Journal of
Cardiology & Cardiovascular Therapy
ISSN: 2474-7580

Juniper Publishers
key to the Researchers

Short Communication
Volume 5 Issue 4 – May 2017
DOI: 10.19080/JOCCT.2017.05.555666

J Cardiol & Cardiovasc Ther

Hart Rate Monitor to Prevent Sudden Infant Death Syndrome

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Submission: April 10, 2017; **Published:** May 30, 2017

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Abstract

Sudden infant death syndrome (SIDS) is a major cause of infant mortality throughout the world, yet its cause and mechanism of action remain poorly understood. Here, we discuss a tool to preventing these deaths: the Heart rate monitors (HRM) and why it can work to prevent SIDS deaths.

Death, regardless of cause, occurs exclusively through cardiac arrest, we suggest applying a HRM with programmable alarm sounds that, when properly positioned, could advise an adult when the infant's heart rate decreased beyond a certain threshold, through a sound, a phone call or another mechanism.

This would allow the adult to wake the baby and call the doctor or take the child to the emergency room. If our theory is correct, waking the baby will be enough to reactivate the stimulation of the brain stem and save his life. This has the potential to be a low cost means of significantly reducing infant death worldwide and warrants further scientific attention.

Keywords: GABA; SIDS; HRM; CAP rate; Me5

Introduction

According to our model of aetiology SIDS occurs by a deficiency of neurotransmitters (NT): acetylcholine (Ach), serotonin (5-HT), dopamine (DA) and, above all, orexin (OX); this shortage is caused by the poor functioning of the mesencephalic trigeminal nucleus (Me5) [1], due to the physiological lack of teeth in this period of life, which normally stimulates the nuclei manufacturers of the aforementioned NT [2]. Babies who die of SIDS do not have an efficient Me5 system due to lack of teeth, so there is not enough non-specific activation of the cortex [3].

The inhibition exerted by the neurotransmitter gamma-aminobutyric acid (GABA) on the nuclei of the ascending reticular activating system (ARAS) and on hypothalamic cells that produce orexin causes a significant depletion of the relative neurotransmitters (NT) with less control of the respiratory functions, prevalence of parasympathetic effects and gastro-oesophageal reflux disease (due to lack of OX).

Because of these deficiencies the central nervous system (CNS) does not have the ability to react to possible internal stimuli and a trivial infection or a cardiac problem, as a long QT, can be fatal. The active Me5 the pacifier and can save the lives of children [4]. We can demonstrate this with the polysomnography (PSG) findings? Probably yes [5].

At birth, both the C process (circadian) is the S (homeostatic) are not working properly, ventrolateral preoptic nucleus (VLPO) is not stimulate and is not produced enough GABA. The cyclic alternating pattern (CAP) rate is 100%.

Immediately after birth the two processes are activated, GABA is produced and the CAP rate falls [6]. In infants the Me5 mechanism does not work yet because they have no teeth; infants at risk for SIDS/ALTE, should have an excess of GABA compared to not at risk peers. If the GABA is high the CAP rate must be lower than children of the same age, therefore less stimulation of ARAS nuclei, less control of the cardio-respiratory functions, prevalence of parasympathetic effects and lack of orexin (eg. gastro-oesophageal reflux, etc.), the higher apnea/hypopnea index; as in the NCAP phases of Obstruction Sleep Apnea Syndrome (OSAS) patients.

In the newborn and before birth, sleep is essentially only rapid eye movement (REM) sleep, but the duration of non-REM (NREM) sleep grows rapidly from the first month of life. At birth

and during the first months of life the baby does not distinguish between day and night, and his sleep rhythm is independent of the environment and governed only by internal needs like hunger and thirst, lasting around 25 hours (Meier-Koll 1979).

The decrease of REM sleep is physiologic due to the production of GABA by VLPO. The increase of GABA determined a lowering in the CAP rate and the non-stimulation of the ARAS nuclei responsible for production of Ach. There are less CAP A1 phases because the Me5 is enough stimulated.

The A2 and A3 subtypes are, instead, increased since they are indices of greater activation of ARAS nuclei, exaggerated, sometimes pathological, (in this case is the danger to succumb to GABA inhibition that activate the organism responses which, in turn, activate the nuclei of ARAS), as the pathological bruxism, OSAS, the restless legs syndrome (RLS) etc. [7].

To sum up in a healthy baby, not in danger of ALTE/SIDS, at birth the CAP rate should be 100% and gradually decline soon after and should consist almost exclusively of A1 phases; A2 and A3 stages may, however, be linked to more or less important pathological events [8].

