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Review Article

Identification of Risk Factors and the Preventive Measures for Sudden Infant Death Syndrome (SIDS)

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ABSTRACT

Sudden infant death syndrome (SIDS) also known as crib death is a diagnosis that is made when an apparently healthy baby dies unexpectedly and for no clear reason. Deaths that remain unexplained even after thorough investigation, autopsy, and clinical history are classified as SIDS. An average of 133 babies have died each year over the past five years in cases where co-sleeping is a factor, according to Department for Education data. It revealed 141 children died while co-sleeping in 2017, compared with 131 in 2016, 121 in 2015, 141 in 2014 and 131 in 2013.
Sudden infant death syndrome (SIDS) occurs less frequently in the first month of life, peaks between 2 and 4 months of age, and decreases thereafter. Prone sleeping (placing an infant to sleep on his/her stomach), bed sharing, parents not sleeping in the same room as the infant, an infant not using a pacifier during sleep, overheating, and maternal smoking during pregnancy have been suggested as contributing factors for SIDS. There is no guaranteed way to prevent SIDS, but you can help your baby sleep more safely by following certain preventive measures.
In this article we focus on the identification of risk factors for SIDS based on triple risk model and prevention of risk factors for SIDS. Mothers and family members should be aware of the prevention methods for SIDS. This review is beneficial to all medical practitioners as well as mothers and family members with a newborn.

Key words: *Sudden Infant Death Syndrome (SIDS), Triple Risk Model.*

INTRODUCTION

Sudden Infant Death Syndrome (SIDS) also known as Cot death or Crib death is the most common cause of death between the ages of 1 month and 12 months. SIDS is the most common cause of death among babies at this age. The cause of SIDS is not known. There are no tests currently available that can detect an infant who will die from SIDS. Reduction of SIDS risks for populations has been achieved by providing health education on preventive measures for SIDS. SIDS is the most common of several infant deaths causes that the U.S. felt the need for Centres for Disease Control and Prevention categorizes as Sudden Infant Death Syndrome (SIDS). Modifiable factors, such as prone sleeping and tobacco exposure, facilitate hypoxia and the resultant decreased cerebral oxygenation may play a mechanistic role in SIDS. Other causes of SIDS include suffocation, infections, magnesium deficiency and poisoning. A small number of high-risk babies may require electronic monitoring of the heart rate and breathing. This practice has not been shown to protect babies, and you should be aware that monitoring doesn't help a baby breathe.

Depending on the precise definition of SIDS, the historical era, demographics, and ethnic status of the population, SIDS affects approximately 0.1 to 2 infants per 1000 live births, with a peak incidence between the ages of 2 and 5 months. The etiologic basis for SIDS can be neurologic, endocrine, metabolic, pulmonary, immune, or cardiac with a genetic basis implicated for each. [(1)]

The biggest subgroups of explained SIDS deaths were those attributable to infection (46%). The age distribution of this subgroup was similar to the explained SIDS group as a whole; most deaths occurred in the first month of life (21%) and more than half of deaths occurred after 6 months. There was a winter peak of deaths from infection, the highest number occurring in December (21%).The key findings are that the socioeconomic context of SIDS has altered, that seasonality seems to have almost disappeared, and that there is little epidemiologic difference between those infants dying suddenly of ascertained causes and those dying as SIDS, apart from different age distributions, a greater prevalence of congenital abnormalities at birth among the explained deaths, and an increased risk associated with maternal smoking during pregnancy among the SIDS. [(2)]

Epidemiology

SIDS is rare, with a reported incidence in the United States of 1.2 deaths per 100,000 children, compared to 54 deaths per 100,000 live births for SIDS. According to WHO, over 3.7 million premature deaths per annum may be attributed to the harmful effects of ambient air. [(3)]

SIDS deaths have occurred due to the following reasons: [(4)]

- Death at home, history provided: 79%
- Crib or bassinet: 54%
- Adult bed: 36%:
- Placed supine, side, prone: 10%, 2%, 3%
- Found prone: 89%
- Found face position: down, side: 10%, 8%
- Co-sleeping, sweating when found: 3%, 1%

RISK FACTORS FOR SIDS:

The risk for SIDS can be better explained by Triple risk model (Figure-1) in which the risk factors are categorised into 3 types of risks as described by Filiano and Kinney. In other words, it involves the intersection of three risks: (1) a vulnerable infant, (2) a critical developmental period in homeostatic control, and (3) an exogenous stressor(s).

