

nerves derived from the affected cord-area, yield symmetrical masses of fat.

As to the practical value of the occlusion of blood vessels by the metal band, I have in mind the gradual occlusion of large arteries, particularly the aorta, abdominal and thoracic. A vessel partly closed by a nicely rolled band may subsequently be completely occluded either spontaneously (ideal result) or by the pressure of the fingers or of a forceps on the band. It is conceivable that in some instances this subsequent tightening of the band might be accomplished subcutaneously, but usually a second, though comparatively insignificant, operation would be required. Aortic aneurisms if situated too high for the subdiaphragmatic application of the band might be cured by a band on the thoracic aorta. To apply a band to the thoracic aorta is not difficult and may be executed rapidly without the excision of a rib. When it may be necessary to test the effect of blocking an artery before permanently occluding it, as in carotid, popliteal and high femoral ligations, the metal band might be desirable because (1) it serves the purpose of both clamp and ligature; (2) during the operation it may be safely removed if advisable, for the arterial wall is uninjured by it; (3) if too tightly rolled it may be removed at any time after the operation, even days thereafter, in case gangrene threatened or cerebral symptoms developed for, the arterial wall being uninjured, the normal lumen remains.

I have at present under observation a dog whose thoracic aorta has been experimentally occluded. The aluminum band was not rolled so tightly as completely to occlude the artery or to produce demonstrable weakness of the hind legs. The recovery from the operation was uneventful, but about three weeks thereafter paraplegia developed suddenly and, coincidentally, disappearance of the femoral pulse on both sides. I expect to find that complete occlusion of the thoracic aorta has been spontaneously accomplished, and in the manner above described. Partial occlusion, not becoming complete, might of itself occasionally cure an aneurism.

In the human subject I have partially occluded the innominate once and the common carotid four times, successfully, with the aluminum band. In a case of large popliteal aneurism I employed the metal band to occlude completely the femoral artery because this method enabled me particularly well to test the blood pressure during the gradual process of occlusion. In the case of a woman asphyxiated to unconsciousness by an aneurism of the aortic arch I exposed, carefully and freely and without puncturing either pleural cavity, the heart and the arch of the aorta, hoping possibly to be able to encircle with a band the aortic arch between the regions of the innominate and left carotid arteries, but the aneurism so involved the entire arch as to defeat the earnest endeavor to execute the procedure.

I may assume that it is not necessary to remind this audience of the results which have attended ligation of the human aorta. The most successful of these operations was performed in 1899 by Dr. W. W. Keen, whose patient, the thirteenth case, lived forty-three days.

DISCUSSION.

DR. W. W. KEEN, Philadelphia, said that when he commenced the operation, which he did about seven years ago, it was with the intention of getting at the sac safely and without injuring the abdominal viscera, in order to introduce wire into it, but when he found the rather long portion of the aorta apparently free from disease, he felt that it was "flying in the face of Providence" not to tie it, and he did so, making the thirteenth case on record. He regretted to say that the pro-

cedure was followed by a thirteenth death; but the patient lived for forty-three days, the longest period on record.

Since then Tillaux and Körte have tied the aorta twice more, making fifteen cases and fifteen deaths. After his own experience, when Dr. Keen had felt the enormous rebound of that aorta and the narrow ligature that we put on such vessels, he made up his mind that never again would he be tempted to tie the aorta in that way. And accordingly he devised an instrument for the purpose of its obliteration, not by a narrow ligature, but by a broad metal clamp, one clamp behind the artery and the other in front of it and adjustable by a screw.

The work done by Dr. Halsted, Dr. Keen said, certainly is most notable and it is precisely along the line of what he believes is going to bring success after the fifteen recorded failures, that is, that the aorta must be obliterated not narrowly, as by a ligature, but by a band in order that we shall avoid exactly what happened in one of Dr. Halsted's cases, that the edge of the ligature cuts through the vessel and as the result of this the animal died from secondary hemorrhage.

REDUPLICATION OF HEART SOUNDS.

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Reduplication means doubling; that is, the sounds still strike the ear, not as a single tone, but as a double tone. Both the first and the second sounds may be reduplicated. The appearance of extra beats is a totally distinct phenomenon.

This subject has received scanty attention. Potain, in a study of 500 hearts, makes the first mention in literature. In a certain percentage of normal hearts, he found a doubling of the first sound at the end of expiration and beginning of inspiration; of the second sound, at the end of inspiration and beginning of expiration. All later observers have agreed as to the accuracy of Potain's observation. But as to the interpretation of the phenomena the theories advanced are as variable as possible.

