

Traffic noise effects on autonomic arousals during sleep

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ABSTRACT

Noise causes non-specific transient excitations

- of the central nervous system as measured with the EEG and
- of the autonomic nervous system as indicated by alterations of autonomic functions.

During wake autonomic arousals occur as gradually decreasing orienting reflexes or as persistent defensive reflexes. During sleep responses are only defensive with lower thresholds and greater magnitudes than during wake. Their patterns and magnitudes are modified by physical parameters of noise, by individual and situational influences and vary systematically with the duration of associated cortical arousals. They are bi- or triphasic if sleep continues and become monophasic, larger and inert against most influences in case of awakening. The sleep-wake-transition is obviously the dominating stress that is only influenced by the momentary sleep stage.

Patterns of spontaneous and of evoked arousals are similar but magnitudes of the latter are larger. The sum of spontaneous and noise-evoked arousals remains stable indicating a redistribution i.e. a disturbance of the endogenous ultradian rhythm under the influence of noise. This imposes the possibility that noise acts as a health hazard in particular as people do not habituate to noise during sleep.

PHYSIOLOGICAL EFFECTS OF NOISE

Suitable methods for the registration of physiological effects of noise became available at the beginning of the last century. Since then it has been shown that noise causes transient excitations also labelled as arousals of both the autonomic and the central nervous system during wake and during sleep as well. These effects are non-specific and are evoked by other environmental stressors as well.

- **Cortical arousals** are transient excitations of the central nervous system (CNS) that are measured with the electroencephalogram (EEG).
- **Autonomic or subcortical arousals** are transient excitations of the autonomic nervous system (ANS) that are indicated at the periphery by alterations of various autonomic functions. In general, noise reduces the cardiac output, increases the peripheral resistance

while decreasing the width of peripheral blood vessels and elevating the blood pressure; noise causes alterations of heart rate, of ventilation, of skin resistance etc. [1-4].

INDICATORS OF AUTONOMIC AROUSALS

Though many autonomic functions are affected by noise, research focused mainly on 2 highly reliable indicators, on vasoconstriction and on heart rate alterations [5, 6].

- **Vasoconstriction** (Fig. 1, lower part) is easy to measure and to evaluate. The finger plethysmography registers the rhythmic oscillation of blood flow at the finger tips. Vasoconstriction starts 3 seconds after noise onset, reaches a minimum after 8 to 10 seconds and returns then steadily to baseline values.
- **Heart rate alterations** (Fig. 1, upper part). The detailed second-by-second analysis reveals alterations of heart rates that show characteristic quantitative and qualitative variations with various exogenous and endogenous conditions.

