutral deteriorgen of wool, then generates arsine gas; stibine is similarly generated if antimony is present in the soil.

Raw wool samples from sheep reared on different soils in the south of England and Wales were tested on Petri dich malt/soya plates. No gases were detected from wool from the chalk soils of Hampshire and Willshire which are free from arsenic and antimony, but there were arsine reactions from most of the samples from Devon and south Wales. Samples from mining areas in Cornwell known to have high arsenic soils gave very strong arsine reactions.

DISCUSSION

These investigations have shown that cot mattress materials become naturally infected in use by the fungus Scopulariopsis brevicaulis. This organism is commonly found in the domestic environment, usually infecting protein substrates such as milk, cheese and meat, as well as damp wool and leather.[29-30] Mattress infections are largely confined to the area affected by the warmth and perspiration of the infant, the infection apparently encouraged by nitrogen compounds in perspiration.[42]

S. brevicaulis infection is invisible in PVC cot mattress covering because it develops in the slime form within the fabric, but the infected area can usually be detected because the PVC becomes distorted and eventually brittle as the plasticizer is destroyed. Phosphate plasticizer causes pink coloration, sometimes in the shape of the sleeping infant. S. brevicaulis also develops, usually with other organisms, on the area affected by dribble and vomit on exposed foam 'vented' mattresses, and on cotton mattress coverings.

The presence of the fungus can only be readily detected by placing small samples of mattress material on malt/soya agar plates which encourage growth to spread away from the sample. Usually the fungus develops in a buff colored slime form, although the pinkish hyphal growth that is more often associated with this fungus sometimes develops; it is important to appreciate that the buff colored slime is an alternative form of growth and not a contaminating bacterium or yeast.[30]

It is well known that S. brevicaulis is able to convert nitrogen and arsenic compounds into the trihydrides gases ammonia and arsine.[29-41] This investigation of cot mattress materials was prompted by the observation that some PVC fabrics contain the arsenical biocide OBPA (10,10'oxybisphenoxyarsine) as a plasticizer preservative, and it was considered that, if this preservative was used in cot mattress covering which became infected by an arsenic tolerant fungus such as S. brevicaulis, arsine might be produced.

PVC fabric containing OBPA was found to produce arsine gas in this way, but cot mattress materials did not normally contain OBPA. Instead it was found that S. brevicaulis infection was also able to convert phosphorus compounds such as plasticizers and fire retardants and antimony trioxide fire retardant additive into the trihydrides phosphine and stibine. Generation of phosphine and stibine had not been previously reported but it was not particularly surprising as nitrogen, phosphorus, arsenic and antimony are progressive elements in Group V/Vb of the periodic table.

Phosphine, arsine and stibine gases are 1.17, 2.68 and 4.29 times heavier than air. The threshold limit values are usually quoted as 0.3, 0.05 and 0.1 ppm respectively, about 300, 2,000 and 1,000 times more toxic than carbon monoxide which has a UV of 100 ppm. All three gases are colorless and odorless, although alkyl compounds formed in association with these trihydrides have distinctive odors which are often attributed to the trihydrides themselves. Thus the standard toxicological literature usually states that phosphine has a 'dead fish' or 'garlic' odor, arsine is more distinctly 'garlic' and stibine odor is usually described only as 'very unpleasant'; with arsine the garlic oder is usually detectable only at concentrations well in excess of the TLV.[46-53]

The detection methods that have been used for the present investigations, involving the development of colored complexes with silver and mercury, confirm only that reducing gases containing phosphorus, arsenic and antimony are present, but these methods are most sensitive to the trihydrides and the temperature used of 22 ^{o}C ensured that an antimony reaction indicated that only the trihydride stibine was present as alkyl compounds are not volatile at that temperature, and arsenic and phosphorus reactions indicated that trihydrides phosphine and arsine were most likely to be present, although theoretically the methylhydride compounds might also be present. At normal cot temperatures the methylhydride compound of antimony is also volatile as well as the dimethylhydride and ethylhydride compounds of phosphorus and arsenic. Doubts over the identity of the generated gases have arisen only because recent work on arsenic has shown that alkyl compounds are generated by biodeterioration, although it has not been shown that trihydride is not generated.[41,43-45] The distinction between trihydrides and related alkyl compounds is largely irrelevant as all the compounds are toxic with similar poisoning actions.

The typical symptoms of arsine poisoning in adults are well known.[46-53] Poisoning by phosphine and stibine is not so frequently reported and is not described in such detail in the literature, but they are exceedingly poisonous, heavier-than-air gases, with the same mode of action and symptoms as arsine; the main differences arise only through differences in density and toxicity.[49-53]

Poisoning by these gases is usually diagnosed through the erythrocyte hemolysis which develops some hours after exposure, but such symptoms are not normally associated with SIDS. If phosphine, arsine and stibine are a cause of SIDS, death must result from some other action before erythrocyte hemolysis develops, either because infant erythrocytes are more resistant to hemolysis or because infants are more susceptible to some other poisoning action; there are several reports of phosphine and arsine poisoning causing child fatalities but only illness in adults.[31-38,56] These gases are reported to cause depression of the central nervous system and suppression of respiration which would be consistent with SIDS, but it seems that they are also anticholinesterases which cause cardiac inhibition and vasodilation through progressive accumulation of acetylcholine, a mechanism that be consistent with SIDS, as it would explain certain unusual observations, such as death a few minutes after lifting the infant from the cot.[46,51]

