ELECTROCARDIOGRAPHIC, RESPIRATORY AND BLOOD PRESSURE CHANGES AFTER BLOOD/SALINE INJECTION IN THE ORBITO-FRONTAL REGION OF DOG'S BRAIN

N.K. MISRA AND J.N. PRASAD

Department of Physiology,
Jawaharlal Nehru Medical College, Aligarh Muslim University, Aligarh.

Summary: Cerebrovascular episodes were simulated by injecting 5 *ml* of blood/saline in the orbito-frontal region of dog's brain and its effects on E.C.G., arterial pressure and respiration were observed. Extrasystoles, P wave amplitude increase, S-T segment depression, and T wave changes were frequently observed after injection.

Heart rate as calculated from E.C.G. tracings, and arterial pressure both showed an initial fall and a later rise after these injections. Respiration was always inhibited. The initial effects could be due to an immediate rise of intracranial tension, and the delayed effects, due to the direct involvement of orbito-frontal lobes.

Key Words: *orbito-frontal lobes electrocardiographic effects simulation of cerebrovascular episode.*

INTRODUCTION

Smith and Tomlinson (12) found sub-endocardial hemorrhages in cases of intracranial lesion but did not lay any significance on the site of lesion. Anand and Dua (1) reported circulatory and respiratory changes induced by electrical stimulation of limbic system, and suggested that the electrocardiographic changes as a result of cerebrovascular accidents could be due to disturbances affecting cortical area 13 on the orbital surface of frontal lobe. Korteweg *et al* (5) found that the hypothalamic stimulation gave different effects depending upon the site of stimulation. Cropp and Manning (2) reported E.C.G. changes simulating myocardial ischaemia and infarction associated with spontaneous intracranial hemorrhage. Melville *et al* (7) suggested that hypothalamic stimulation might be involved in some of the arrhythmias in patients with cerebrovascular accidents. Menon (8) found evidence of a massive anterior infarct on the E.C.G. in a case of infarction of most of the anterior portion of the temporal lobe, but on necropsy the heart was found to be normal. He is of the view that the site of the primary lesion in the brain is of no significance. McKean and Hitchcock (6) gave an intrathecal hypertonic saline injection in patients of carcinoma, and reported transient E.C.G. abnormalities which appeared within five minutes and were most marked in the first minute.

It is thus accepted that cerebrovascular accidents produce E.C.G., respiratory and blood pressure changes but the role of site of lesion is disputed. This study is an attempt to find out

the effects of simulated cerebrovascular accidents involving the orbito-frontal region.

MATERIALS AND METHODS

Fourteen, healthy mongrel dogs weighing 8-15 *kg* were anaesthetised with chloralose (80-100 *mg/kg*). Cerebro-vascular accident was simulated by injecting 5 *ml* of blood/saline in the orbitofrontal lobe at a place which was 3-5 *mm* deep from the surface. Injection was made by a syringe through a hole burred in the skull after opening the frontal air sinus.

A 12-lead electrocardiogram, arterial blood pressure and respiration were recorded in every animal before injection in the orbitofrontal lobe. After the injection, continuous records of respiration and blood pressure were taken. The electrocardiogram was taken immediately, 15 min, 30 min, and 60 min after the injection. In two dogs a 90 min record was also taken. The site of injection was confirmed by autopsy in each animal. Respiration was registered by a Marey's tambour via an endotracheal tube inserted after anaesthesia, and arterial pressure was recorded from the femoral artery using a mercury manometer.