It seemed at once obvious that the reduplication must have a distinct relation to the respiratory cycle. Acting on this basis, Sibson¹ and Barr² engaged in a controversy. Each observer based his views on the observation of a limited number of cases. Each observer assumed that the heart sounds were reduplicated only during the appropriate respiratory cycle. As pointed out by Ohastzow,³ if these observers were correct, then all normal hearts should be reduplicated; furthermore, in many case the reduplication persists at all stages of respiration.

CAUSE OF REDUPLICATION.

Sansom⁴ considered that reduplication is due to asynchronous ventricular contraction. In support of his contention is a case of cleft sternum, direct cardiographic tracings showing that the right and left ventricles did not beat in unison in that particular case. Barr,² in an experiment on a cat, proved that the two ventricles do not beat synchronously when the chest is opened. Barr also advanced a case of adherent pericardium, with mitral stenosis and regurgitation. One lever placed near the xyphoid and another over the apex did not move in unison. Because of the adherent pericardium the experiment is of little value. A case is

1. Sibson: *Jour. of Anat. and Phys.*, 1880, p. 238; *Lancet*, 1874, pp. 438 and 506.

2. Barr: *Liverpool Med and Surg. Jour.*, July, 1882; also, *Med. Times and Gazette*, 1877.

3. Ohastzow: *Ztschr. f. Klin. Med.*, Berlin, 1905, vol. lvii, pp. 70 to 90.

4. Sansom: *Med. Times and Gazette*, 1881.

reported by Bindley⁵ in which by auscultation two first sounds were heard and by palpation a double apex impulse was felt. Ohastzow³ mentions a number of similar cases. Three such cases have occurred in my own series. Bindley⁵ attributed this to asynchronous ventricular contraction. Austin Flint⁶ agrees with Sansom that the cause of reduplication is asynchronism. Bramwell⁷ considered that reduplication was due to auricular sound. He is supported by Ohartzow.³ Whatever the cause of reduplication may be, it is a sign of very definite clinical value.

METHOD OF EXAMINATION.

The results appended are based on examination of 162 cases. In every possible case the record included: presence or absence of reduplication of the first and the second sounds; age, height and weight of the patient; general condition, past history and present illness; presence or absence of palpitation, cardiac pain, dyspnea, cyanosis and edema; situation of the apex and right border; presence or absence of murmurs, condition of the artery and the pulse; duration of disease; permanence of reduplication, whether following exercise or holding breath; presence or absence of an extreme degree of nervousness; habits of using tea, coffee, alcohol and tobacco. The cases were compiled at the Male Medical Out-patient Department of the Massachusetts General Hospital, in the wards of the same hospital, at the Boston City Hospital Relief Station, and at the Massachusetts State Hospital in Tewksbury. Whenever possible the cases were followed from day to day.

In bringing out a reduplication more than the ordinary care must be exercised. All surrounding conditions must be favorable. The heart should be auscultated both in the sitting and dorsal position. Furthermore, the heart should be accelerated. This is best brought about as follows: Patient takes a deep breath and holds it; the heart beats more slowly. When the slowing has reached the maximum he lets out his breath and holds it so; the heart rate is accelerated. Repeating this maneuver will cause the heart rate to be accelerated, sometimes up to 30 beats a minute. Patient may then breathe naturally, and the acceleration of the heart will persist for a short time. This is superior to exercise, as the heart beats are not increased in force, but only in rate. At this time, when the heart is racing, a reduplication can be heard, if at all. Frequently a reduplication heard only during the appropriate respiratory phase (as pointed out by Potain) persists throughout the entire cycle.

Reduplications of the first sound are heard best in the third and fourth intercostal spaces, just to the left of the sternum. Often it can be detected here when it can not be heard over the apex. Rarely the rule is reversed. Pronounced reduplication can be heard over the whole precordia and in the axilla. Reduplications of the second sound are heard best in the second and third intercostal spaces to the left of the sternum. The reduplication is best heard with the Bowles stethoscope, using the large-sized tambour. It can not be heard as well with the unaided ear.

Pathologic reduplications, and sometimes those heard in normal hearts, are commonly so clear that it is unnecessary to go through all these maneuvers.

RELATION OF THE SECOND SOUND TO THE FIRST SOUND.

The first sound is reduplicated much more frequently than the second. Not uncommonly both are reduplicated. With the exception of mitral stenosis and those conditions altering the relative blood pressure in the greater and the shorter circulations the second sound is reduplicated only when the first sound is reduplicated.

