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EFFECT OF ALTERED VENTILATIONS ON HUMAN SPINAL AND SUPRA SPINAL REFLEXES

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Abstract: The present study was aimed at evaluating the alterations in the human reflexes brought about by briefly altered ventilations. In 20 healthy, young male volunteers, the H reflex and blink reflex were studied during normal ventilation, voluntary hypoventilation and voluntary hyperventilation. The latencies of these reflexes were compared. It was observed that the amount of altered ventilations used in the present study could not produce any significant change in these reflexes except in the case of the early response of the blink reflex.

Key words: voluntary hyper and hypo ventilations H-reflex blink reflex

INTRODUCTION

Impulse transmission in nerves and synapses is affected by hypoxia, and possibly by hypercapnia. Hence reflexes, which are based on neural and synaptic transmissions, may be affected by hypoxia and hypercapnia. It is documented that the knee jerk is inhibited by hypoxia (1).

Earlier studies in spinal animals have shown that asphyxia for about 3 min was sufficient to abolish all the reflexes (1). Such immediate effects of the altered blood gas tensions on reflexes have not been quantified for human subjects. Voluntary hyperventilation for 2-3 minutes is sufficient to produce changes in the electrical activity of CNS (2, 3). Thus this study attempts to evaluate the immediate effects of voluntary hypo-and hyperventilations on the spinal and supraspinal reflexes viz., the H-reflex and the blink reflex respectively.

METHODS

The study was conducted in twenty male healthy volunteers of age 16-20 years. All subjects gave informed consent and the study was performed in a thermoneutral laboratory (27 ± 1°C), usually 2 hrs after a light breakfast. The conventional electrophysiological stimulating and recording procedures were employed.

The H-reflex and the blink reflex were assessed for their latencies during normal ventilation and voluntary hypo and hyperventilations. The amplitudes of these reflexes were not quantified as they are known to exhibit a wide range of variation (4).

H-reflex: The method is adopted from that of Braddom et al (5). In brief, the tibial nerve was stimulated by surface electrodes in the popliteal crease. The evoked response was picked up by surface electrodes from the gastrocnemius muscle and recorded on a storage oscilloscope at 1 mv/9 mm sensitivity and 5 m sec/9 mm sweep (Fig. 1). The latency was measured.

Blink reflex: The method of Braddom et al (6) was followed for this reflex. The supraorbital nerve was stimulated with the cathode over the supraor-

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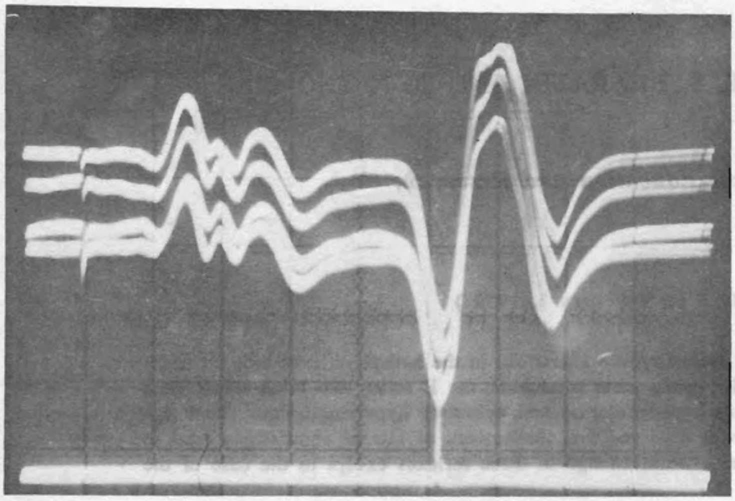

Fig. 1: Typical recording of H-reflex, showing the direct motor response (earlier waves) and H-reflex response (later waves). Calibration: 1000 µV/vertical division; 5 msec/horizontal division.

bital notch. The evoked response was picked up by surface electrodes from the ipsilateral orbicularis occuli and recorded in the storage oscilloscope at 0.2 mv/9 mm sensitivity and 5 m sec/9 mm sweep (Fig. 2). Latencies of the early and the late responses were measured.

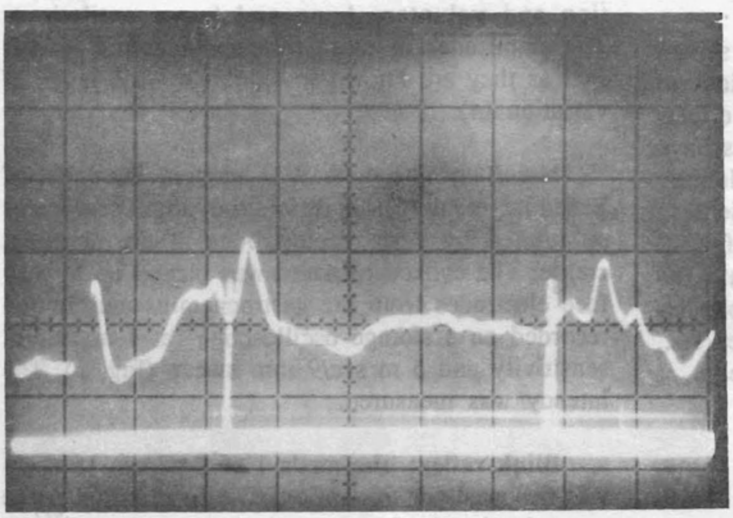

Fig. 2: Typical recording of Blink reflex, showing the early and late responses. Calibration: 200 µV/vertical division; 5 msec/horizontal division.

Hypo and hyper ventilations: The subject voluntarily controlled the ventilation so that minute ventilations of 3.3 ± 0.8 litres (mean ± SD) and 15.3 ± 1.3 litres (mean ± SD) were maintained for a duration of 4-5 minutes during hypo- and hyperventilations respectively.

The reflexes were studied during normal breathing (for control values) and during the last minute of hypo- and hyperventilatory procedures. A minimum interval of 15 min was allowed between these voluntary ventilatory procedures.

The paired Student's t-test was used for comparing the latencies of the reflexes studied. The latencies obtained during the voluntary ventilatory procedures were compared with corresponding latencies obtained during normal ventilation.

RESULTS AND DISCUSSION

The results are summarised in Table I. The hypo- and hyperventilations of about 4-5 min period did not cause any significant changes in these reflexes except in the case of the blink reflex where hyperventilation decreased the latency of the early response significantly ($P<0.05$).

The unaltered reflex latencies suggest that nerve impulse transmission and the synaptic transmission were not affected by the degree of alteration in ventilation produced in the present study. It is, however, possible that both transmissions were affected but in opposite directions. The latter possibility is very unlikely because our earlier study (7) has indicated that similar amount of hyperventilation has not caused any change in the conduction velocity of the nerve impulse, including the distal motor latency. In this context, we have no explanation

TABLE I : The latency values of H-reflex and Blink reflex. The values are Mean ± SD.

Reflex	*Latency (msec)*		
	During normal ventilation	*During voluntary hypo ventilation*	*During voluntary hyper ventilation*
H-reflex	28.47±1.52	28.79±1.46	28.25±1.78
Blink reflex:			
Early response	10.13±0.71	10.36±1.07	9.83±1.02*
Late response	32.50±3.47	31.35±2.65	31.65±3.33

* $P < 0.05$

for the significant decrease we observed in the latency of the early response of the blink reflex.

Thus the present study indicates that the reflex latencies are usually stable in the face of transient alterations in ventilation. Incidentally, the study has also yielded the normal values of these reflexes in younger individuals as these reflexes, especially the H-reflex (5), are highly correlated with age, and such values are not widely reported for the younger age group.

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