Impulse noise and startle

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Abstract: The Acoustic Startle Effect (ASE) can be one of the consequences of unexpected exposure to impulsive noise caused by gunfire, explosive devices and high speed overflight by aircraft at low altitudes. The ASE consists of a sequence of largely defensive reflex actions with physical, physiological, and psychological components. It is generally accepted that the ASE can be a direct contributor to accidents in inherently risky situations, but what is more controversial is the possibility of some, presently undefined, linkage to longer term health effects. Based on a critical review of the literature, this paper reports some general findings on the possible theoretical bases for any such linkage and makes a number of suggestions for future research.

INTRODUCTION

Unexpected exposure to impulsive noise caused by gunfire, explosive devices, and high speed overflight by military aircraft at low altitudes can contribute to an Acoustic Startle Effect (ASE). The ASE always consists of a sequence of largely defensive reflex actions with physical, physiological and psychological components, and is one of many possible startle effects which can be associated with unexpected stimuli. The relative strength of the ASE seems to depend upon the maximum sound level; on the character and frequency content of the sound; on the onset rate of the sound; on the predictability of the sound; on the reactivity of the person exposed; and on the extent to which the person exposed might have habituated or adapted to the sound depending on experience. Impulsive sound even without an ASE is generally considered to be more annoying than steady sound of the same long term rms average sound level, and the ASE seems likely to exacerbate this difference. However, at the time of writing, there are no quantitative procedures, guidelines, or recommendations laid down to deal with the possibility of ASE.

REFLEX RESPONSES AND HABITUATION

The ASE is both well-known and little understood. Virtually everyone has direct personal experience of the startle effect and there are a number of descriptions of the underlying sequence of physiological reflexes (1, 2, 3). On the other hand, the available scientific data cannot be used to support either quantitative predictions or to generalise from one experimental situation to another. A primitive type of startle response is observable in many types of fish, where the flexor muscles down one side of the body contract rapidly so as to translate the body sideways away from the source of any vibrational disturbance. In crayfish, the escape response is initiated by the Mauthner cells located adjacent to the VIIIth cranial nerve. This nerve also connects the vibration sensing and balance organs to the brain, perhaps encouraging the evolution of a more direct involvement of the auditory system in the startle response in higher order animals.

The somatic reflex connections are very much more complex in humans than in crayfish, involving compensatory extensor responses and additional time delays so as to maintain more effective avoidance behaviour. However, even allowing for some initial reverberation of the nervous signals around multiple reflex connections, the immediate somatic or musculoskeletal components of any startle reflex are mostly complete within the first 500ms after the stimulus onset time. This means that the slower acting autonomic system response involving more general neural and hormonal activity can play a useful role in sustaining a protective response against the unexpected threat. An important component of the autonomic system response seems to be some kind of feed-forward mechanism whereby the cardiovascular and other systems are 'kick-started' in expectation of increased metabolic demands being placed upon them in advance of the anticipated depletion of biochemical resources that would otherwise trigger a slower acting homeostatic response to maintain internal equilibriums.

After a further delay for conscious appraisal of the threat, the organism as a whole can then begin to take co-ordinated action. The survival advantages of having always taken immediate avoidance action and then having immediately prepared the visceral systems to support further sustained activity probably outweigh the disadvantages of having engaged in unnecessary action if the threat turns out to have been benign. However, there would seem to
be some advantage to being able to stand down the autonomic response as soon as the threat has been determined to be benign. The extent to which different individuals can achieve this under different circumstances probably varies.

A working hypothesis about the role of habituation in the ASE can be set out as follows:

- Any unexpected acoustic stimulus can elicit an involuntary somatic reflex response. The magnitude of the response is related to the magnitude and unexpectedness of the stimulus.
- Somatic response habituation depends on stimulus magnitude and on unexpectedness, and applies mainly to the later stages of the initial somatic response.
- The somatic response can be followed by an involuntary autonomic response which also varies depending on stimulus magnitude and unexpectedness.
- Autonomic response habituation then depends both on stimulus magnitude and unexpectedness and also on cognitive appraisal of the threat. Individuals are likely to vary both in terms of their initial reactivity and also in terms of how quickly they can master the process of cognitive appraisal and start to stand-down their response.

Both the magnitude of any initial response and the extent to which it might be habituated over time are of great interest in determining objective criteria for the assessment of impulsive stimuli against which ASES might be expected to occur.

RELATIONSHIP TO HEALTH EFFECTS

It is generally accepted that the ASE can be a direct contributor to accidents where any temporary loss of concentration might be a risk factor. What is more controversial is the possibility of some presently undefined linkage or association with longer term non-auditory health effects. For example, there does not seem to be any evidence that the ASE and other startle effects are anything other than normal adaptations to transient changes in the external environment, having evolved from primitive responses with apparent survival value. Exercise causes similar short term changes in physiological variables and this is generally thought of as being beneficial (except perhaps in individuals with existing health problems due to other causes).

When considered in the light of continuing uncertainty over noise and health effects in general (4) it is clear that any contribution that this particular topic might be able to make to the general debate might be extremely valuable. It is not difficult to envisage a number of simple experiments that could be carried out in the laboratory to determine the physical parameters of typical exposures that might contribute to the ASE, and to find ways of identifying the most reactive and least habituating individuals, assuming that such individuals exist. Epidemiological studies of environmental noise and health have shown that, if they exist at all, then the relative risk ratios of adverse health effects resulting from chronic exposure to environmental noise must be quite low. One of the problems here could be that only a minority of the population might be susceptible to noise induced health effects. If this were true, then in the absence of any independent means of being able to identify these susceptible individuals in advance, then the effects on these people alone might be swamped by the general variability in the results (5). Laboratory based studies of individual differences in habituation to ASE caused by impulsive noise might provide some means of identifying susceptible individuals in advance.

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REFERENCES