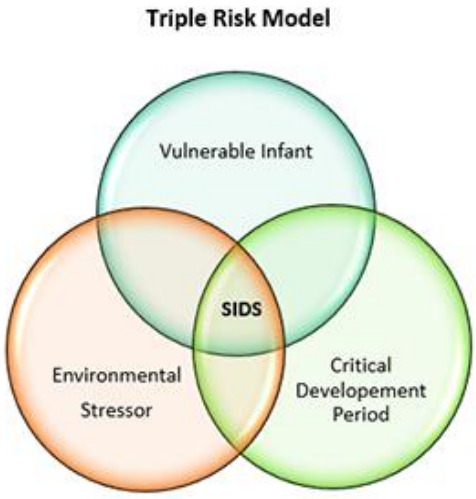

Figure 1: Triple risk model for SIDS (Sudden Infant Death Syndrome). [(5)]

The following table-1 shows the possible risk factors for SIDS (Sudden Infant Death Syndrome):

Table-1: Classification of risk factors for SIDS based on Triple Risk Model.

Vulnerable infant	Critical development period	Environmental stressors
• Preterm birth. • Maternal smoking/alcohol. • Low birth weight. • Genetic polymorphism. • Defects in brain.	• Sleeping and waking patterns. • Alterations in body temperature. • Variations blood pressure, heart rate and breathing.	• Sleep positions. • Head covering. • Co-sleeping. • Infection. • Soft-bedding. • Passive smoking. • Bottle feeding. • Magnesium deficiency. • Air-pollution. • Altitude.

Though the cause of SIDS is unknown, there are certain factors that put a baby at a greater risk of SIDS. A lot of these factors can be controlled, like poor prenatal care or the mother smoking, drinking and using drugs during pregnancy. A baby that is premature, has a low birth weight or is born to a woman under the age of 20 also is at a greater risk for SIDS. Using too much bedding, wearing sleepwear that is too heavy or sleeping on the stomach also increase the odds of SIDS.

The similarity between the epidemiologic characteristics of the unexplained and the explained deaths (many of which were attributable to infections and some to previously unrecognized congenital anomalies) is consistent with the triple risk hypothesis, in which infants compromised by prenatal or perinatal factors are at increased risk of dying if subjected to an insult at a vulnerable stage of their development. However, this hypothesis is clearly not applicable to all causes of non-SIDS postneonatal death, for example accidents. [(2)]

