

## EDITORIAL

### Cot Death Today

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It is 16 years since my research established that natural microbial activity can generate dangerous toxic gases in an infant's sleeping environment, a discovery that prompted the development of my hypothesis on the cause of cot death or sudden infant death syndrome (SIDS), 16 years of frustration during which periods of praise for my research have alternated with determined attempts by disgruntled cot death researchers and uncaring officials to discredit me and the precautions that I have recommended in order to protect their careers and reputations. As a result, infants have continued to die unnecessarily, although the cot death rates have decreased steadily as my recommended precautions have been adopted despite this opposition. For half a century, cot death was the greatest fear of new parents, as it was the main cause of infant deaths, reaching a peak rate in England and Wales of about 2.3 per 1000 live births in 1986–88. However, in 2003 it was only about an eighth of this at 0.28 per 1000 births, and in some other previously affected communities the rate is now too low to be significant.

While there have always been some unexpected and unexplained infant deaths, it was recognized in about 1952 that the rate was increasing, prompting the adoption of the term cot death or crib death, as most of the victims died during sleep. In 1969 it was proposed that the condition should be described as SIDS, defined as the sudden death of any infant or young child, which is unexpected by history, and in which a thorough post-mortem examination fails to demonstrate an adequate cause of death. The cause of SIDS could not be identified despite intensive medical research, but in 1986 the situation attracted the attention of Dr Sprott, a consulting chemist in New Zealand who thought that SIDS might be caused by accidental poisoning and who investigated the various toxicants that an infant might encounter in the course of normal care practices.

In 1988, my own research on microbial deterioration of polyvinyl chloride (PVC) identified an additive OBPA that could be converted by various natural infections into extremely toxic gaseous arsines, and it became apparent that this gas might be generated from PVC cot mattress coverings. I soon discovered that there were various common additives, such as plasticizers, preservatives and fire retardants, in PVC that could also generate the similarly toxic gaseous phosphines and stibines, and when my findings were first publicized in 1989, Dr Sprott recognized that these were the poisons he had been trying to identify. We soon discovered that the generation of these toxic gases was not restricted to PVC cot mattress coverings and that phosphines could also be generated from woven fabric coverings treated with fire retardants containing phosphorus. Even fleeces could similarly generate arsines or stibines if the sheep had been feeding on grass contaminated by soil containing arsenic or antimony compounds.

In cases of severe acute poisoning by phosphines, arsines and stibines, one of the main diagnostic features is haemolytic damage to erythrocytes, but this damage is not seen in SIDS victims. Instead, protracted chronic exposure causes anticholinesterase poisoning. While this effect interferes with nerve synapses and causes disturbance of the central

nervous system in adults, this effect is much less obvious in infants, with their immature nervous system, and instead interferes with cardiac control by allowing choline released by the vagus to accumulate in the circulating blood through the absence of cholinesterase, the choline progressively reducing cardiac activity until the heart eventually stops. As choline moderation of cardiac activity is a normal physiological process in infants it is not identifiable on autopsy, except if it is possible to analyse blood samples for choline level immediately after death, a very difficult task. It has been established that some families are more likely to suffer cot deaths, apparently because they suffer the inherited genetic disorder long QT syndrome, which makes their infants particularly susceptible to cardiac failure through anticholinesterase poisoning.

I am particularly pleased to see two articles on cot death in this issue, one by Dr Sprott in New Zealand, who has successfully promoted mattress wrapping as a means to isolate infants from the toxic gases, and the other by Dr Kapuste in Germany, who has researched the world literature on cot death for many years. All three of us are certain that cot death can easily be eliminated if health authorities recognize simple precautions. The elimination of phosphorus, arsenic and antimony compounds from mattress and bedding materials is the most effective precaution; in the UK the elimination of arsenic and antimony from cot mattress materials is the main reason for the dramatic decrease in the cot death rate in recent years. Mattress wrapping in polyethylene sheet to isolate an infant from generated toxic gases is equally effective and must be used by families with fleeces or previously used mattresses with established microbial infections. Avoiding overheating will reduce the risk by limiting microbial activity, and avoiding the prone or face down sleeping position will reduce the risk by 30–40% by reducing exposure to generated toxic gases. While parental smoking is often suggested as the main cause of SIDS, this suggestion arises from an error in epidemiological analysis; the parents who smoke the most are in low income families who tend to re-use old mattresses in which infections are already established and the gas generation risk most severe.

Hopefully, cot death will soon be totally eliminated, except perhaps in families that insist on re-using old mattresses containing phosphorus, arsenic and antimony compounds.