VAGAL TONE*

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Weber brothers (19) were first to show that vagus nerve is cardio-inhibitory. They first demonstrated the exact situation of cardiac vagal centre in the floor of the fourth ventricle, and identified the centre with the dorsal motor nucleus of vagus. Hoff *et al.* (9), by their transaction studies, concluded that upper pontine structures exert an inhibitory influence on the vagal nuclei of medulla. Truex (18) in his book of human neuronanatomy described the vagal nuclei in greater details. He described that the dorsal motor nucleus of vagus occupies the medical portion of trigonum vagi. The nucleus is composed of relatively small spindle shaped cells, among which are large ones, with coarse chromofilic bodies and scattered pigmented cells. The nucleus in connection with the thalamus and cortex since the emotional states give rise to changes in the heart rate. Glasser (6) supported the concept of a cardiovascular augmentor area in the caudal pons and cardio-vascular depressor area in a more rostral portion of the brain stem.

TONIC ACTION OF NERVES

Marey (13) drew attention to the inverse relation between the heart rate and the systemic blood pressure level, whereas Franck followed by a score of others believed that the systemic blood pressure level directly excited the cardiac vagal centre. Hunt for the first time postulated that the heart rate is controlled by tonic and reciprocal action of the accelerator and inhibitory centres and the nerves exert a continuous restraint upon the actions of the heart. He noted the importance of diminution of vagal tone in reflex acceleration of the heart. But Hering disagreed with the statement of Marey and showed that relationship resulted from the reflexo-genic sino-aortic areas. Clarke (4) observed marked tonic activity of vagus in normal animals adopted by training to endure prolonged exercise. The pulse rate in tame rabbit was fast (approximately-205 per minute) and the heart was small. Section of vagi produced only a relatively slight acceleration (an increase to 320 or change of 50%) since their tonic activity was low. But he observed that the resting pulse rate of hare was much slower (64 per minute), the heart was larger and effect of vagal section was considerable (an increase to 264 or a change of 312%). Pulse rate was slow in race horses and in atheletes. Clarke concluded that this slow pulse rate was a true bradycardia and was accompanied by normal electrocardiogram. McDowell (12) after studying the activity of cardioinhibitory centre said that sensory stimulation and asphyxia caused cardiac acceleration, with inhibition of cardio-inhibitory reflexes. Subsequently the vagal centre become more

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excitable and the normal vagal restraint on the heart is increased, due to the after discha and facilitation of cardio-inhibitory centre. McDowell stated that this may be the cause relatively slow pulse rate in atheletes.

Samaan (16) demonstrated a sudden increase of heart rate almost synchronously wi the onset of work in normal dogs due to nervous influence on heart and not due to a physical or the chemical change in the blood. He added that the cardiac acceleration is n due to cardio-accelerator tone as the latent period of heart in response to cardiac sympathet impulses is about 3 to 7 seconds. This was also confirmed by Hunt. Samman conclude that the increase in heart rate at the beginning of the exercise is due to inhibition of vag tone for which he quoted the certain evidence in favour of his statement. (i) with the ons of work, sinus arrythmia disappears and respiratory movements are increased. The rap breathing produces reduction in vagal tone. (ii) Shortening of P-R and R-T intervals of ele trocardiogram. He noted that whenever vagal tone was eliminated by injecting atropin (0.2 *mg/kg*) in experimental dogs, no initial tachycardia appeared. He said that persis tance of cardiac acceleration throughout work is also associated with marked inbition of vag impulses. He confirmed this by cutting both the vagi when the tachycardia was maximum and observed that the heart rate increased only by 22 per minute, on the other hand vagotomy a rest was followed by an increase of 110 to 130 beats per minute. Brougha *et al.* (3) confirmed the findings of Samman and stated that cardiac acceleration during exercise is due to reductio in the tonicity of the cardio-inhibitor effect of the vagi. Recently Shepard and Whitty (17) studies the effect of bilateral cervical vagotomy in unanaesthetised dogs. They found change in heart rate, blood pressure and respiration and a progressive deterioration of cardiac func tions. Anrep *et al.* (1) showed in innervated heart lung preparation that cardiorespirator rhythm is very complex and have many central and complex reflex components. He further stated that it is mainly the vagal tone in early inspiration which is responsible for increase in the heart rate at that time.

Bond (2) studied the problem of sympathetic and vagal interaction in emotional responses of the heart rate. He described some accelerating mechanism in the vagus. To prove this he deprived the dogs of the accelerators and excluded the effect of adrenaline, even then there was acceleration of the heart rate upon emotional stimulation. This rate never exceeded the rate of denervated heart, except for the first few fast beats. He explained that main cause of increase heart rate was due to inhibition of vagal tone. Bouckaer and Haymans agreed with Bond but disagreed with the findings of Brough *et al.* (3) that there is high degree of cardiac acceleration in response to emotioned stimulation in dogs in whom the sympathetic chain was removed from stellate ganglion to secral ganglion.

Carlsten studied the vagal tone of heart in man during total neck dissection by blocking normal tonic discharge of the fibre by injecting xylocaine in the nerve. By comparing the heart response to known stimulating rates, with heart rate before nerve block; they concluded that normally present vagal tone could be roughly evaluated to about 2 to 4 impulses per second.

ATROPINE AND VAGAL TONE

Cayon and Ludwing (5) demonstrated that bradycardia caused by stimulation of depressor nerve can be abolished by atropine. Mines (11) applied atropine on the sinus venosus of the frog and found out that the effect of vagal stimulation on the heart was eliminated. MeGuigan (14) reported that small parentral doses of atropine (0.3 to 0.6 *mg.*) cause bradycardia. This was due to the stimulant action of drug on medullary vagal nuclei. Lewis (10) showed that the vagal inhibition of the heart is blocked by atropine and there is release of sympathetic accelerator tone and subsequently there is increase in heart rate.

Herx-Heimer (7) reported that the subcutaneous injection of 1 *mgm.* atropine into 28 sportsmen was associated with an initial bradycardia followed by an average increase of 5.4 beats over the resting heart rate. Six of his subjects showed no cardiac acceleration. Kauf (9) also found that 1 *mgm.* atropine injected subcutaneously did not have a marked acceleratory effect on the resting heart rate of the trained individual and that this dosage level was associated with an initial bradycardia. Same dosage in untrained individual produced marked cardiac acceleration. Raab *et al.* (15) injected atropine 0.025 *mgm/kgm* body weight intravenously in trained and non-trained individuals but found no difference in their responses.

In our laboratory we studied the degree of vagal tone in 100 male, 100 female students and 30 rickshaw pullers.

Atropine 0.06 *mg./kg.* body weight was injected subcutaneously in male students and rickshaw pullers and 0.04 *mg./kg.* body weight in female students. The maximum increase in heart rate attained after atropine injection was recorded and the degree of vagal tone determined.

Vagal tone in rickshaw pullers was greater than in male students. The average vagal tone in rickshaw pullers was $103.52 \pm 30.58\%$ as compared to the average vagal tone of $68.32 \pm 24\%$ in the male students. Male students who were atheletes or were doing physical exercises regularly had a higher vagal tone as compared to the sedentary students. The vagal tone in the female students was the lowest. Average being $57.73 \pm 22\%$.

It seems that exercise tends to increase the vagal tone and is responsible for increasing the cardiac reserve and hence the efficiency of the cardiac muscle.

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