It is difficult to confirm that an infant has absorbed sufficient phosphine, arsine or stibine to cause death. Blood samples from three infants who died on mattresses found to be generating only stibine were found to have antimony levels of about 2.8, 4.8 and 1.9 ng/ml,

compared with normal ambient levels of 0.7 to 3.0 ng/ml in adults but less than 0.85 ng/ml in infants. These results suggest that the blood antimony level was increased by about 2.0 ng/ml by stibine poisoning, although the normal maximum level of 0.85 ng/ml in infants probably includes some contribution from mattress stibine in non-fatal situations. The known comparative toxicities of arsine and phosphine would suggest that poisoning might increase arsenic and phosphorus levels in the blood by about 1 and 6 ng/ml respectively but such increases would be undetectable with normal arsenic levels of 2 and 5 ng/ml and much higher phosphorus levels. These calculations would suggest that lethal doses of phosphine, arsine and stibine in infants might be 60, 10 and 20 ng/kg body weight respectively. Mixtures of gases would probably be additive in effect because of their identical mode of action.

Phosphine, arsine and stibine will not be generated to appreciable amounts until S. brevicaulis infection is well established; there are few deaths at less than one month old, and deaths in younger infants are always associated with mattresses in current use by older children with active infection. The gases cause headache and irritability, an older active infant dislodging bedding and dispersing gas; risk of SIDS decreases at ages above about 5 months.

First children are at lower risk because they are more likely to have new mattresses in which gas generation will not develop to a significant extent at lower ages. Infants with low weight for their ages are at higher risk to a greater age as they are less likely to be able to dislodge their bedding, and use of analgesics will increase the risk by preventing the infant from detecting and reacting to the headache that is the first indication of poisoning. As phosphine, arsine, and stibine are heavier-than-air and generated from the mattress materials, infants sleeping in the prone, or face down, position are at the most risk[8,9,14,15]

Overwrapping and deep carrycots will also increase the risk by obstructing dispersal of the gases, but overwrapping in relation to the environmental conditions will also result in hyperthermia and, as S. brevicaulis is a thermo-tolerant fungus, activity and gas generation will increase sharply with the increase in temperature, greatly increasing the poisoning risk.[10-13]

Overwrapping is associated with cold weather, hyperthermia developing particularly when central heating begins to operate early in the morning.[6,10-12,57] The SIDS mortality rate can be plotted against an average temperature, but a much closer relationship can be established with 'coldness' measured in terms of domestic fuel consumption. Plots of SIDS mortality rates against domestic fuel consumption for England and Wales show a close relationship in recent years, except for the first quarter in 1985 and the last quarter in 1989 for which the SIDS rate was about 20% lower than expected. The first quarter of 1985 followed warning that overwrapping might be a cause of SIDS.[10] The last quarter of 1989 followed public warnings by the author of this paper in the summer of 1989 that cot mattress biodeterioration might be a cause of SIDS, and that a new mattress should be provided for every new ehild or an old mattress should be covered to isolate the child from gas generation. Some parents responded to these warnings and there was an increase of about 15% in purchases of new mattresses between August and December 1989. The difficulty in establishing that deaths are due to poisoning means that it is important to acknowledge the probability that deaths are caused in this way so that appropriate precautions can be adopted in manufacture of cot mattress materials.

SIDS is the most common cause of death of infants in western-style countries because it is only in these countries that synthetic and fire retardant treated cot mattress materials are used which contain compounds of the critical elements phosphorus, arsenic and antimony. SIDS is not recognized as a special problem in Russia, China, India and Africa, and in Hong Kong an intermediate situation exists. In Australia and New Zealand the SIDS rate is lower than for many other western-style countries amongst Caucasian infants, but is high amongst aborigines. These high levels seem to be associated with locality rather than race; the probable explanation being that these are communities in which lamb skins are used as cot bedding and, if the lambs have been grazing on grass contaminated with soil containing arsenic or antimony, these elements are concentrated in the wool which is then deteriorated in the cot environment by S. brevicaulis, a natural deteriorgen of wool, generating arsine or stibine gas; the localities with exceptionally high SIDS rates are those

in which the soil are known to contain unusually high levels of arsenic and antimony.[54-55]

CONCLUSION

lt is recommended that care should be taken in the long term to ensure that cot mattress materials do not contain compounds of phosphorus, arsenic or antimony. Arsenical biocides, antimony trioxide fire retardant additive and phosphate plasticizers must not be used in PVC cot mattress coverings, and fire retardants based on phosphorus or antimony compounds must not be used in foam, cotton or any other mattress materials. There will be some delay in introducing such safety precautions, particularly amongst Government bodies and manufacturers who have introduced fire retardants as a precaution without appreciating the dangers. Meanwhile it is recommended that every new child should be provided with a new mattress or, if a new mattress cannot be provided, an old mattress should be wrapped in clear polyethylene (polytene) sheet, carefully secured with adhesive tape beneath the mattress, in order to isolate the infant from the mattress deterioration and toxic gas generation.

The purpose of this paper is to explain the situation in detail in order to encourage pathologists and SIDS researchers to recognize this probable cause of death In their investigations, and to encourage the general adoption of appropriate precautions.

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