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Contributors

Albert Abrams

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as well as a few scattered nodules. Some of the nodules had coalesced, while others were isolated. The individual masses were from 2 mm. to 5 mm. in diameter. Upon the superior surface of the diaphragm were small nodules ranging in size from 3 mm. to 5 mm. None were caseous. Several masses were seen upon the stomach and several upon the liver. Spreads and cultures were positive for acid-fast bacilli.

RAT No. 4.—Inoculated with the butter bacillus of Grassberger. Weight at time of inoculation 90 gm.; at death 185 gm. Scattered throughout the peritoneal cavity were numerous small nodules of a whitish color. A few small masses were present upon the inferior surfaces of the liver and diaphragm. Recent adhesions were present between the diaphragm and the liver. None of the masses was more than 5 mm. in diameter. In addition to the lesions just mentioned, a small cyst, which contained a tapeworm 30 cm. in length, was present in the liver. Spreads and inoculations from the lesions contained acid-fast bacteria.

RAT No. 5.—Inoculated with Moeller's grass bacillus No. 2. Weight at time of inoculation 210 gm.; at death 212 gm. Upon the abdominal wall at the site of inoculation numerous small nodules were present. Throughout the peritoneum were many spheric masses varying from 5 mm. to .5 cm. in diameter, most of them firm and dense, while some were quite soft and resembled caseous material. A few firm nodules were also observed around the liver and spleen and upon the inferior surface of the diaphragm. Spreads and cultures were positive for acid-fast bacilli.

RAT No. 6.—Inoculated with the mist bacillus. Weight at time of inoculation 212 gm.; at death 220 gm. Upon the belly wall small, firm nodules were found. Small, firm masses were abundant in the peritoneum; some were present in the liver, others between the liver and the stomach and between the spleen and stomach, ranging in size from 5 mm. to 1 cm. in diameter. Intimately adherent to the small intestines was a firm, grayish-white mass, irregularly nodular, measuring 1.5 cm. in length and 8 mm. in thickness. The left testicle was enlarged, swollen and adherent: upon section it was caseous (?). Spreads and inoculations from all the lesions contained acid-fast bacteria.

RAT No. 7.—Inoculated with the margarin bacillus. Weight at time of inoculation, 209 gm.; at death, 198 gm. Numerous very firm nodules were observed in the peritoneum, especially in the region of the liver and spleen, and few were seen in the hepatic structure. A few adhesions extended from the nodules upon the liver to similar structures upon the inferior surface of the diaphragm. These nodules varied from 3 mm. to 5 mm. in diameter. No bacteria were obtained either from spreads or in cultures.

RAT No. 8.—Inoculated with the blindschleichen bacillus. Weight at time of inoculation, 180 gm.; at time of death, 182 gm. Two nodules, one caseous, were found in the abdominal wall near the site of inoculation. There were numerous tubercles in the peritoneum from 3 mm. to 6 mm. in diameter, none of which was caseous. Around the spleen and between the liver and stomach nodules were also evident. Acid-fast bacilli were obtained both in spreads and cultures.

RAT No. 9.—Inoculated with *B. tuberculosis piscum*. Weight at time of inoculation, 215 gm.; at death, 225 gm. Scattered throughout the peritoneal cavity were nodules varying in size from 5 mm. to 1 cm., some firm and fibrous, others caseous. The right kidney was adherent to the right lobe of the liver, and showed numerous whitish nodules upon its surface. Tubercles were also present between the liver and stomach, and between the latter organ and the spleen. Imbedded in the liver parenchyma were several small nodules 3 mm. to 5 mm. in diameter. The under surface of the diaphragm was completely studded with small firm tubercles, 2 mm. to 4 mm. in diameter. Acid-fast bacilli were obtained in spreads and cultures.

RAT No. 10.—Inoculated with the grass bacillus of Korn, No. 2. Weight at time of inoculation, 210 gm.; at death, 220 gm. Only a few small masses were present in the peritoneal cavity, and one large nodule, 1.5 cm. in diameter, the center of which was distinctly caseous. A few nodules, ranging in size from 2 mm. to 6 mm., were also present between the liver and stomach and around the spleen. Spreads and cultures contained acid-fast bacilli.

RAT No. 11.—Inoculated with the grass bacillus of Moeller, No. 1. Weight at time of inoculation, 185 gm.; at time of death, 200 gm. Nodules were abundant in the peritoneal cavity, and a small number were also seen in the liver and around the spleen. The under surface of the diaphragm contained a few masses, none caseous. No acid-fast bacilli were obtained in cultures or in spreads.

RAT No. 12.—Inoculated with Karlinski's bacillus. Weight at time of inoculation, 195 gm.; at death, 210 gm. In the peritoneal cavity was a large agglutinated mass made of small, firm, grayish-white nodules, extending about the liver, stomach and spleen. Tubercles were observed upon the inferior surface of the diaphragm and in the liver, but none showed signs of softening. Acid-fast bacilli were obtained in cultures and spreads.