TRANSITORY REDUPLICATIONS.

Not infrequently the reduplication is present one day and gone the next. For example:

CASE 1.—M. D. had hypertrophy and dilatation of the heart. When first examined he was in very poor condition. The apex was two inches to the left of the nipple line, the right border three-fourths of an inch to the right of the sternum. There were no murmurs. Cyanosis and dyspnea were marked. Pulse was very irregular in volume and tension. Both the first and the second sounds were reduplicated. Two days following his condition was much improved. Cyanosis and dyspnea were markedly less. The reduplication was gone.

CASE 2.—E. had same disease and symptoms. There was reduplication of the first sound, not of the second. As his condition improved the reduplication became more difficult to hear, and finally disappeared on the tenth day, simultaneously with a much improved condition.

CASE 3.—A. J. had had typhoid and was convalescent. There was reduplication of the first sound for four days. On the fifth day it disappeared. On the sixth day he sat up for the first time, and reduplication of both the first and the second sounds appeared.

These three cases illustrate well the relation between the efficiency of the heart and the presence of reduplication.

RELATION BETWEEN REDUPLICATION AND GALLOP RHYTHM.

There is a very close relation between reduplication and gallop rhythm.

CASE 4.—Mrs. V. had myocarditis. There was extreme irregularity of the heart and gallop rhythm. Both the first and the second sounds were clearly reduplicated.

Ohastzow³ has shown that between the reduplication that can barely be heard in the normal heart and gallop rhythm there is a perfectly definite relation; that the barely perceptible reduplication stands at one extremity of the scale; gallop rhythm at the other. He classifies all cases of reduplication thus:

1. Reduplications barely heard in the dorsal position, with reference to respiration, i. e., for the first sound, at the end of expiration and beginning of inspiration; for the second sound, at the end of inspiration and beginning of expiration.

2. Reduplications also heard in the sitting posture, but only in the third and fourth intercostal spaces to the left of the sternum.

3. Reduplications heard at the apex throughout the respiratory cycle.

4. Palpable double apex impulse.

5. Gallop rhythm.

This classification is, in the most part, true, but there may be a very great difference in efficiency between a heart that exhibits a reduplication of the first sound with a double-apex impulse and an actual gallop rhythm. The classification is principally of value in illustrating the close relation between the reduplication heard in normal hearts and gallop rhythm. Gallop rhythm is merely an accentuation of this not uncommon sign.

5. Bindley: *Lancet*, 1881, p. 46.

6. Flint, Austin: "Auscultation of the Heart," 1890.

7. Bramwell: "Diseases of the Heart," 1884.

THE RELATION BETWEEN REDUPLICATION AND EXTRA BEATS.

The relation of reduplication to extra beats is very well illustrated by the case of gallop rhythm cited in the last paragraph.

CASE 4 (continued).—Mrs. V. had marked dyspnea, cyanosis and edema. Heart beat was tumultuous and irregular. In addition to the clear reduplication, there were extra beats, followed by no second sound. That is, at all times both the first and the second sound were reduplicated. Occasionally there was a third sound, heard best about 1 inch to the right of the apex, not reduplicated, and not followed by any second sound. This third sound differed entirely in quality from either of the two components of the first sound.

PHYSIOLOGIC REDUPLICATION.

It has been mentioned that the sounds of the normal heart may be reduplicated. Obviously, therefore, in settling the question of the cause of pathologic reduplication some means must be determined to rule out those cases that would, under normal conditions, present a reduplication.

Unfortunately, the criterion is one that depends, to a large degree, on the judgment of the examiner. Out of 77 hearts examined under conditions that ruled out any pathologic interference in their action, a reduplication was present in 41. In 13 of the 41 there was an excessive indulgence in tea, coffee, alcohol and tobacco, which, as will be proved subsequently, will cause reduplication. Of the 28 cases of reduplication remaining, all but one were found to be in persons with thin chest walls.

Examining the problem from the converse point of view, out of 31 persons with thin chest walls a reduplication was present in 27, absent in 4; out of 34 persons with thick chest walls a reduplication was present in 1, absent in 33; 12 cases were on the border line between thick and thin.

The criticism may be at once advanced, however, that in many of these cases it rested with the judgment of the examiner to determine whether a given chest wall might be classed as thick or thin. Wherever possible the patients' height and weight were recorded, and the judgment based on this evidence. So many cases are on record in which there is no possible chance of error as to the thickness of the chest wall that this rule is proven. Given a patient with a thin chest wall, a reduplication to some degree can be heard; if the chest wall be thick, no reduplication can be heard under normal conditions.