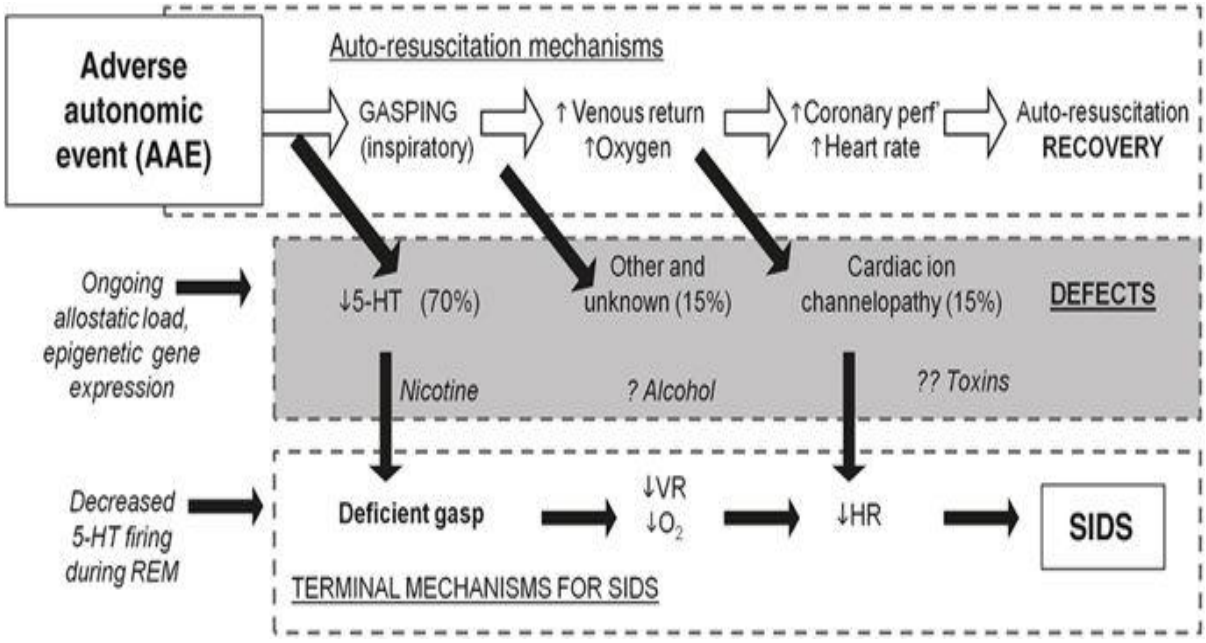

Figure-2: Terminal mechanisms involved in SIDS. [(6)]

ALTITUDE:

Altitude of residence is independently associated with an increased incidence of SIDS, specifically among infants born to mothers living at >8000 feet of altitude. It has been suggested that hypoxia may contribute to the pathophysiology of SIDS. Living at high altitude also is associated with decreased oxygenation, [(7)] and it is therefore may be that a greater incidence of hypoxia is responsible for the greater incidence of SIDS at high altitude. However, further investigation is required to understand the relationship. Additionally, it remains unclear if altitude has a linear or threshold effect. It is possible that the incidence of SIDS increases linearly with increasing altitude. [(8)]

SMOKING:

Epidemiologically it is difficult to distinguish the effect of active maternal smoking during pregnancy from involuntary postnatal exposure to smoking by the mother. The mechanism for SIDS is unknown; however, it is generally believed that the predominant effect from maternal smoking is from *in-utero* exposure of the fetus. [(9)] There is substantial evidence to conclude that maternal smoking caused a marked increased in SIDS. Schellscheidt and collaborators investigated if the association between SIDS and low birth weight may be attributed to maternal smoking. [(10)] Their data suggest that maternal smoking could account for the lower birth weight and BMI found in SIDS victims.

Objective measurements of nicotine and its metabolites in babies diagnosed as dying from SIDS indicate that significant exposure has occurred around the time of death [11,12] The high percentage of exposed infants also means that tobacco smoke exposure is a major confounder in evaluating post-mortem findings since few investigations have been stratified to take parental smoking into account. Morphological findings considered to be indicative of SIDS include decreases in the size of visceral organs. [13, 14] It delays in the normal pattern of maturation and tissue scar formation conceivably after hypoxic or ischemic insults. [15, 16] Similar abnormalities have also been described as being consequences of fetal growth retardation, [17] which in turn is frequently caused by maternal smoking.

VARIATIONS IN BREATHING, HEART RATE AND BLOOD PRESSURE:

The pathway to SIDS is complex and involves the immaturity of cardiorespiratory autonomic control, and failure of the arousal response from sleep. [18] Arousal response is critical to survival. Physiological changes during sleep activate the arousal response. Normal responses of arousal during sleep include an increase in heart rate, arterial pressure, and ventilation, as well as a behavioural response enabling body movements to avoid life-threatening stimuli. These responses are similar to cardio-respiratory responses of fight-or-flight reactions. Specific regions of the hypothalamus and brainstem play a role in the relay and integration of responses. [18] Chemoreceptors present in the carotid bodies, are coordinated with respiratory control. Studies have indicated that abnormalities of arousal are involved in SIDS. A reduced number of spontaneously caused arousals were found in infants who died from SIDS. [19-21]

MAGNESIUM DEFICIENCY:

Some data support the hypothesis that magnesium deficiency contributes to SIDS. Muscle weakness in the upper half of the body in infants and shoulder hypotonia in near-miss for SIDS infants. An infant sleeping face-down in the prone position could be jeopardized if he lacked the muscle strength to shift his position or turn his head to rescue himself from a life-threatening situation. Muscle strength is seriously impaired in the young magnesium deficient subject, while normal magnesium rapidly reverses muscle weakness. It was concluded that magnesium deficiency is at least one major unifying factor that explains increased SIDS in prone sleeping infants. [22]

LOW BIRTH WEIGHT AND WEIGHT GAIN:

A low gestational birth weight (suggesting poor intrauterine growth) is one of the risk factor for SIDS. Poor weight gain should be seen as a thread in the web of factors that render an infant vulnerable to SIDS and is both a consequence of adverse health and social conditions and a cause of poor health outcomes. Monitoring weight gain may be of particular importance among those families which are identified from other criteria as being at increased risk for SIDS, and provides a basis for generating both epidemiological and physiological research questions with important public health implications. [23]

Poor postnatal weight gain was independently associated with an increased risk of SIDS and could be identified at the routine six week assessment.

