

REGULATION OF HEART RATE: EFFECT OF INFLATION OF LUNGS

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The effect of respiration on heart rate has been described as sinus arrhythmia in anaesthetized animals by Heymans (6). Each inspiration is accompanied by an acceleration and slowing accompanies every expiration. A sustained inflation of the lungs in dog has been shown to cause bradycardia (1) *i. e.* the opposite of the classical sinus arrhythmia. The above results have been obtained on animals under the artificialy produced conditions in the laboratory. The aim of the present study was to test the validity of the above observation on concious human subjects where the inflation of lungs is produced entirely by the voluntary effort of the inspiratory muscles instead of the respiratory pump and the responsiveness of the regulating centres is unhampered by anaesthetics. The results obtained in this way are presented below and an attempt has been made to explain the underlying physiology.

METERIALS AND METHODS

Ten healthy human subjects ranging from 18 to 32 years of age were experimented upon. The subjects had had the practice of breath holding after full inspiration earlier. The respiratory excursions of the chest were recorded stethographically by tying a corrugated rubber tube round the chest whose open end was connected by a rubber tube to a Marey's tambour which recorded on smoked paper on a moving drum. That the respiration was held at maximum inspiratory level was ensured by the subject who had to keep an eye on the lever and see that it recorded a horizontal line. This was latter confirmed by the examiner. A tendency to relax was at once recorded by moving up of the lever.

The heart rate was recorded electrocardiographically. The resting rate was compared with that during the period of breath holding in full inspiration while sitting and lying down. The resting rate was recorded only after it had been found constant for five minutes by counting the pulse at the wrist. The experiments were done in a quiet room with only the examiner and the subject there to avoid extraneous stimuli.

OBSERVATION AND RESULTS

Observation were made on these individuals sitting on a stool with legs hanging. Nine of them showed a fall in heart rate when breath is held after a maximum inspiration. 100 observations were recorded. Mean resting rate was found to be 81/min, values lying in the range 68-92 min (S. D. \pm 0.56). The mean rate during breath holding in maximum inspiration was found to be 71/min (S. D. \pm 0.50) range 60-80/min. The difference between the two, therefore, is statistically significant $t > 2.5$. One subject out of these ten showed a rise when tested sitting up on eight occasions. He was a male of small build, five feet tall having no sign of any physical disability.

Same subjects when tested in recumbent posture, showed a variable response; three showed an average acceleration of 7 beats per minute. Two showed no change and five showed a fall of 4-6 beats/min.

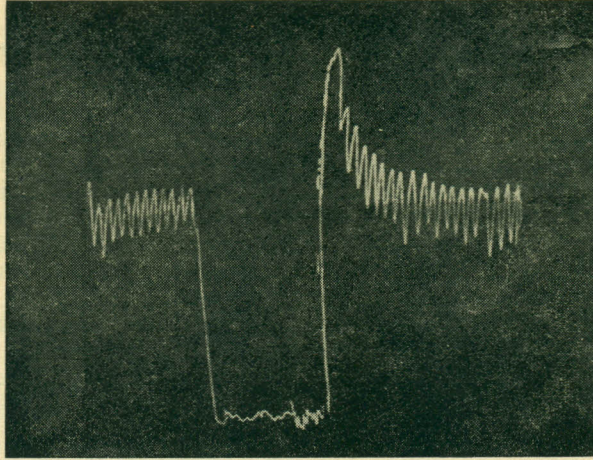


Fig. 1. The stethographic record of normal respiration followed by breath holding in maximum inspiration till the breaking point, the following expiration and the breaths thereafter.

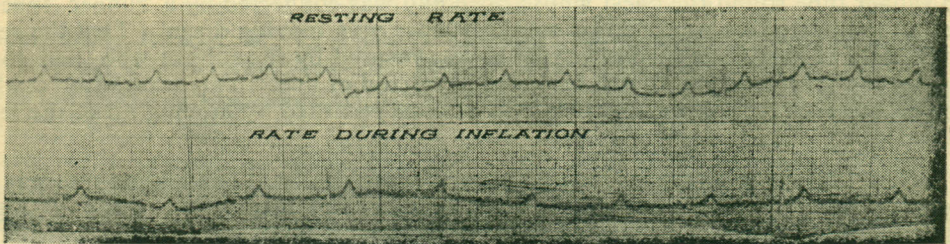


Fig. 2. Electrocardiographic recording of the heart rate during resting condition and during maximum inflation of lungs by voluntary effort.