No relation can be made between the age of the patient and the presence of reduplication. In his recent article Ohastzow³ claims that the tendency to reduplication increases as age advances. This observer divided his cases of reduplication into decades, and was struck with the fact that the larger numbers were found in the decades over 40. This was true, but he does not give the percentage of reduplications in men of approximately the same age. That is, if he has 100 patients between 40 and 50 years of age and 50 patients between 10 and 20, it is not surprising if more reduplications are recorded in the later decade.

As opposed to this contention, however, is the fact that my series was grouped in 5-year periods, with both positive and negative cases. There was no marked difference, the percentage of reduplications being about the same in each period, with the exception of the years 15 to 20 and 20 to 25, which showed an excess of positive cases over negative.

TEA, COFFEE, ALCOHOL AND TOBACCO.

The question of the influence of the excessive use of tea, coffee, alcohol and tobacco on the appearance of reduplication is difficult to determine with a sufficiently great number of cases. In the first place, patients' stories as to their habits are frequently untrustworthy. This at once reduces the list to those whose account may be relied on. Furthermore, it has been already noted, that in those persons possessing a thin chest wall a reduplication can practically always be heard. There must be no organic disease of the heart, and no interference with the heart's action, by the traction of a contracted lung, pressure of exudates, etc. Lastly, the patient must be addicted to one habit only. It is considered preferable to advance a few cases of pure dosage rather than a large number in which there is a greater uncertainty.

In the limited number of recorded cases of excessive indulgence in tea and coffee, there is no clear evidence that such abuse has resulted in causing reduplication when reduplication would otherwise be absent. One case only is positive:

CASE 5.—G., height 5 feet, 9 inches, weight 175. Present illness, strained knee.

Habits: Six cups of tea a day; no coffee, alcohol, or tobacco. The first sound was reduplicated when the heart was accelerated as above described, by holding and letting out the breath.

As against this case are four negative cases:

CASE 6.—N. Mc., used Oi to Oii of tea daily.

CASE 7.—A. F. used Oi to Oii of coffee daily.

CASE 8.—T. C. used Oi to Oii of tea daily.

CASE 9.—J. T. used Oii of tea daily.

It is at once evident that the four negative cases ingested much larger quantities of tea and coffee than did the one positive case. In view of the prevalent opinion that tea and coffee have an influence on the heart's action, this limited number (5) of cases is insufficient to draw definite conclusions from.

Concerning tobacco the result is far different. It is well known that the tolerance to tobacco varies widely with different persons, and, further, that the same bulk of tobacco smoked out of doors will have less effect than a similar quantity taken in a closed room. Reduplication was due to tobacco in three cases:

CASE 10.—S. J., aged 17, well built, smoked 5 or 6 cigars daily and drank 3 cups of tea and coffee daily. Present illness, neurasthenia. Reduplications of the first and second sounds after breathing exercises.

CASE 11.—J. L., aged 16, well built, used \$1 worth of tobacco a week. He smoked and chewed. Patient used no alcohol, tea or coffee. Present illness, pulmonary. Reduplication of both first and second sounds during the appropriate respiratory phase.

CASE 12.—H. R., aged 23, height 5 feet 7 inches, weight 149, smoked 6 cigars a day. He used no alcohol, tea or coffee. Present illness, dyspepsia. Reduplication of the first sound; not distinct.

All these persons used tobacco to excess. None used tea, coffee or alcohol to any degree. All were of the type that should show no reduplication. As against these three cases is one negative case:

CASE 13.—M. O., aged 29, well developed, chewed and smoked one pound of tobacco a week. Patient used no tea, coffee, or alcohol. Present illness, dyspepsia. No reduplication.

In general, it may be shown that persons consuming four ounces or more of tobacco in the week will show the effect on the heart in a reduplication of the first sound.

Excessive indulgence in alcohol will cause reduplication beyond question.

CASE 14.—T. K., aged 37, 5 feet 8 inches tall, 138 pounds weight, stripped. For some time patient had been taking whisky before breakfast and during the day; 3 cups of tea a day; no coffee; tobacco, 10 cents a week. Present illness, contused shoulder. First sound was reduplicated at the end of expiration, beginning of inspiration.

CASE 15.—B., aged 36, well built, took 4 glasses of whisky, 3 or more of beer daily; tea, 3 cups; tobacco, 25 cents a week. Present illness, gonorrheal knee. There was well marked reduplication of the first sound at all stages of respiration and reduplication of the second sound at the end of inspiration, beginning of expiration.

