A COMPARATIVE STUDY OF RIGHT AND LEFT VAGUS ON THE HEARTS OF TOADS AND DOGS

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The effect of stimulating the right and left vagus using different intensities of stimuli has been studied in normal and atropinized hearts of pithed toads and anaesthetised dogs. Variation in the time interval in the appearance of vagus escape exists between right and left vagus stimulation in toads which is due to unequal distribution of vagal inhibitory fibres on the two sides. Increased sympathetic response has been observed in relatively more cases on the left side which is possibly due to proportionately more sympathetic fibres on that side. In the dogs no marked difference in the time interval in the appearance of vagal escape and in the action on the heart has been observed on stimulating the two vagi. Presence of cardio-accelerator fibres in the vagus has been re-established. Any possible reflex mediating through the vagi in the causation of vagus escape has been ruled out.

The discovery by Weber brothers (1845), that the vagus nerves are cardio-inhibitory is the first important step in our present day knowledge about the nervous regulation of action of the heart. Following this discovery a number of investigators working on different animals under different conditions in the later part of nineteenth century, not only demonstrated cardio-inhibitory action but also cardio-accelerator action of the mammalian vagus. Dale et al. (1910) also demonstrated accelerator action of the vagus in cats. Tulgan (1923) working on this problem reported that distribution of fibres in dogs and cats was different and separation could not be easily made out. Recently Choudhary et al. (1962) working on toads observed difference in the action of the vagus on both sides.

The present study has been aimed to have a comparative study of the effects of stimulation of both right and left vagus on heart in dogs and toads and to find out if any difference exists in their action.

METHOD AND MATERIAL

For all experimental work, toads and dogs have been used. Electrical stimulation was given by faradic current from a -Dubois- Reymonds induction coil. In toads, experiments were conducted in five different series. In first four series different strengths of currents have been used and effects of right and left vagus on toad's heart have been recorded. In the fifth series, effect of vagal stimulation has been studied on atropinized hearts. The heart beats were recorded on a smoked paper fixed on a Kymograph, moving at a slow speed. In dogs, heart rate was

recorded by using a Hurthle's manometer connected to the left common carotid artery and tracings were taken on a smoked paper fixed on a Kymograph, at a medium speed. The vagi were exposed, sectioned and sufficient time was allowed for stabilisation of the heart and then experiments were carried out. The experiments were conducted both in atropinized and non-atropinized dogs, using different intensities of currents. During experimental period, heart was kept constantly bathed with Ringer's solution.

RESULTS & DISCUSSION

The effect of stimulating right and left vagus using different strengths of stimuli has been studied in normal and atropinized heart of toads and dogs.

It was observed that when the vagi are cut in toads and dogs, tachycardia develops. It shows that the vagus remains in a state of tone. Tachycardia following removal of vagal influence develops though the stellate ganglia have been removed previously, thereby showing that it is not due to an increased action of accelerator nerves (Best & Taylor 1961). Hunt (1899) and Gasser and Meek (1914) have suggested that fibres also exert a continual tonic influence as the removal of stellate ganglia with the vagi intact causes marked fall in heart rate.

In toads it was observed that on stimulating the vagi, when the distance between the primary and secondary coil varied from 8 to 12 cm. with a current strength of 2 volts in the primary circuit, it was possible to bring the heart to standstill in all experiments. When the distance of the secondary coil was between 16 cm. to 20 cm. variation in result occurred. It was also observed that when the stimulation of the vagus of one side was not effective, stimulation of the other vagus made the heart standstill. This is possibly due to unequal distribution of cardio-inhibitory fibres in the vagi of both sides. Amin & Best (1961) and Choudhary et al. (1962) have suggested unequal distribution of cardio-inhibitory fibres in vagi on both sides.

It was also observed that with weaker currents greater number of hearts showed vagus escape. When the strength of the current was gradually increased the number of hearts showing vagus escape became much less. However, even with secondary coil at 8 cm. distance three animals showed vagus escape on both sides. The animals which showed vagus escape with stronger currents may be due to varied threshold of responsiveness in different animals. In the vigorously beating hearts, vagus escape occurred immediately even with stronger currents. This may be as suggested by Hough (1895) that weaker the heart, the more effective is vagus stimulation. However, if the vagus escape depended entirely on the activity of the heart, the vagus escape should occur on both sides. As this was not the case, it may be explained on the basis of unequal distribution of cardio-inhibitory fibres on both sides.