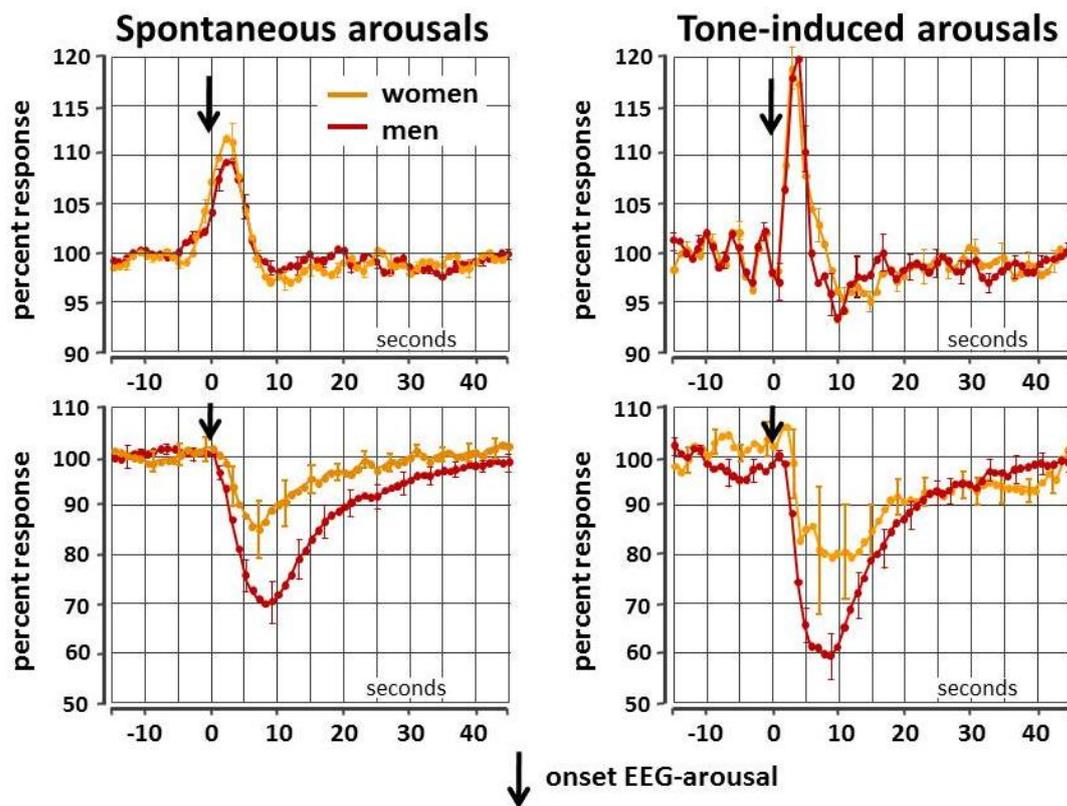


Figure 1. Spontaneous (left part) and tone-induced (right part) cardiac arousals (heart rate alterations, upper part) and vasoconstrictions (lower part) in men and women. The arrows indicate the onset of spontaneous cortical arousals (left) or of acoustic stimuli (right). (after Jordan [18]).

NOISE-INDUCED AUTONOMIC AROUSALS DURING WAKE AND SLEEP

Though the same physiological functions that are affected by noise during wake are also affected by noise during sleep there are some decisive differences.

- During wake noise-induced responses might diminish over time and are then called orienting reflexes or they persist even in the long run and are then labelled as defensive reflexes. This categorization does not apply to sleep. The organism does not habituate to noise during sleep, neither during a single night nor over successive nights [7, 8].

- As the sympathetic tone decreases during sleep it was – due to the law of initial values – expected that responses to noise are larger during sleep than during wake. This was verified in several studies. Identical stimuli cause significantly larger responses during sleep than during wake [9].
- The resulting assumption that the thresholds for noise-induced arousals are lower during sleep than during wake was verified for some but not for all effects. Significantly lower thresholds were ascertained in all sleep stages for cortical arousals, for cardiac arousals and for vasoconstriction, but not for ventilation, galvanic skin resistance etc [9].

AUTONOMIC AND CORTICAL AROUSALS

When evoked by noise autonomic arousals are usually associated with cortical arousals. Though both types of arousals were ascertained in many studies they were scarcely related to each other. The separate analyses for situations where cortical arousals indicate awakenings or continuation of sleep revealed – as demonstrated in Figure 2 – great differences [10].

- **Arousals in case of continued sleep.** In the overwhelming number of cases noise does not wake up. The cardiac responses are then bi- or even triphasic (Figure 2, left part). Heart rate accelerates due to an inhibition of the parasympathetic tone soon after stimulus onset and reaches the maximum after 3 to 4 seconds. The consecutive decline is caused by an increase of the vagal and a decrease of the sympathetic tone. The minimum is often below the baseline and occurs after 8 to 10 seconds. The prestimulus baseline values are regained after 15 to 20 seconds.
- **Arousals in case of awakening.** In case of concomitant awakening the cardiac response becomes monophasic with a long lasting acceleration that reaches a maximum after 20 to 30 seconds (Figure 2, right part). The latter is 3 times higher than without awakening. The deceleration is even slower and the baseline is not regained within a minute.