- The lower weight of SIDS infants compared to the control infants which was apparent at birth was even more notable in the two weeks before death
- SIDS infants, particularly those of normal birth weight, exhibited poorer weight gain than their controls
- Although poor growth was evident among SIDS infants there was no evidence of accelerated retardation in the weeks prior to death
- The difference in growth between SIDS and control infants was apparent within the first five to seven weeks of life

SLEEP AND WAKING PATTERNS:

SIDS peaks at 2-4 months, is more prevalent in the cold winter months and typically occurs in the early morning hours, when darkness is prolonged where most babies are asleep, suggesting that sleep may be part of the pathophysiological

mechanism of SIDS. There are abnormalities specific to night time sleep which may be indicative of a central nervous system (CNS) deficit that contributes to a high frequency of SIDS during the night. REM sleep abnormalities in vulnerable infants are indicative of a pervasive CNS immaturity. This is in relation to the possible involvement of the circadian rhythm of melatonin. [(24)]

ALTERATIONS IN BODY TEMPERATURE:

Neuronal noise is very much affected by body temperature; if body temperature is high, neuronal noise is low, and vice versa. Elevated room temperature, extensive crib bedding and prone sleeping position are known SIDS risk factors - and are all factors that contribute to higher body temperature. So far, the mechanism of why higher body temperature increases the risk of SIDS is unknown but neuronal noise and brief arousals could be a key. Since thermoregulation in young infants is not yet fully developed, their body temperature is highly affected by the environment/room temperature. [(25)]

SLEEP POSITION:

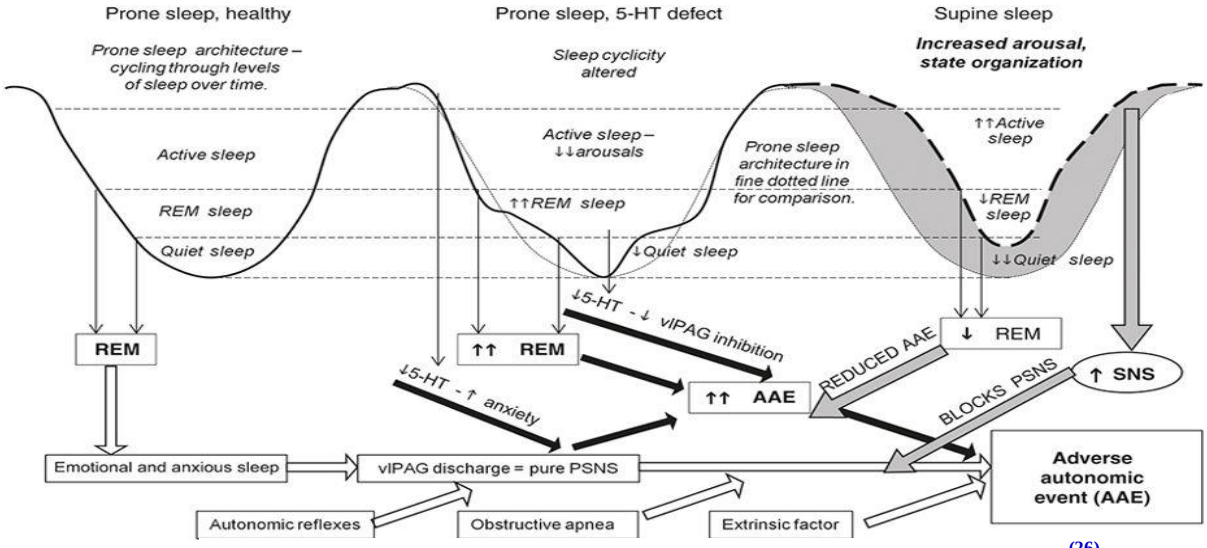

Figure-3: Model for mechanisms of supine sleep's protective effect on sudden infant death syndrome. [(26)]

Compared with back sleeping, stomach sleeping carries between 1.7 and 12.9 times the risk of SIDS. [(27)] The mechanisms by which stomach sleeping might lead to SIDS are not entirely known. Studies suggest that stomach sleeping may increase SIDS risk through a variety of mechanisms, including:

- Increasing the probability that the baby re-breathes his or her own exhaled breath, leading to carbon dioxide buildup and low oxygen levels
- Causing upper airway obstruction.