RAT No. 13.—Inoculated with the milch bacillus. Weight at time of inoculation, 200 gm.; at death, 210 gm. The abdominal wall contained a small nodule near the site of inoculation. The peritoneum contained a few small nodules; none was caseous and none measured more than 4 mm. in diameter. A small number of masses were present around the spleen and between the liver and stomach. Cultures and spreads were entirely negative for acid-fast bacilli.

From these notes, it will be observed, that tubercles or tuberculiform nodules were produced in every case, and in one instance there was effusion into the peritoneal cavity. To me, the naked-eye appearances of these lesions resemble those of similar lesions brought about by tubercle bacilli, and only by cultures and histologic examination can the true nature of the processes be determined. True caseation was not constant, and when this degeneration did take place, the substance formed did not respond to stains, as does the caseous material in a frank tuberculous process, due to true tubercle bacilli.

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THE VAGOVISCERAL REFLEXES WITH SPECIAL REFERENCE TO THE VAGOSTOMACH REFLEX.

BY

ALBERT ABRAMS, A.M., M.D.,

of San Francisco, Cal.

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The vagus nerve may be stimulated in its cervical course, and such stimulation is available for clinical purposes. Czermak was able to press his vagus nerve against a little bony tumor in the neck, and by thus subjecting the nerve to mechanical stimulation was able to slow or even stop the beating of his own heart. This was the first demonstration in a healthy person of a fact already known to the physiologist. If, in a healthy person, the carotid artery, or a point immediately adjacent to it in the neck, is compressed, as a rule, slowing or complete inhibition of the heart action and pulse ensues. The latter may be made to disappear for fully 75 seconds; with prolongation of the pressure, the cardiac function is resumed. Such pressure may induce vertigo or syncope. Not infrequently the preceding phenomena are only observed after pressure is made on both vagi, in other instances, after pressure on one vagus, which is usually the right vagus. Many persons can voluntarily suspend heart action, and among Indian sorcerers, the phenomenon is regarded as a marvelous feat. Donders solved the riddle as follows: By voluntary contraction of the neck muscles, innervated by the nervous accessorius, the branches of the latter running in the vagus path are irritated, resulting in temporary stoppage of the heart action. I published a contribution[1] on "Inhibition of the Heart as an Aid in Diagnosis" in which I demonstrated, after many observations, that heart inhibition could be best effected for clinical purposes by directing the patient to draw the head slowly backward, though forcibly, thus inducing hypertension of the cervical muscles. During the time hypertension of the cervical musculature is maintained, the vagovisceral reflexes persist. Usually a few seconds elapse before the reflexes become manifest after forcible extension of the neck. In the contribution on "Cardiac Inhibition" I formulated the following conclusions:

1. The inhibition maneuver will cause organic cardiac murmurs to become faint, and in exceptional cases will render them inaudible.
2. Transmitted murmurs are more amenable to the maneuver.
3. The fainter the murmur, the more easily is it suppressed by the maneuver.
4. When a transmitted murmur can be inhibited, the tone which it masks can be auscultated.
5. Heart tones are less amenable than are heart murmurs to inhibition.
6. Hemic murmurs are more readily inhibited than are the organic murmurs.
7. As a rule, the murmurs of anemia may be suppressed and their evanescence is marked by the reappearance of tones.
8. Exocardial murmurs are easily influenced by the inhibition maneuver.

9. When the inhibtion maneuver is incorrectly executed, the result will be to increase the intensity of the murmurs, owing to increased exertion which intensifies the force of the heart's action.

10. The inhibition maneuver, when often repeated, is futile in its results, owing to overstimulation of the vagi.

11. In irregular cadiac action or in delirium cordis, the inhibition maneuver, by momentarily inhibiting the rapidity of the heart, renders signal service in determining the time of a murmur; the maneuver being practically in its effects like the physiologic action of digitalis on the heart.

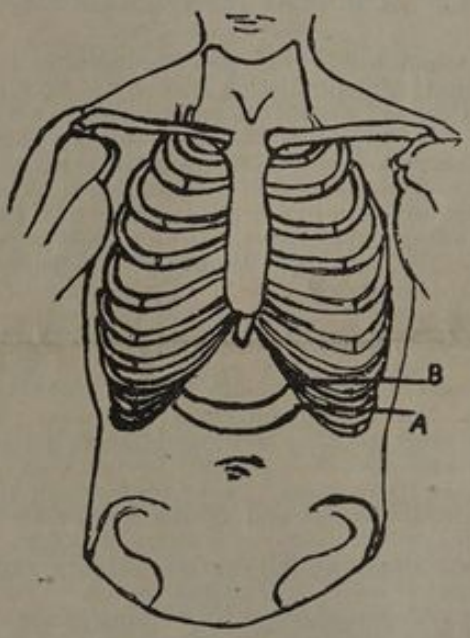
12. The inhibition maneuver enables us to determine the condition of the vagi as inhibitors of the heart and guides us in the administration of cardiotonics.

