Sudden infant death syndrome: What questions should we ask?


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Received 12 November 1998; accepted 24 February 1999

Abstract

A limited historical perspective can affect the questions we pose about the sudden infant death syndrome (SIDS) issue. Evidence is presented from the literature that the SIDS rate in Western countries was low prior to World War II and peaked in the 1980s. An analogy is drawn with the trends in the prevalence of some infectious diseases, and questions are posed from the perspective of a bacterial toxin hypothesis of SIDS causation. © 1999 Published by Elsevier Science B.V. All rights reserved.

Keywords: Sudden infant death; Sudden infant death syndrome rate; Bacterial toxin

Inevitably, the questions we ask about a problem influence the answers we obtain. This certainly applies to sudden infant death syndrome (SIDS), which was only defined as such in 1969 [1]. The tracing of statistics relating to sudden death of infants prior to this date is not a straightforward task and it is easy to lose historical perspective.

It is widely recognised that there has been a significant and widespread fall in SIDS rates in Western countries since 1990. These rates are now similar to those in Asian regions such as Hong Kong and Japan where, as far as can be determined, there has been less than one SIDS case per 1000 live births for at least 18 years [2]. The falling death rate in Western countries is indeed gratifying, but underlying mechanisms are still sought. While SIDS organisations have worked hard to decrease the prevalence of risk behaviours, it is simplistic to assume that the reduced SIDS rates can be attributed wholly to changes in infant-care practices such as supine sleeping and reduced heat while sleeping.

It seems that few pathologists or medical scientists have journeyed to the basement, blown off the dust, and analysed the records of the sudden deaths of infants prior to the adoption of the SIDS definition. Where this has been done, there have been some interesting results. In the USA, McLaughlin et al. [3] reviewed all the original reports of infant death cases in Olmstead county, Minnesota, from 1945 to 1992. For this period, 82 cases of SIDS were identified. The incidence increased from 0.18 per 1000 live births in 1945–49 to a peak of 2.11 in 1982–85 and subsequently decreased to 1.28 in 1990–92.

A 1986 analysis of post-neonatal deaths [4] during the period 1924–1983 was carried out in an attempt to determine whether or not the probable SIDS rate in the Dunedin region of New Zealand had changed during that period. Records of police investigations and coroners’ enquiries are available that include place of death, length of final illness and official
cause of death. This study concluded that the probable SIDS rate for that region had risen from 0.33 to 4.73 per 1000 live births between the decades 1924–33 and 1974–83, reaching a rate of 8.14 per 1000 live births in the 3-year period 1981–83.

The health district of Christchurch in New Zealand is directly to the north of Dunedin and its 1968–1983 post-neonatal mortality rate records have also been analysed [5]. During this period, the post-neonatal mortality rate increased from four to eight per 1000 live births. It was concluded that the rise reflected a greater number of SIDS cases.

A more recent example of an increase in SIDS is that of Scandinavia during the period 1978–88. In 1972–75 the SIDS rate in Denmark was 0.4 per 1000 live births but this increased to 1.9 in 1987. Norway also had a clear increase in the reported SIDS rate during this period [6]. Although there appears to be no evidence of any movement toward prone sleeping, at the present there is a significant increase in SIDS cases in Japan and Hong Kong [2].

What is the message behind these geographically diverse, retrospective studies? During the earlier part of this century, was there a genuine increase in deaths from SIDS in Western nations, peaking in the late 1980s? Have areas such as Olmstead county, and perhaps Norway [3,6], experienced this increase as fluctuations within a wave? The answers to these questions are unknown. If SIDS does indeed come in waves, it is possible that the recently observed decline in incidence may represent a temporary phenomenon rather than defeat of this scourge.

If the 1990s have seen a decline to the SIDS rate which was typical prior to the 1950s, i.e., to what may be its ‘normal’ low level, perhaps our pertinent questions should be:

1. Why was the SIDS rate so high in the 1960s through to the 1980s in the developed countries?
2. Why did that very high SIDS rate appear to be a phenomenon in these more wealthy countries with temperate climates?
3. Will the SIDS rate increase in those Asian countries that are now experiencing lifestyle changes associated with increased standards of living or an increase in the prevalence of smoking among women?
4. If the causal factor(s) of SIDS remain unidentified, might we see, as part of a cycle, a return to the very high SIDS rates of the 1960s to 1980s in developed countries as well?