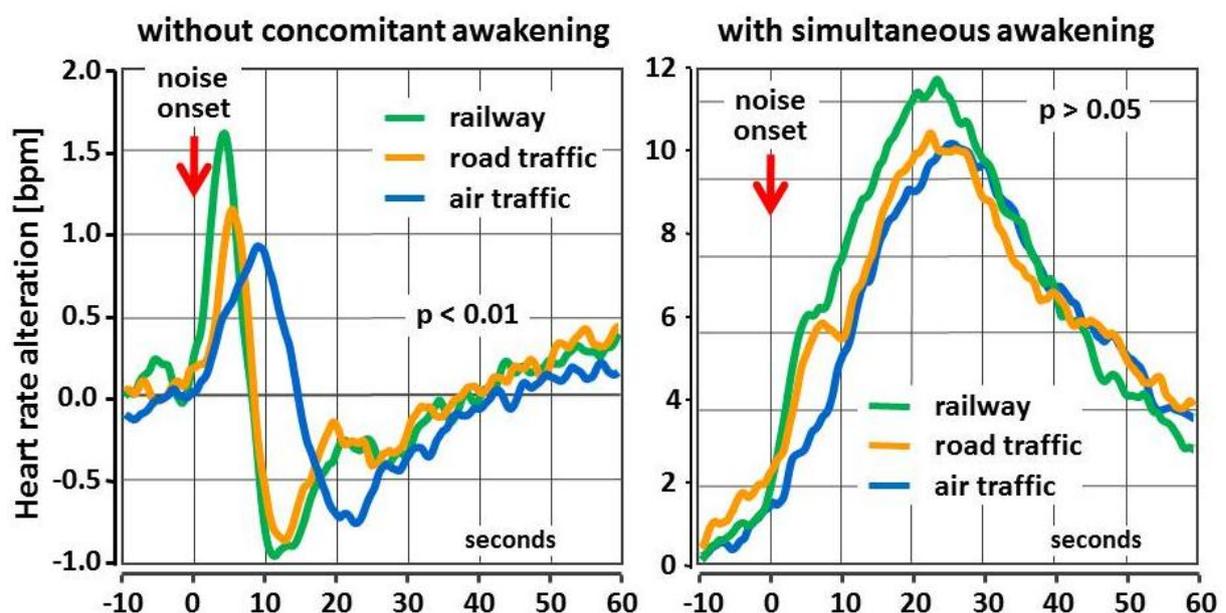


Figure 2. Cardiac arousals related to cortical arousals and traffic modes. Response patterns without awakening (left) and with concomitant awakening (right) [10].

INFLUENCES ON AUTONOMIC AROUSALS

As long as sleep continues the patterns and the magnitudes of autonomic arousals are significantly modified by physical parameters, by individual and situational influences. In case of concomitant awakening, however, the response becomes almost inert against most acoustic, situational and individual influences. Significant influences detected so far are the

- the duration of the wake period (Figure 3). The longer the wake period the longer is the heart rate acceleration and the larger the maximum [10].
- the momentary sleep stage where the cardiac responses increase with the depth of sleep prior to awakening (Figure 6).

This indicates that the transition from sleep to wake is certainly the dominating stress that causes a maximal response. Despite the fact that patterns and magnitudes of arousals then are scarcely affected by other influences, the probability of event-related awakenings and thereby of the occurrence of strong monophasic cardiac arousals is strongly determined by the same factors that influence the patterns and magnitudes of autonomic arousals without awakening [10].

The following paragraphs focus on situations with continued sleep. Influences on autonomic responses with concomitant awakenings will only be mentioned if significant.