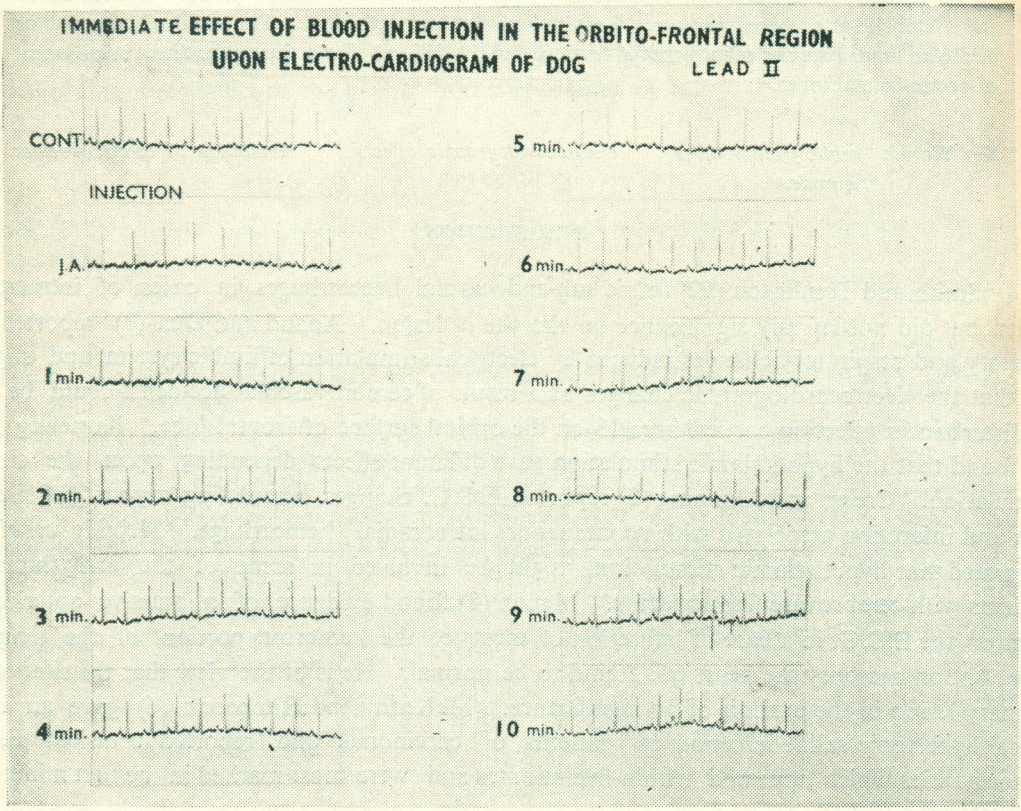

Fig. 1 : *Strips from a continuously recorded electrocardiographic tracing. Note the increase in R-R intervals and S-T segment depression after injection of blood in the orbito-frontal bobe.*

RESULTS

Electrocardiographic changes—(Fig. 1 and 2): An initial decrease in the heart rate followed by a persistent increase by 5-75 beats/min was seen in majority of dogs. All animals

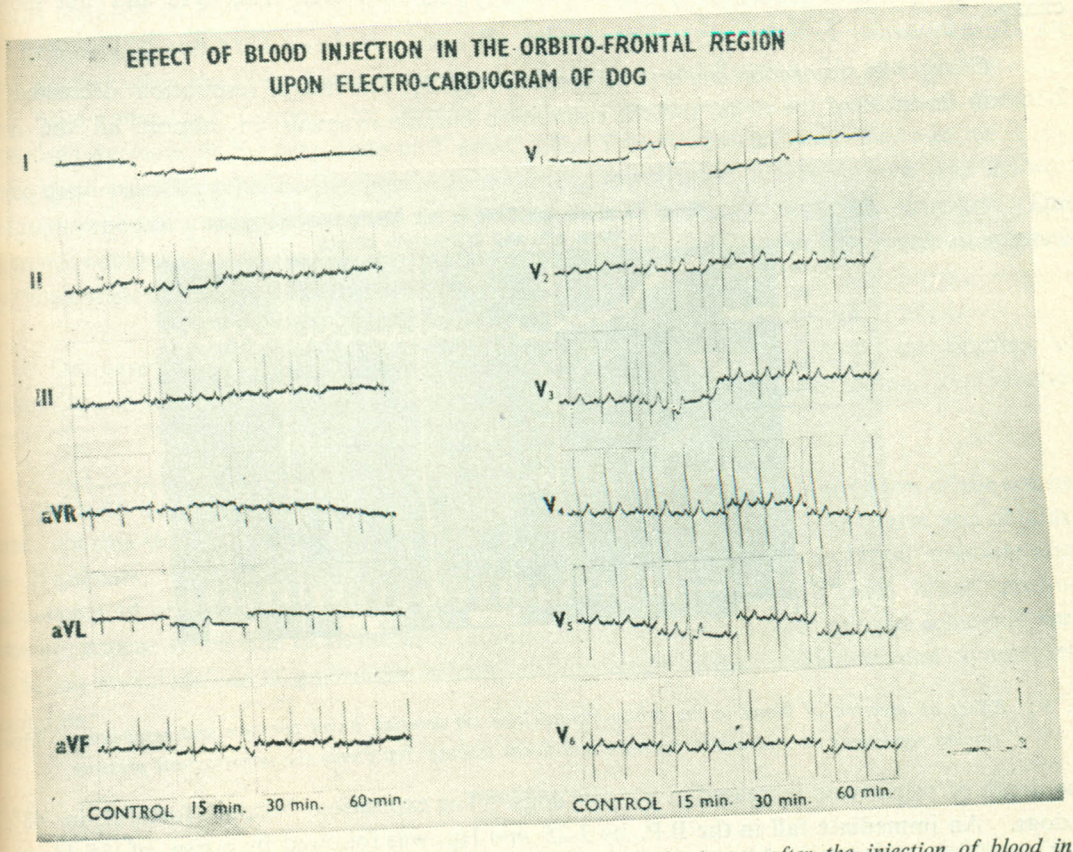

Fig. 2 : Twelve lead E.C.G. tracings picked up before, and 15,30, and 60 minutes after the injection of blood in the orbito-frontal lobes. Note the appearance of extrasystoles and changes in S-T segment.

showed sinus irregularity. 57% showed occasional extra-systoles in different leads. The polarity of P wave did not show a change in majority of dogs although the wave became bifid in few animals. The amplitude of P wave showed a slight but consistent increase in almost all leads in majority of animals. Changes in the duration of P wave were inconsistent. The P-R interval showed a persistent decrease of 0.02-0.06 sec in most of the dogs. Changes in the intrinsicoid deflection and QRS interval were negligible.