DISCUSSION

The sinus arrhythmia i. e. the acceleration of heart rate during inspiration has been attributed to a twofold depression of the cardioinhibitory centre, namely the activity of the respiratory centre and the inflation reflex from the lungs initiated by the stretch receptors. It has been observed that a significant number of healthy individuals show a slowing down of the heart rate when breath is held after a maximum inspiration. Such response is more frequent when subjects are tested sitting up than in the recumbent posture. Slowing is also obtained while sitting during inspiration in a number of subjects who show acceleration while in recumbent posture,

The distribution of blood and venous drainage is different in the two positions of the body and an explanation for the different response should, therefore, be sought in the changes that are thus brought about. Inflation of the lungs causes subatmospheric pressure in the thoracic cavity and raises the pressure in the abdominal cavity. This increases the abdominal-thoracic pressure gradient which helps to draw blood out of the portal system and inferior venacava into the right atrium which to start with is also at its lowest pressure, being a thin walled chamber and subjected to the maximum stretch that the respiratory muscles are capable of exerting (3, 4, 11). The increased venous return, unless accompanied by simultaneous emptying of the right atrium to the same extent would increase the discharge of the atrial type *B* receptors (7). The right ventricle has an augmented diastolic volume and therefore increased stroke volume according to the Starling's law. Regarding the efficient emptying of the right side it is worth while considering the resistance in the pulmonary circuit. Maximum inflation of lungs has been shown to increase the size of the big and medium size arteries and veins whose walls are subjected to pull from all sides. The effect on the small capillaries in the walls of the alveoli are however the opposite. They are compressed by the stretched alveoli from all sides and consequently narrowed considerably or are completely obliterated depending on the degree of inflation of the alveoli (8). The reduction in the pulmonary bed leads to an increase in resistance offered to the right ventricular outflow, hence a rise of pulmonary artery pressure.

Since the breath is held for about 35-45 seconds, the above effects tend to have a cumulative effect *i. e.* the venous return to the thorax is increased leading to increased filling of right atrium, more volume available for the right ventricle, increased stroke volume of right ventricle and a rise of pressure in the pulmonary circuit. Thus a series of depressor reflexes are set up from the right atrium, right ventricle and pulmonary artery as a result of the above stimuli, the vagus nerve endings acting as the afferents (2).

The cardio-acceleration accompanying inspiration has been explained by Anrep *et al* (1) as due to an inhibition of the vagus centre by impulses from the inspiratory centre and from the stretch receptors of the lungs. They feel that this acceleration tends to be reduced by the Hering Breuer inflation reflex which inhibits the inspiratory centre and thus cuts off the irradiation of motor impulses to the cardioinhibitory centre. How much is the contribution of these central effects under the above said experimental conditions is difficult to say but the depressor reflexes mentioned above could certainly be held responsible for the slowing of heart rate observed in these cases. Further in these experiments, certainly the Hering Breuer inflation reflex sends inhibitory impulse to the inspiratory centre whose activity is being kept up by a voluntary effort originating in the cerebral cortex. Their contribution in conscious subjects is impossible to judge, but certainly these are masked by the depressor reflexes already described resulting in bradycardia.

Regarding the difference of response observed in the same subjects in recumbent and sitting positions it can be argued that while lying down, by the moving up of the diaphragm and there being already more blood in the thoracic cavity the expansion of lungs is not so much as in the sitting posture (5). Thus the respiratory pump for venous return is not so efficient and as said earlier, the depressor reflexes are all dependent on that. This effect being reduced, probably the central effect gets an upper hand and thus cardio-acceleration results.

Van saalfield (10) showed that the effects of inflation of lungs on circulation are mediated *via* the vagus but they are eliminated when the experiments are done after

cocainizing the pleura in the isolated perfused lung preparation. The response in intact conscious human beings seems to be different from above. If the contribution of pleural endings is the same in both the postures, then one can assume that the cardio-inhibition is certainly a result of the depressor reflexes from the cardiovascular territories. Reflex bradycardia from extreme inflation of lungs has been shown to occur in lung perfusion experiments on dogs (1).

SUMMARY AND CONCLUSIONS

Effect of maximum inflation of lungs by voluntary effort on the heart rate has been studied in conscious human subjects and the series of depressor reflexes that are set up have been described. It has been shown that inflation in sitting posture causes bradycardia while the response is varied in recumbent posture. Thus while describing the effect of respiration on heart rate it is advisable to keep in view that sinus arrhythmia was observed under a particular set of experimental conditions and it certainly is not the physiological response in conscious human subjects.

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