Compared with infants who sleep on their backs, infants who sleep on their stomach:

- Are less reactive to noise.
- Experience sudden decreases in blood pressure and heart rate control.
- Experience less movement, higher arousal thresholds, and longer periods of deep sleep. [(28,29)]

These characteristics might put an infant at higher risk of SIDS. The simple act of placing infants on their backs to sleep significantly lowers SIDS risk.

Moreover, babies may benefit in other ways from sleeping on their backs. A 2003 study found that infants who slept on their backs were less likely than infants who slept on their stomachs to develop ear infections, stuffy noses or fevers. When the temperature is lower, an infant has higher neuronal noise level that yields more arousals during which the infant can change his position to help himself

breath more freely or move a blanket that may be covering his face.[30,31]

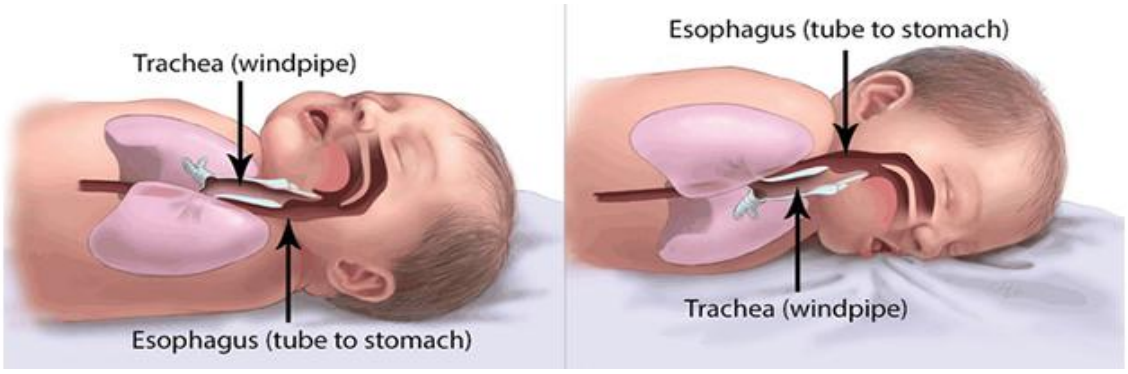

Figure 4: Position of trachea in both the back sleeping and stomach sleeping position. [32]

BRAIN DEFECTS:

Horne et.al, have used a combination of behavioural, respiratory, and cardiovascular responses to detect arousals. These measures would have detected most brainstem arousals. It is noteworthy that cortical arousal is the end point most commonly used in infant and adult studies of arousal. [33, 34] Overheating, rebreathing and sleep apnoea may require the infant to arouse in response to asphyxial or thermal stimuli. These stimuli may affect arousal differently than tactile or auditory stimuli because hyperthermia or asphyxia can depress, as well as provoke, arousals. Although several studies of infant responses to hypoxemia or hypercapnia have been conducted, it will be important in the future to evaluate their combined effect because this would better reflect the primary stimulus for arousal during rebreathing or sleep apnoea. This is important because as asphyxia progresses, carbon dioxide, a potent stimulus for arousal, increases; however, at the same time, as hypoxemia, a relatively weak stimulus for arousal, advances, it rapidly becomes an inhibitor of arousal during the course of ensuing hypoxic coma. [35, 36] In fact, a recent report suggests that when mild asphyxia is the stimulus, sleeping infants are more readily aroused when prone than when supine. [37]

INFECTION:

"Common Bacterial Hypothesis" proposed by Morris et.al, which states that common organisms, bacterial and viral, can cause SIDS. [38] Viral cultures can be difficult to obtain post-mortem, [39] making it hard to prove a viral association; however, more than half of SIDS infants have been reported to have a recent mild viral illness near the time of death. [40] More sophisticated techniques of viral identification could prove useful in establishing the connection between viral and bacterial infection.