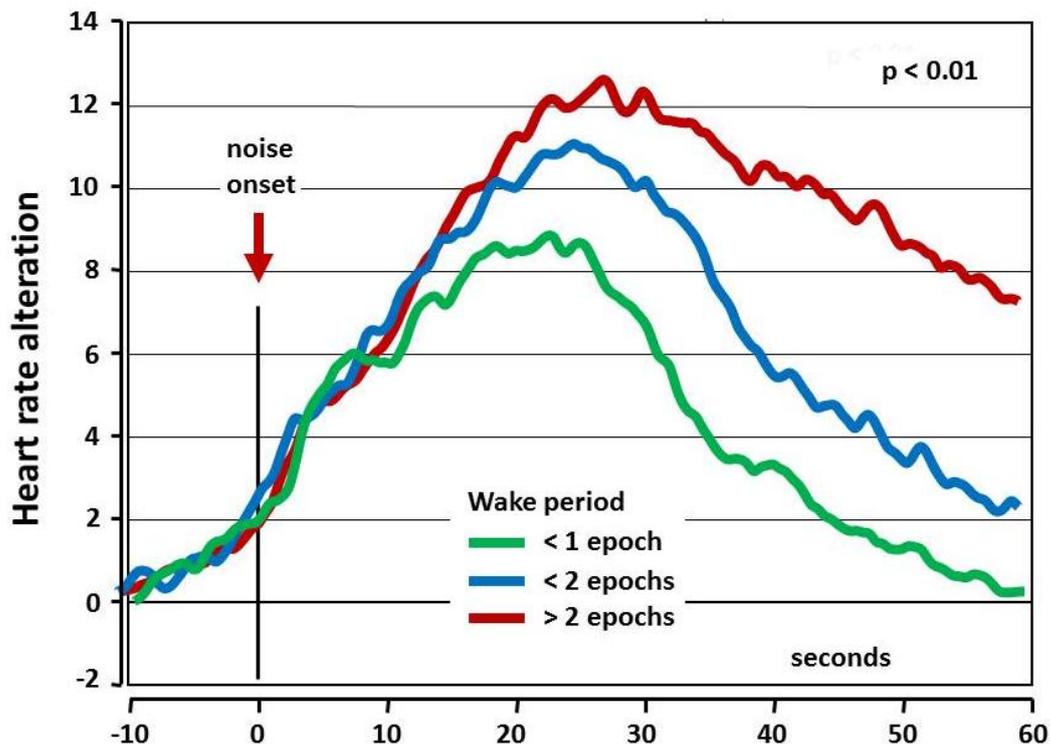


Figure 3. Cardiac arousals related to the duration of the wake period (epoch = 30 s) [10].

Physical parameters

- **Noise mode.** Fourty years ago it has been shown that broadband noise and tones – applied with the same noise level and with the same duration – caused qualitatively and quantitatively different autonomic responses [9]. This applies – as shown in Figure 2 – to traffic noise as well [10]. Different traffic noises – presented with the same sound pressure level – evoke different responses. Heart rate acceleration is steepest, the maximum is highest and is earliest reached with railway noise. For road vehicles the maximum occurs

somewhat later and is less pronounced. Aircraft noise causes the lowest and latest maximum. The minimum occurs at the same time for both surface transport noises but almost 10 seconds later for aircraft noise.

This means that – when it comes to the assessment of sleep disturbances of residents living near airports, along railway lines or busy streets, near military training camps or industrial enterprises – evaluations must focus on the specific noise exposure and can scarcely be deduced from the effects of other noises. However, research in this area is still insufficient and therefore preliminary conclusions still base on studies with artificial noises.

The differentiated response to the 3 traffic modes confirms the significance of the personal meaning of sounds for the individual. This was first shown with the presentation of own and other names to sleeping persons [12]. The own name caused more awakenings, more changes of sleep depth and autonomic arousals than other names or tones. However, the ability of the brain to distinguish between different sounds even while asleep and to respond adequately is not the only explanation for different reactions. Acoustic parameters of sounds and various individual and situational influences are decisive as well.

- **Maximum noise level.** It is plausible to assume that the magnitudes of autonomic arousals increase with noise intensity (see Figure 4). This assumption was verified with artificial tones and with traffic noises as well. This applies, however, not to sounds with extremely short rise times such as sonic booms or shots of tanks.