Later,[2] I described another vagovisceral reflex under the title, "The Tracheal Traction Test as an Aid in the Recognition of the Asthmatic Lung." Some of the conclusions then summarized were:

1. When the head is thrown forcibly backward, the normal resonance obtained by percussion over the manubrium sterni and lungs contiguous thereto becomes translated into a dull or flat sound. This maneuver I called the tracheal traction test.

2. The tracheal traction test is positive in health and in all cardiopulmonary affections, but it is negative in cases of idiopathic asthma.

3. The recognition of this test, which is always present in the interparoxysmal period, affords a valuable aid in the diagnosis of idiopathic asthma and assists in its differentiation from symptomatic asthma and other spasmodic affections which suggest an asthmatic genesis.



4. The maneuver specified as tracheal traction evokes contraction of the bronchial muscle by stimulation of the pneumogastric nerves.

5. In asthma the tone of the bronchial muscle is so reduced that it no longer responds to vagus stimulation brought about when the neck is forcibly extended on the sternum; hence the tracheal traction test in idiopathic asthma is negative.

I explained the dull sound supplanting the resonance in the normal subject by tracheal traction by supposing that, owing to contraction of the bronchial muscle, the air in the trachea and bronchi is under considerable tension, the pitch becomes higher and the volume and intensity so decreased that, while percussion formerly yielded resonance, the same act now elicits a dull or even flat sound. More recently, I have observed that while tracheal traction is maintained, dull areas on percussion may often be evoked over the anterior chest, but notably in the lower lung lobes posteriorly.

The third conclusion must be modified to this extent. I have described[3] a condition under the designation "Spasmodic Bronchostenosis," which is practically asthma without paroxysms. Patients with bronchospasm suffer from a persistent spasmodic cough, with or without expectoration, usually without, which rarely yields to conventional medication. Bronchospasm is specially influenced, like asthma, by climatic conditions. Auscultation of the chest in a bronchospasmodic patient elicits rales. These rales, as I have suggested,[4] may be differentiated from the rales of bronchitis by the inhalation of a few drops of amyl nitrite. Such inhalation carried to its physiologic effect will temporarily dispel rales due to bronchial spasm, while on the rales of bronchitis no effect is produced. In spasmodic bronchostenosis, like in true asthma, the tracheal traction test is negative. Before reference is made to the vagostomach reflex, I wish briefly to refer to one of my stomach reflexes.[5]

The "stomach reflex of contraction" is elicited by placing the palm of the left hand in direct contact with the Traube area, that half-moon shaped space which normally yields on percussion a tympanitic sound, owing to the presence of the cardiac end of the stomach. The Traube area is bounded above and laterally by the contiguous borders of the liver, lung and spleen. After placing the palm according to the method indicated, one strikes the fingers of the left hand with the clenched fist of the right hand (avoiding the employment of the knuckles, which is painful) a series of vigorous blows. Percussion of the entire stomach region now elicits a tympanitically dull sound, and after this manner the lower stomach border may be accurately defined. This reflex, as well as the vagostomach reflex, is best elicited with the patient standing. The reflex is of brief duration, hence if percussion of the lower stomach border has not been obtained in its entirety, repercussion of the Traube area must be made. This stomach reflex has been controlled by the röntgen rays, the gastrodiaphane and by other conventional methods and found to be correct. There is one drawback to accuracy, however, and that is that the lower stomach border in the normal will recede from its usual position from about 2 cm. to 4 cm.

We will note presently that the latter fact suggests a method for determining the motor power of the stomach. To explain the altered percussion sound in the stomach reflex of contraction, we must have recourse to the Skodaic interpretation of the condition which exists when dulness supplants tympanicity. In the stomach reflex of contraction, the gastric walls become tense, thus putting the air or gas within them under increased tension, and, for this reason, we have the physical elements necessary for the transition of a tympanitic to a dull sound.

The vagostomach reflex enables us to define accurately the lower stomach border without the concomitant dislocation of the border which attends the elicitation of the stomach reflex of contraction. All that is necessary is to percuss the lower stomach border during the time the patient forcibly extends his head as far back as possible. When he is unable to do this satisfactorily, an assistant may do it for him. That is practically all there is to this reflex. Comparing it with the stomach reflex of contraction, reference is made to the accompanying illustration. A, signifies the lower stomach border elicited by percussion during the time the head is forcibly extended; B, signifies the lower border of the stomach after concussion of the Traube area. The difference in the distance between lines *A* and *B* represents the degree of stomach contraction and is in direct ratio to the motor power of the organ. Both stomach reflexes are practically constant in the normal, but in anomalous abdominal conditions may be considerably influenced to the extent of annihilation. Having the patient incline his body backward after my method[6] of eliciting the lower liver border accentuates the dulness of the lower stomach border in both reflexes.

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