Conversely, a baby at risk for SIDS should have:

a) Lower CAP rate (excessive GABA for that time of life).

b) Lower A1 (the Me5 is not working).

c) A2 and A3 high (a defensive response of ARAS nuclei to GABA excess).

d) Events related to the increase in GABA (less functionality).

e) Manifestations in vital functions of lower OX (breathing, cardio-circulatory, etc.).

f) Manifestations of the alterations of Simpathetic/ Parasympathetic functions (prevalence of parasympathetic with bradycardia, for less stimulation of the LC) [9].

Hypothesis

As an adult, even in a child's the sleep process to begin requires that the nuclei VLPO and median preoptic nucleus (MnPO) release GABA in the brain stem and hypothalamus. When the GABA reaches its target cells, these are inhibited because the membrane potential difference is sharply negative following the entry of Cl-. This inhibition prevents both the release of neurotransmitters both the synthesis of critical proteins (eg, dopamine transporters or monoamine oxidase).

The results of the autopsies of babies that died for SIDS show deficient levels of these proteins; their deficiency is, in our opinion, an effect rather than a cause of SIDS. During sleep, when the level of GABA is increased and many cells are inhibited, an intrinsic mechanism in the brainstem is activated to counter any harmful effects of excessive inhibition by GABA, Is the Me5 that triggered shall stimulate the ARAS nuclei [10].

This nucleus is largely constituted by pseudounipolar cells, but its caudal termination (Me5c) consists of small multipolar cells, typically GABAergic, positioned just in front of the trigeminal motor nucleus (Mo5) which, under normal conditions, is inhibited by Me5c [11]. When the GABA released from the hypothalamus inhibits the cells of Me5c, the inhibition of Mo5 fails [12].

The masticatory muscles contract, the teeth are touching, and the Me5 is activated [13] and induces the release of glutamate on the ARAS nuclei, on the periaqueductal gray (PAG) and on parasympathetic nuclei.

The result is the activation of some nuclei of ARAS, but without compromising the action of GABA, thereby maintaining a deep sleep, but also maintaining a certain degree of cortical activation that can allow us to respond to any eventual need [14]. When the Me5 is not working properly and there is an excess of GABA, some nervous cells die and activated glial cells that release IL 1 beta and prostaglandins which increase the levels of the substance P.

These events are possibly very common, but rarely end with fatal outcome; however, they may influence child development and cause extreme weakness of the system «brain stem», with many nuclei and cells which do not work properly. In this scenario, a child may have a high sensitivity to a number of factors that are not usually life-threatening, but may become so due to the weakness of the system at that time (eg, long QT, trivial infections, etc.). In this context, one would expect to find gliosis; leukomalacia; hypoplasia of the brain; increase in substance P levels; and decreased levels of serotonin, dopamine and orexin.

Conclusion

As prevention we first propose the use of the pacifier that can activate Me5; then a systematic PSG towards the end of the first month of life, to highlight children at risk (low CAP rate) and at more broad-spectrum, the application of an heart rate monitor (HRM) with an audible alarm in case of excessive bradycardia (heart frequency for the alarm activation is established by the cardiologist).

Such a device, appropriately positioned, may warn if the heart rate should decrease beyond the value by the cardiologist programmed, and thus make it possible to wake up the child, in order to give him stimuli so to stop the action of the VLPO and let the brain stem return to work, or at least alert an emergency room.

This tool may also work for the dead in the womb (SIUDS), much more frequent than SIDS; in the world there are about 4.5 million foetal deaths each year. In developed countries a pregnancy every 150 ends with the death of the foetus. This

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dramatic event occurs suddenly and unexpectedly (like in SIDS) mostly in the last weeks of gestation.

According to many institutions, up to two-thirds of foetal deaths are unexplained even after the diagnostic examination, just like in SIDS [15]. Therefore, the unexpected foetal death, at the end of pregnancy, is the most frequent cause of death in the perinatal period in the western countries. The above mentioned HRM could be applied overnight on the mother's belly to monitor the baby's heart rate.

This idea stems from our scientific research, but we believe that such a device would work anyway, even if the causes of SIDS were other, because anyone to die must have a cardiac arrest and, given the type of disease, it is virtually certain that in SIDS the cardiac arrest takes place gradually, with a slow decrease in heart rate, as the GABA becomes increasingly active; and even if the cardiac arrest was sudden, we could equip the HRM with an heart rate recorders to register the behavior of the heart and, in highest risk children, equip the HRM with small defibrillators that could be activated in case of cardiac arrest. The HRM could prove useful both to save the lives of these children both to ascertain the real causes of SIDS.

Ethics Statement

The study presented in the manuscript does not involve human or animal subjects.

Author Contribution Statement

i. Andrisani Giovanni: research and study of the pathology, theory development.

ii. Andrisani Giorgia: research, writing.

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