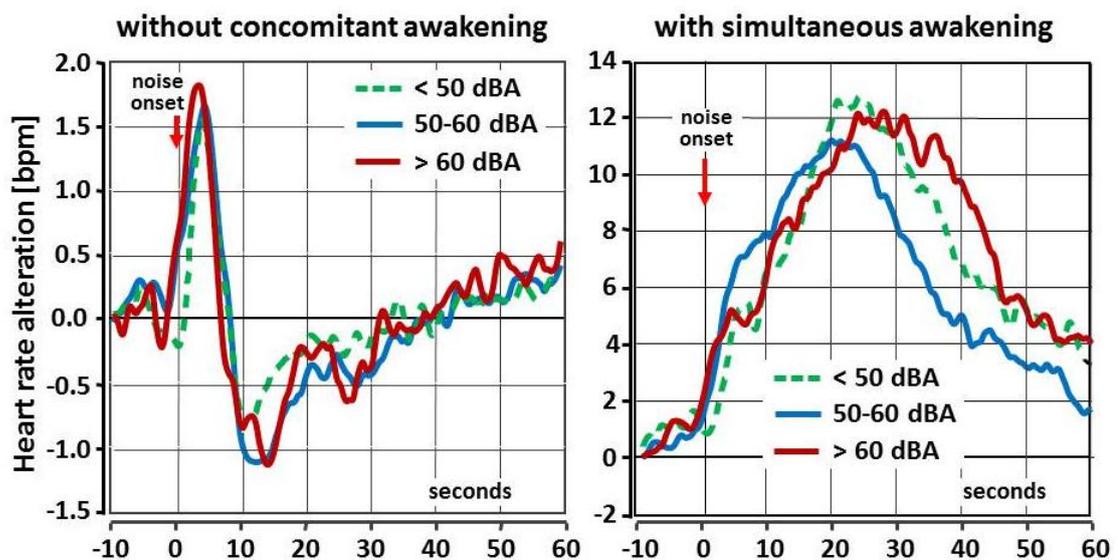


Figure 4. Cardiac arousals related to maximum noise level (here railway noise). Response patterns without awakening (left) and with concomitant awakening (right) [10].

- **Rise time.** The rise time, i.e. the time elapsed between the onset and the maximum of noise, is most important for the patterns and the magnitudes of cardiac arousals (Figure 5 [10]). The sounds of different traffic modes were by median split divided into those with shorter and with longer rise times (faster and slower vehicles). The separate analysis of arousals caused by slower and by faster vehicles revealed that the rise time is least important for aircraft noise with anyway relative long rise and decay times. Concerning surface vehicles the sounds of fast driving vehicles (short rise times) are associated with steeper accelerations and decelerations of heart rate and with significantly larger maxima and minima than sounds of slower vehicles. The most dramatic influence is observed for railway noise. A suitable explanation is that counter measures of the organism start already with the onset of the stimuli and thus are more effective with slower rise times.

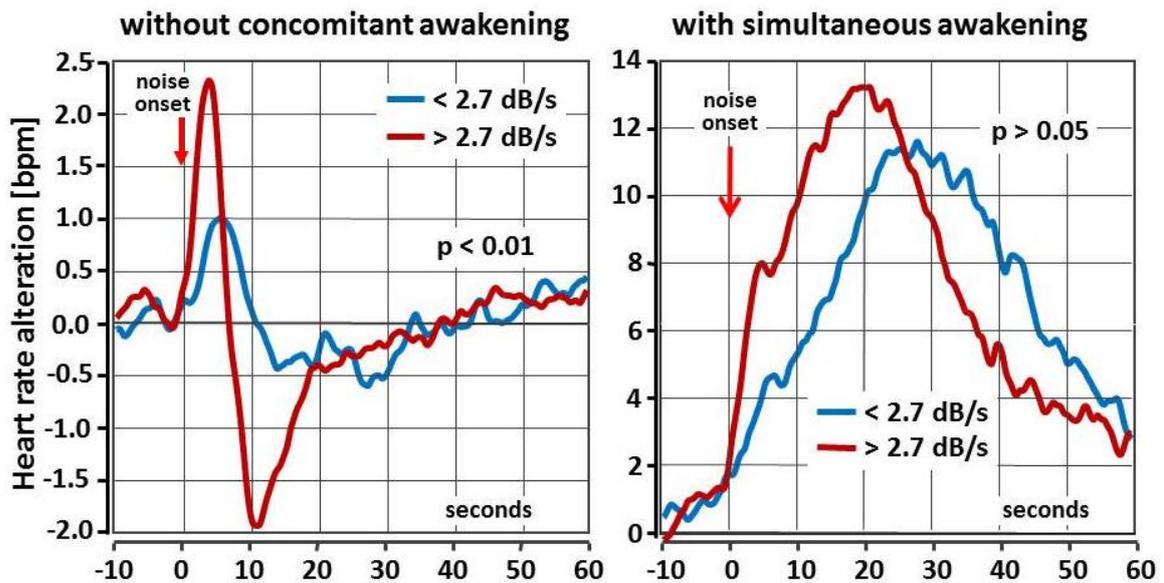


Figure 5. Cardiac arousals related to rise time of noise (here railway noise). Response patterns without awakening (left) and with concomitant awakening (right) [10].