GENETIC POLYMORPHISM:

There are various possible links between genetic variations in the neuroadrenergic system and the occurrence of sudden infant death syndrome. The X-chromosomal Monoamine oxidase A (MAOA) is one of the genes with regulatory activity in the noradrenergic and serotonergic neuronal systems and a polymorphism of the promoter which affects the activity of this gene has been proclaimed to contribute significantly to the prevalence of sudden infant death syndrome (SIDS). Variations in single nucleotide polymorphisms of MAOA should be subjected to further analysis to clarify their impact on SIDS. [41]

Interleukin-10 (IL-10) is a regulatory cytokine. IL-10 has been shown to influence both the susceptibility to various diseases, and the various polymorphisms in the IL-10 gene promoter have been linked with disease prevalence and severity. The genes involved in the immune system also thought to be of importance with regard to sudden infant death syndrome (SIDS). The specific halotypes in the IL-10 gene promoter have

been shown to be associated with both SIDS and unexpected death due to infection. [(42)]

AIR POLLUTION

Traffic-related air pollution can have a negative impact on birth outcomes and perinatal health. A number of studies have also suggested a link between ambient air quality and the incidence of sudden infant death syndrome (SIDS). [(43, 44)] There is a potential association of sudden infant death with PM_{10} and NO_2 and the association with particulate matter and infant mortality in particular is widely recognised. [(45)]

PREVENTION:

There is no precise way to prevent SIDS, but you can help your baby sleep more safely by following these tips:

- **Back to sleep:** During sleep place your baby on his or her back, rather than on the stomach or side, every time you put the baby to sleep for the first year of life. This is not necessary when your baby is awake or able to roll over both ways without help. Do not assume that others will place your baby to sleep in the correct position insist on it. Advise sitters and child care providers not to use the stomach position to calm an upset baby.
- **Keep the crib as bare as possible:** Use a firm mattress and avoid placing your baby on thick, fluffy padding. Don't leave pillows, fluffy toys or stuffed animals in the crib. These can interfere with breathing if your baby's face presses against them. Use a mattress that is firm, flat, waterproof and in good condition. Dress your baby in fitted, light sleepwear that makes them comfortable at room temperature.
- **Don't overheat your baby:** To keep your baby warm, try a sleep sack or other sleep clothing that doesn't require additional covers. Don't cover your baby's head. Keep your baby's head uncovered – their blanket should be tucked in no higher than their shoulders. Babies often have minor illnesses that you do not need to worry about. Give your baby plenty of fluids to drink and do not let them get too hot.
- **Have your baby sleep in your room:** Ideally, your baby should sleep in your room with you, but alone in a crib, bassinet or other structure designed for infants, for at least six months, and, if possible, up to a year.
- **Adult beds aren't safe for infants:** A baby can become trapped and suffocate between the headboard slats, the space between the mattress and the bed frame, or the space between the mattress and the wall. A baby can also suffocate if a sleeping parent accidentally rolls over and covers the baby's nose and mouth.
- **Breast-feed your baby, if possible:** Breast-feeding for at least six months lowers the risk of SIDS.
- **Offer a pacifier:** Sucking on a pacifier without a strap or string at naptime and bedtime might reduce the risk of SIDS. One caveat - if you're breast-feeding, wait to offer a pacifier until your baby is 3 to 4 weeks old and you've settled into a nursing routine. If your baby's not interested in the pacifier, don't force it. Try again another day. If the pacifier falls out of your baby's mouth while he or she is sleeping, don't pop it back in. Use a pacifier, starting once breast-feeding is well established and stopping before one year of age (before dental complications may arise).
- **Immunize your baby:** There's no evidence that routine immunizations increase SIDS risk. Some evidence indicates immunizations can help prevent SIDS.
- **Quit smoking:** Do not share a bed with your baby if you or your partner smoke or take drugs, or if you've been drinking alcohol. Do not smoke during pregnancy or let anyone smoke in the same room as your baby – both before and after birth.
- **Prenatal care for baby:** During the pregnancy, make sure that the expectant mother receives good medical care and adequate nutrition.

If you notice a pause longer than 10 seconds in your baby's breathing, wake him or her up with a small jolt or stimulus such as a

flick of your finger on the feet. If this doesn't set the baby breathing again, the next step is more vigorous stimulation such as a pinch. If that doesn't work, begin mouth-to-mouth resuscitation. Never shake the baby, as it could harm the baby. Even minimal shaking may cause a serious head injury.

CONCLUSION

There is no precise way to prevent SIDS, but you can help your baby sleep more safely by following some of the above given preventive measures. Mothers and family members should be aware of the preventive measures for SIDS. This review is beneficial to all medical practitioners as well as mothers and family members with a newborn.

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