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An Experimentally Produced Bigeminal Arhythmia in Rabbits.*

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Confirmation is made of Kratschmer's[1] and Koblanck and Roeder's[2] observations that a premature systolic arhythmia occurs in tracheotomized rabbits after insufflation of irritating vapors and from mechanical and faradic stimulations of the nasal septum before and after double vagotomy. It also follows faradic stimulation of the central end the vagus and insufflation of benzol into the trachea. In one instance it came on while the small intestine was being uncoiled. It does not follow faradic stimulation of the central end of the lingual, phrenic, depressor, ulnar and sciatic nerves or the peripheral end of the vagus and cervical sympathetic trunks.

Simultaneous apical beat, carotid pressure and vena cava tracings show the right atrium to be beating normally while the left ventricle has a bigeminal arhythmia and electrocardiograms demonstrate this arhythmia to be of left ventricular origin.

It would appear that this arhythmia is not due to a direct peripheral nerve reflex from 2 observations: (a) that it never comes on immediately with the insufflation stimulation as is the case of the arrested respiration and the rise in blood pressure; (b) that it requires a very powerful stimulation or sometimes a number of stimulations to produce it.

In view of the fact that this bigeminal arhythmia normally follows an arrested respiration, a rise in blood pressure and a slowed and strength-pulse it is necessary to show that these secondary factors and certain resulting internal secretions are not the actual cause of the arhythmia.

That an arrest of respiration did not elicit this arhythmia from asphyxia or lack of stimulations from failure to breathe is shown by experiments where no bigeminal pulse results from occlusion of the trachea or from inhalation of air highly concentrated with carbon dioxide. On the other hand this 'insufflation' arhythmia is readily obtained in rabbits having their thoraces opened on both sides and

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[1] Kratschmer, *Kreislauf. Sitzungsb. d. Wiener Akad.*, 1870, **62**, 147.

[2] Koblanck und Roeder, *Pflüger's Arch.*, 1908, **125**, 377.

breathing by artificial respiration. Apnea following forced artificial respiration has not resulted in a bigeminal pulse.

Numerous very similar and more pronounced rises in blood pressure than those which follow insufflations of benzol have been produced from occlusion of the abdominal aorta and from intravenous injection of adrenalin without evoking an arhythmia. On the other hand this arhythmia has been obtained from benzol insufflation when blood pressure has been prevented from rising by the use of an equalizer, the intravenous injection of ergotamine and from sectioning the spinal cord in the middle thoracic region. Many bigeminal pulse records have been obtained from insufflations without being preceded by any pulse changes; while the enormous slowing and strengthening of the pulse following occlusion of the trachea has never resulted in a bigeminal arhythmia. It is obvious also that the normal changes in blood pressure during insufflations are not contributory to anemia of the brain.

All humoral factors such as increased secretions from the adrenals, thyroid, liver, etc., are excluded as factors for producing this arhythmia by several cross circulation experiments, where the inner juxtaposed carotids of 2 rabbits were joined. An arhythmia produced in one animal from insufflation was never carried over to the other by means of the circulation.

That the carotid sinus of Herring is not the peripheral center for producing this arhythmia is shown by the absence of a bigeminal pulse in all of the mechanically produced rises in blood pressure and by a large number of 'insufflation' arhythmias obtained from the femoral arteries after both carotid sinuses had been removed.

That an impulse or series of impulses capable in some manner of producing a bigeminal pulse (probably arising somewhere in the *formatio reticularis* of the brain stem) descends the spinal cord to the left ventricle by way of the sympathetics is demonstrated by the following tests: (1) Transecting the spinal cord at the level of the 6 C vertebra always blocked an arhythmia from benzol insufflation; while sectioning the spinal cord from the 3-6 T vertebrae sometimes permitted an 'insufflation' arhythmia and sectioning the spinal cord at the 7-9 T always permitted the 'insufflation' arhythmia. (2) Removal of both stellate ganglia always blocked the 'insufflation' arhythmia, while removal of one stellate and sectioning the opposite sympathetic cord below the stellate generally permitted the 'insufflation' arhythmia. (3) Transecting the spinal roots at the level of the 7 C, 1 and 2 T also blocked the 'insufflation' arhythmia.

Over one hundred animals were used in the various phases of this problem.