➤ **Individual and situational influences**

Personal variables such as age and gender were only occasionally studied with contradictory results. At present it seems that gender has an only minor influence on the magnitude of the response.

- **Sleep stage.** The effects of the momentary sleep stage on patterns and magnitudes of autonomic arousals were repeatedly studied. Minimal reactions were always found during slow-wave-sleep (SWS) (see Figure 6, left). Responses in sleep stages S2 and SREM are larger. The contrary is true for situations with concomitant awakenings. The momentary sleep stage is here again most decisive, however in the opposite direction. The response is largest when wakin up from deep sleep and least when waking up from SREM.

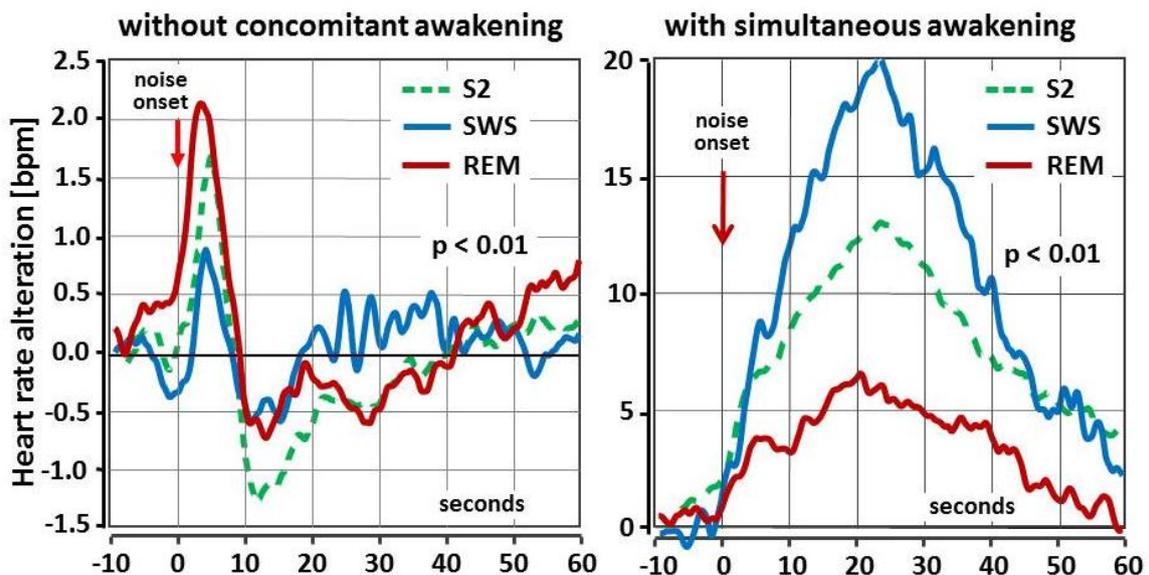


Figure 6. Cardiac arousals related to the momentary sleep stage (here railway noise). Response patterns without awakening (left) and with concomitant awakening (right) [10].

- **Time of night.** As the amount of both, sleep stages S2 and SREM increases with each successive sleep cycle the response to noise in the late night is larger than to noise in the early night.
- **Sympathetic tone.** The actual sympathetic tone of an individual may vary considerably and cause – due to the law of initial values – variations of the response. The magnitude of noise-induced heart rate alterations is indeed inversely related to the momentary sympathetic tone as indicated by the prestimulus heart rate (see Figure 7). The lower the sympathetic tone the steeper is the acceleration of heart rates and the probability increases that the acceleration is preceded by a short initial deceleration and followed by a deceleration with a minimum below the baseline.

