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Effect of caffeine on central auditory pathways: An evoked potential study

Research paper

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Abstract

Caffeine is consumed in various forms like tea, coffee, chocolates and colas. The present study evaluated the effect of caffeine on auditory brainstem response (ABR), mid latency response (MLR) and slow vertex response (SVR) in 40 male volunteers. The recordings were done using a computerized evoked potential recorder by 10–20 electrode placement system. The subjects consumed 3 mg/kg body weight of caffeine after 12 h abstinence from caffeine in any form. The data obtained revealed that latencies of waves IV and V along with I–V interpeak interval of ABR decreased significantly. This was accompanied with significant increase in amplitude of wave V. MLR latencies and latency of P1 wave of SVR was significantly decreased following caffeine ingestion. The results indicated that caffeine improves transmission in the peripheral and central brain auditory pathways.

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Keywords: Auditory brainstem response; Mid latency response; Slow vertex response; Peak latency; Interpeak latency

1. Introduction

Caffeine is a methylxanthine, present in various substances like tea, coffee and some medications. Various hypotheses such as blocking of adenosine receptors, mobilization of intracellular calcium, inhibition of phosphodiesterases and binding of caffeine to benzodiazepine receptors have been postulated as the possible mechanisms of action of caffeine at cellular level (as reviewed by Fredholm et al., 1999). The amount of caffeine in food items ranges from

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40–180 mg/150 ml of coffee, to 24–50 mg/150 ml of tea and 15–29 mg/180 ml for colas (Fredholm et al., 1999). Caffeine in doses of 150–250 mg produces a sense of well being, alertness, allays fatigue, improves motor performance and increases motor activity (Tripathi, 1999). Caffeine interacts with neurotransmitters in the brain and promotes higher functions such as vigilance and attention (Fisone et al., 2004).

Stimulus related potentials (SRPs) are records of the changes in electrical potentials in the nervous system in response to an adequate external stimulus. They reflect the functional integrity of the anatomical sensory/motor pathways in the brain or spinal cord or at periphery.

The response to an auditory stimulus has been divided into three sequential time periods

- a) Early latency (0-8 ms) or auditory brainstem response (ABR).
- b) Mid latency response (8–50 ms).

Abbreviations: ABR, auditory brainstem response; IPL, interpeak latency; kg, kilogram; mg, milligram; ml, milliliter; MLR, mid-latency response; ms, millisecond; SPL, sound pressure level; SVR, slow vertex response

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c) Long latency (50–300 ms) or slow vertex response (Picton et al., 1974).

The ABR is a series of potentials arising from the acoustic nerve and auditory pathways in brainstem that are volume conducted to surface recording electrodes at the scalp. The ABR waveforms are labeled from I–V and correspond to the sequential activation of peripheral, pontomedullary, pontine and midbrain portions of the auditory pathway (Stockard et al., 1992).

The component waveforms of the MLR are labeled as No, Po, Na, Pa, Nb and Pb. The MLR is believed to arise probably from thalamus, primary auditory cortex and association cortex (Picton et al., 1974).

The long latency responses or SVR waveforms i.e., P1, N1, P2 and N2 are distributed widely over the fronto-central scalp area. Vaughan and Ritter suggested that the origin of these potentials was from primary auditory cortex and temporo-parietal association area (Vaughan and Ritter, 1970). SVR provide information about the stimulus and its evaluation. Dickerson and Buchwald suggested various areas of the brain like polysensory areas, pericruciate gyrus, anterolateral gyrus and medial suprasylvian gyrus as generators of SVR. They believed that these potentials are involved in information processing (Dickerson and Buchwald, 1992).

Various factors like temperature (Stockard et al., 1978) and age (Salamy et al., 1975) affect auditory evoked responses. Previous studies from our laboratory have shown that ABR, MLR and SVR are affected by female hormones (Yadav et al., 2002; Farah et al., 2003; Yadav et al., 2003).

EEG studies show that caffeine causes decrease in alpha and delta power and increased fast activity (Hasenfratz and Battig, 1994). Seidl et al. found that reaction time improved in response to target stimuli after administration of a drink having caffeine (Seidl et al., 2000). Other researchers have also noted a decrease in reaction time on caffeine consumption (Azcona et al., 1995; Clubley et al.,1979). Studies by Lorist et al. found an improvement in information processing (i.e., processing of the sensory stimuli perceived from the environment) by caffeine (Lorist et al., 1994, 1996). Deslandes found an effect on P300 latency at the Fz electrode when subjects consumed caffeine (Deslandes et al., 2004). Kawamura et al. found that P300 amplitude and area were significantly increased after 30 min of caffeine intake (Kawamura et al., 1996). Ruijter et al. found an increase in P2 amplitude after caffeine intake and interpreted it as an arousal increasing effect (Ruijter et al., 2000). Our earlier work showed that caffeine led to a significant increase in P3 amplitude and decrease in reaction time, thereby indicating facilitation of information processing and acceleration of motor responses (Dixit et al., 2004). This study aimed to see whether caffeine led to changes in sensory pathways such as the auditory pathway.

2. Materials and methods

2.1. Selection of subjects

The study group comprised 40 normal, healthy male undergraduate students of UCMS in the age group of 18–25 years. The subjects had no history of head injury, epilepsy, hearing impairment, migraine, sleeping problems and drug abuse (nicotine, alcohol, opium, etc.). Due written consent was taken from them prior to the study. The subjects served as their own controls to minimize interindividual variation. Prior to the day of recording, the subjects were asked to abstain from caffeine containing substances for at least 12 h and have adequate sleep. The experimental procedure including the recording technique was explained to them prior to the recording session. The recordings were done in a soundproof room before and 40 min after ingestion of caffeine Figs. 1–3.



Fig. 1. Representative waveform of auditory brainstem response: (a) before caffeine intake and (b) after caffeine intake.

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