The Q wave showed markedly variable changes, but were confined mostly to leads II, aVR and aVF. R wave amplitude decreased in 43% of dogs in leads II, III and aVF by 4-14 *mm* and in V_2-V_6 by 4-24 *mm*. Majority of dogs did not show a change in S wave. A slight elevation of J point in the augmented leads and a depression of 0.5-2.0 *mm*. in the standard

and precordial leads was also observed. About 60% of dogs showed a depression of ST segment in III and precordial leads by 0.5-2.0 *mm*. In a number of animals, the T wave polarity was changed in lead I, aVL and aVF. Although the changes in the amplitude of T wave were seen in all the dogs, but they were markedly variable. The QTc did not show a change.

Changes in respiration and arterial pressure (Fig. 3): The rate of respiration decreased by 2-16/min in most of the dogs, and the respiration became irregular in almost all the dogs.

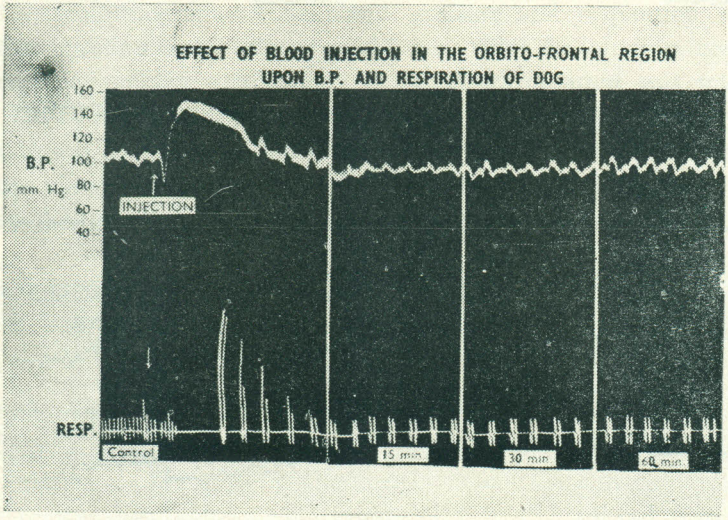

Fig. 3 : *Effect of injection of blood in the arbito-frontal lobe on arterial blood pressure and respiration. Upper tracing represents carotid artery pressure and lower tracing represents the intratracheal pressure.*

The depth of respiration increased in 70% of dogs. The expiration became forceful in 43% of dogs. An immediate fall in the B.P. by 3-25 *mm* Hg. was followed by a rise of 15-44 *mm* Hg in as many as 86% of dogs.

DISCUSSION

Slight to marked electrocardiographic, respiratory and blood pressure changes were seen. An initial decrease in the heart rate occured, a finding similar to that of Anand and Dua (1) who got a slowing of the heart upon stimulation of limbic system. This decrease was followed by an increase in the heart rate. Korteweg *et al* (5) and Melville *et al* (7) also got increase in heart rate which was not immediate. Extrasys oles were also recorded. The initial changes in heart rate and extrasystoles were probably due to sudden increase in the intracranial tension but it is probable that the delayed ehanges were due to sympathetic stimulation. Another possibility is that the injection of blood simulated subarachnoid haemorrhage, a condition in which extrasystoles are more common. A delayed increase in the amplitude of P wave was probably due to pulmonary oedema, a condition described by Russel (10) in cases

of cerebro-vascular accidents. A delayed change in QRS vector was seen in 30% of dogs but the mechanism is not very clear. Our finding of delayed depression of ST segment is in conformity with Weinberg and Bushard (14) and Srivastava and Robson (13). The T wave polarity changed in 86% of dogs, a finding similar to those of Korteweg *et al* (5), Srivastava and Robson (13) and Millar and Abildskov (9), who have described that T wave changes are most marked when the frontal lobe is involved. Srivastava and Robson (13) and Shirley *et al* (11) have noticed tall T waves after frontal lobe involvement whereas we have found both increase and decrease in the amplitude of T wave. The T wave changes may be due to autonomic disturbances probably mediated through the hypothalamus. Srivastava and Robson (13) have reported a frequent increase in QTc whereas we found negligible changes. This difference could be due to the fact that we took records immediately after the accidents whereas they took their records hours to days after the accidents.

Decrease in the rate of respiration recorded by us is in conformity with the findings of Fulton (3) and Kaada (4) who have described inhibition of respiration after frontal lobe involvement.

Anand and Dua (1) have recorded a rise in arterial pressure on stimulation of the frontal lobe. In this study a slight fall in blood pressure followed immediately by a rise was obtained in all animals. Fulton (3) and Kaada (4) reported both a fall and rise in blood pressure upon stimulation of orbital surface of frontal lobe. The initial respiratory and blood pressure changes appear to be due to an increase in intracranial tension but the delayed changes seem to be due to autonomic disturbances which may include vagal inhibition and sympathetic excitation.

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