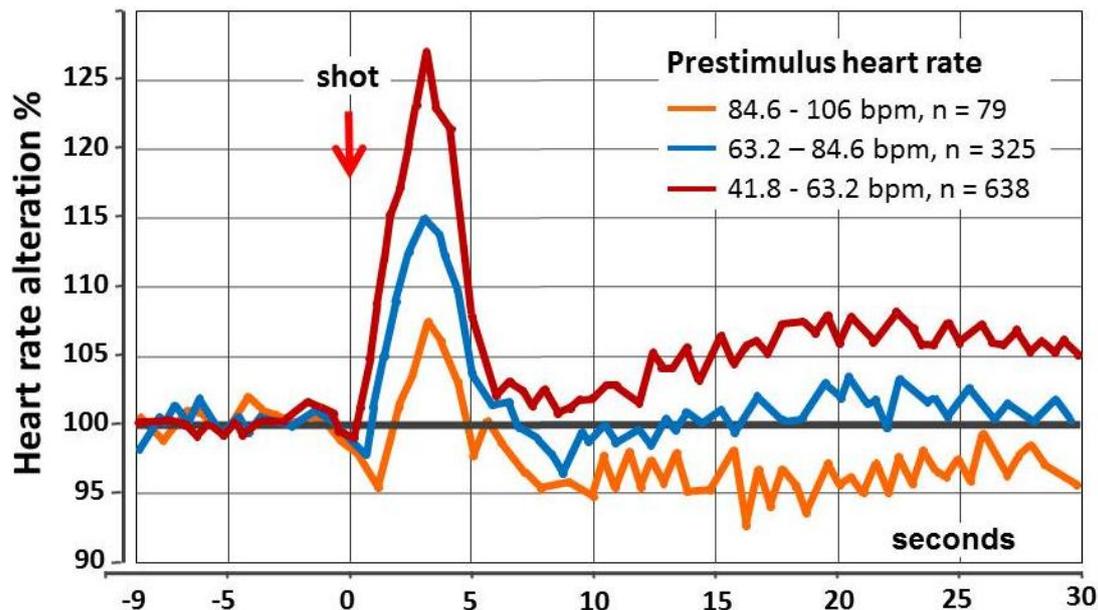


Figure 7. Cardiac arousals related to the sympathetic tone, expressed by the prestimulus heart rate. 23 shots of tanks/3h, 78-82 dBA, 11 pm – 2 am or 4 am – 7 am.

SIGNIFICANCE OF AUTONOMIC AROUSALS

Up to now the significance of autonomic arousals for wellbeing and health is obscure. The analysis of spontaneous arousals and the comparison of spontaneous with evoked arousals might give some hints.

➤ Patterns and magnitudes of spontaneous arousals

As demonstrated by Figure 8 the patterns of spontaneous cardiac arousals are the same as if evoked by environmental stimuli [13-17]. An initial acceleration with a maximum after 3 to 4 seconds is followed by a deceleration with a minimum after 8 to 10 seconds and the baseline is regained after about 15 seconds.

Their magnitudes are obviously associated with the intensity and the duration of the concomitant cortical arousals. Even K-bursts and D-bursts (4, 6 s on average) are associated with though small heart rate accelerations followed by again small declines below the baseline. The magnitudes become larger with microarousals (8 s on average) and increase further with phases of transient activations (PATs, 12 s on average) and even more with concomitant awakenings where the arousals become monophasic [16-17].