There are parallels to such wave-like patterns of incidence in infectious diseases worth considering. For example, Mycoplasma pneumoniae infections tend to recur as a 5-yearly epidemic cycle [7]. Other infectious diseases have been prevalent for a number of decades and then the rate has decreased for no known reason. One example of this is seen in the rise and fall of enteropathogenic Escherichia coli infections of infants. These caused many problems with infantile gastro-enteritis in the 1940s and 1950s [8], especially in the developed countries from which they have now virtually disappeared; however, they remain a problem in the developing countries. An example of an infectious disease which has had more serious consequences in developed countries is polio.

The incidence of polio paralysis has been low amongst the people of developing countries but waves of polio epidemics have swept through the Western nations this century. The disease is actually so prevalent in Third World countries that most people become immune to the polio virus before they reach the age when a polio infection is likely to cause paralysis [9]. Thus, as standards of living rose, so did the incidence of paralysis and mortality and the awareness of polio.

Some might assume that if the fall in the SIDS rate is due to the changed practice of placing infants in the supine rather than the prone sleeping position, the increase must have been due to the practice of prone sleeping becoming prevalent. While it is true that prone sleeping became common in the 1970s in many Western countries [10], one must still account for the fact that an infant does not die because it is placed prone. The prone sleeping position is a risk factor, not a cause.

There has been increasing interest in recent years in the proposition that bacterial toxins are responsible for or at least contribute to SIDS. While no particular toxin has, as yet, been shown definitively to cause SIDS, a number of toxins have been implicated in this syndrome [11]. Most of the organisms commonly associated with SIDS in the literature are found primarily in the gut, particularly the large intestine, which is relatively impermeable to larger
molecules. Arnon et al. [12] have estimated that for a large toxin molecule such as botulinum toxin (≈150 kDa), only one molecule in about 10^{7–10^6} would be absorbed into the circulation. Various factors influence such absorption [13,14]. Clearly, therefore, any factor that causes bacterial overgrowth or that increases the probability of toxin molecules being absorbed into the systemic circulation could influence the outcome for the host. Arnon et al. suggest that exposure of infants to Clostridium botulinum in the USA is probably a fairly common event which only infrequently results in infant botulism. They recognise that other factors must contribute to the pathogenesis of this disease, a disease which shows a pattern similar to that of SIDS with respect to the age of its victims and its tendency to be less common amongst first-born infants. Perhaps this is one of the suggestions we should consider when we attempt to assess the reasons for the changing SIDS rates during this century.

Lifestyle-related factors such as diet influence the concentration and composition of the bacterial flora of adults. The dietary intake of infants affects their Gram-negative flora. Bottle-fed infants are more likely to have a higher faecal coliform count between 1 and 3 months of age [15].

Our ability to grapple effectively with the SIDS problem is directly related to the assumptions we make about this syndrome. There is real disagreement over whether to regard SIDS as a single entity or as a ‘catch-all’ label with which to describe the final results of a wide variety of processes. If the latter view is correct, then probably all we can do is to continue with reducing the risk factors. If we adopt the former stance and treat the bacterial toxin theory with the seriousness it deserves, we might regard the current, lower, SIDS rate as ‘normal’. We might then postulate: (1) that one or more causative agents of SIDS are less prevalent in the environment than they were between the 1960s and the 1980s, and/or (2) that some important risk factor (or factors) was significantly more prevalent during these years, thus magnifying the effect of the causal agent(s). Factors claimed to affect the SIDS rate such as prone sleeping position and other lifestyle-related factors might act by increasing the carriage of a toxigenic organism, increasing the amount of toxin produced by the bacterial flora, or increasing the probability that a toxin will penetrate into the systemic circulation. It is possible that the ‘agent of SIDS’ could be fairly prevalent but relatively ineffective unless another element, even another toxin, is present to help it gain access to the systemic circulation. We suggest that a clearer geographical and historical perspective on the changes in the SIDS rate may lead us to pose new questions and/or to re-interpret existing data from a fresh, more rewarding, perspective.

References