It was also observed that the asystolic period of the heart varied from 2 to 200 seconds according to the intensity of the stimulus. However, there were definite variations in this asystolic period on stimulating the left or the right vagus in the same animal. This variation was not constant on any side in all the animals. This variation in the asystolic period on two sides is possibly due to selective distribution of chronotropic and dromotropic fibres in the two vagi as was suggested by Amin and best (1961). This may also be due to unequal distribution of parasympathetic and sympathetic fibres in the two vagi (Amin and Best, 1961; Chowdhary et al. (1962).

Cessation of stimulation of the vagus was usually followed by acceleration and augmentation of the heart before returning to normal. In most of the animals of all series augmentation was more common whereas acceleration was observed only in a few cases. In a few cases no change was observed which may possibly be due to nutritional changes produced in the cardiac muscle due to prolonged standstill of the heart as suggested by Hough (1895). A few also showed bradycardia and diminution in amplitude due to delayed inhibitory action of the vagus. Dale et al. (1910) explained this delayed and prolonged action of the vagus due to weak paralytic action at the synaptic junction between the pre and post ganglionic neurons or on the cell of the latter.

In the last series of experiments the heart was first atropinized with 10^{-6} atropine sulphate solution and then the vagi were stimulated. Following atropinization increase in heart rate was observed as usual. In most of the cases stimulation of the vagus, after atropine, caused slight increase in heart rate but the amplitude of contraction was markedly increased. This was more with the left than with the right vagus. In many cases with marked increase in amplitude, there was slowing of the heart. Middleton et al. (1949) remarked that vagal cardiostimulation consisted primarily of amplitude of contraction though a positive chronotropic effect may also be seen. The difference in amplitude between the two sides may be due to unequal distribution of sympathetic fibres, probably left vagus having more sympathetic fibres.

The effect of stimulating the vagi with different intensities of stimuli has also been studied in 11 anaesthetised dogs. With weaker stimuli usually there was no effect but one dog showed increase in rate during stimulation. On increasing the intensity of stimulus, the amplitude of contraction showed increase with no change in heart rate. In one dog the increase in amplitude was observed after cessation of stimulation. On further increasing the strength of stimulus there was slowing of heart rate with diminution in amplitude. After cessation of stimulation amplitude increased in most cases. These results can be explained on the basis of Reed and Layman's (1930) observations that the accelerator components of the vagus are in a low threshold irritability which makes possible accelerator responses when the current is subminimal for the inhibitory mechanism. In dog No. 7

cessation of left vagus stimulation was followed with diminution in amplitude of contraction as in toads.

In the last series, the vagi were stimulated after injecting atropine sulphate 0.1 mg./kg body weight intravenously. Atropine at this strength paralyses the vagal endings but usually does not affect the cardiac muscle. Following atropinization the heart rate increased. As in toads stimulation of the vagi resulted in slight increase in heart rate but amplitude was definitely increased on both sides in all cases. Both vagi usually have similar cardiostimulating effect, but occasionally the right or the left may have greater cardio-stimulating effect. Vagal cardiostimulation consists mainly in an increased amplitude of contractions, though, however, a positive chronotropic effect is also observed.

In spite of the extensive study by various workers the exact mechanism concerned in vagus escape is still obscure. Though several theories have been put forward by different workers, still none seems to explain the exact mechanism concerned satisfactorily. As the present experiments have been conducted in pithed toads and anaesthetised dogs with both vagi cut, any reflex mediating through the vagi can be ruled out. It has been observed that some variation in the time interval exists in the appearance of vagus escape on stimulating the right and the left vagus. How refractoriness of the heart to acetylcholine or increased destruction of acetylcholine or exhaustion may cause appearance of vagus escape earlier on one side than the other is difficult to understand. However what seems more probable cause of vagus escape is the development of idioventricular rhythm. Other factors, such as adrenaline and sympathetic nerves, stretching of the nodal tissue due to increased central venous and arterial pressure during vagal stimulation may also contribute further to cause of vagus escape.

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