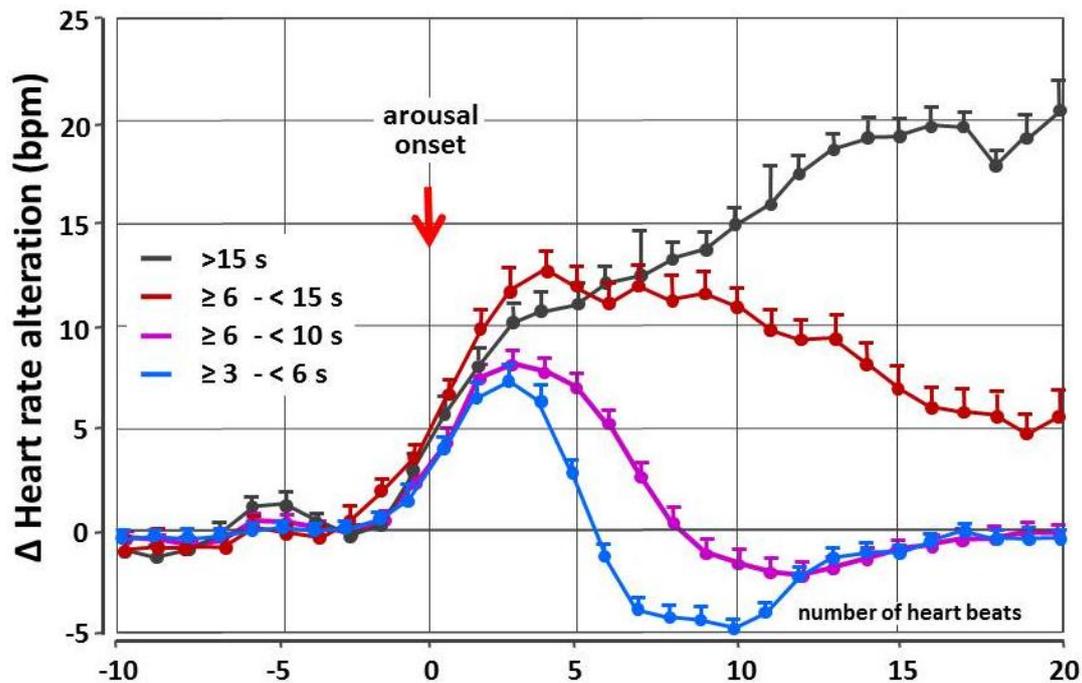


Figure 8. Spontaneous cardiac arousals. Patterns related to cortical arousals (after Sforza [16, 17]).

A remarkable but plausible observation is that autonomic arousals start before the onset of the cortical arousals. Similar observations were made with acoustic stimuli, where heart rates start to increase, at least in case of awakenings, before the onset of an acoustic stimulus (e.g. Figures 2, 3, 4). This suggests that noise becomes particularly disturbing if it occurs in a situation of transiently increased sympathetic tone as indicated by spontaneous arousals.

➤ **Magnitudes of spontaneous vs evoked arousals**

The analysis of spontaneous and of evoked autonomic arousals has shown essentially similar patterns. But – as depicted in Figure 1 – the magnitudes of autonomic arousals (of vasoconstrictions and of cardiac arousals) are larger when evoked by acoustic stimuli [18] as compared to spontaneous arousals.

➤ **Rhythmicity of arousals in quiet and in noise**

Though arousals are undoubtedly caused by noise the total number of arousals scarcely increases, a phenomenon that was repeatedly reported for arousals without awakening as well as for awakenings [19, 20]. This implies a temporal redistribution of arousals. As spontaneous arousals occur rather rhythmically [21] noise disturbs this endogenous ultradian rhythm. This has been demonstrated for spontaneous vasoconstrictions during wake [not yet published]. In quiet arousals occur within a narrow frequency range. This range becomes broader under the influence of noise and there is no reason to believe that this does not apply to nocturnal noise as well.

➤ **Evoked arousals – a possible health hazard**

Taken together the magnitudes of noise-induced arousals are greater than that of spontaneous arousals. The rhythmicity of arousal scenarios that under nocturnal noise consist of spontaneous and evoked arousals is disturbed. This does not prove deleterious effects but imposes the possibility of a health hazard in the long run in particular as people do not habituate to nocturnal noise. This conclusion seems to be justified at least for the maximum response associated with awakening. The autonomic arousals then indicate the

inability of the organism to respond adequately to external stimuli, i.e. to differentiate between noise modes and noise parameters.

DETECTION OF AWAKENINGS USING CARDIAC AROUSALS

The characteristic and systematic alterations of the patterns and the magnitudes of cardiac arousals with the duration and the intensity of cortical arousals suggest that awakenings might be detected by cardiac arousals [19-20]. This is particularly interesting as the electrocardiogram (ECG) is easy to record and can be automatically evaluated whereas the detection of cortical awakening arousals require the visual evaluation of the polysomnogram. The latter is a rather time-consuming process and the inter-rater reliability by far not perfect. Suitable attempts were already made by several authors [e. g. 20, 22-24]. They developed algorithms that rely on the registration of the ECG and of body movements ascertained with accelerometers attached to the wrist. None of the current versions provide yet satisfactory results. They either overestimate or underestimate the true number of spontaneous and/or noise-induced awakenings. They undoubtedly detect 'disturbances' in terms of transient elevations of the sympathetic tone. However, Figure 6 demonstrates the overwhelming significance of the momentary sleep stage on the magnitude of the cardiac arousal associated with awakenings. It suggests that the reliable identification of awakenings must take the sleep stage into account. An indication (rather than an identification) of the sleep stage by the ECG and body movements seems to be possible to a limited degree [24].

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