CASE 16.—J. H., 25 years, height 5 feet 5 inches, weight 148, took whisky and 10 or 12 beers daily, and no tea, coffee or tobacco. First sound was clearly reduplicated; second sound, not reduplicated. Present illness, stomach trouble.

All these cases showed very slight use of tea, coffee or tobacco. All were well-built men. It must be noted that two cases of acute alcoholism showed no reduplication. It is to be regretted that two cases only were recorded, but because of the rattling of mucus in the throats of the patients an adequate examination of the heart could be made in only two cases. Further, because of the unconscious condition of these patients, they could not be put through the routine sometimes necessary to bring out a latent reduplication. Although only 3 cases are recorded of a pure dosage with alcohol and nothing else, 12 cases in all are reported which show a definite and trustworthy account of excessive drinking. Only 4 of these showed an absence of reduplication, 2 of the 4 being the acute alcoholics above mentioned.

ARTERIOSCLEROSIS.

The relation of reduplication to arteriosclerosis is of interest, especially as Ohastzow³ has stated that beginning cardiac degeneration is a cause. Out of 25 cases in my series, in which the arteries were palpable, reduplication was absent in 10, present in 15. Of the 15 positive cases, 3 had nephritis with hypertrophy, 5 myocarditis, 1 mitral stenosis, 2 dilatation with hypertrophy, in 2 the heart was displaced, and in the remaining 2 other causes sufficed to explain the reduplication.

TYPHOID.

Concerning the incidence of reduplication in typhoid fever, there is little to be said. Forty cases were examined, but the findings were at variance with those of hearts under other conditions. In 7 cases of the 40, there was an absence of reduplication, which we should have had a right to expect if these patients were normal. The probable explanation is that the patients were lying quietly in bed. To arrive at any definite conclusion it would be necessary to accumulate a much larger series of cases. Three cases deserve especial notice.

CASE 17.—A. J., aged 23, was fairly developed.

November 20: Apex $\frac{1}{2}$ inch outside of nipple; right border, $\frac{1}{4}$ inch to right of sternum. Habits, excellent. There was reduplication of the first sound.

November 24: Apex at nipple line; right border at parasternal line. Reduplication the same.

November 28: Condition excellent. There was no reduplication.

November 29: Patient was sitting up, feeling rather weak and giddy. Reduplication of both first and second sounds.

CASE 18.—J. M., aged 17, was a tall, thin boy. The apex was at nipple line; the right border at parasternal line. Habits were excellent. There was reduplication of the first sound. Nine days later was feeling much better. Reduplication gone.

CASE 19.—H. S., aged 16, tall, slender, thin chest.

November 24: The apex was at nipple line; right border at parasternal line. There was reduplication of the first sound.

November 28: Reduplication was gone.

November 29, a. m.: Patient had a rectal examination in the morning and was very nervous. Pulmonic second sound was accentuated. There was reduplication of the second sound, but not of the first. Patient was quiet in the evening. There was no reduplication.

In no case was reduplication accompanied by any marked degree of palpitation or cardiac pain.

REDUPLICATION IN ORGANIC DISEASE OF HEART.

In none of the cases to follow were the patients put through special exercises, and, unless otherwise specified, the reduplication was perfectly distinct and persisted throughout the respiratory cycle.

Nephritis with Enlarged Heart.—There were 7 cases of nephritis with enlarged hearts. In one there was no reduplication of either the first or the second sound. In 2 cases there was reduplication of the second sound only. In both these cases the arterial tension was remarkably high. In 3 cases there was reduplication of the first sound only, and in 1 case of first and second sounds both. In the last 4 cases the arterial tension was not remarkably high.

Sansom⁴ reports 4 cases with reduplication of the first sound. Sibson⁵ reports 5 cases with reduplication of the first sound. Broadbent⁹ and Da Costa¹⁰ mention reduplication of the first sound in this condition.

Dilatation of the Heart.—Of this there were 3 cases. In two the first sound was reduplicated. In one, both first and second sounds. As these cases improved under treatment, the reduplication became more and more difficult to hear; finally it was lost altogether in two of the cases. The third case was discharged against advice, unrelieved. The close relation between the presence of reduplication and the efficiency of the heart was very prominent. Two of these cases have already been mentioned under the heading of "transitory reduplications." Sansom⁴ reports 2 cases of "dyspnea with dilatation of the right heart" with reduplication of the first sound. Da Costa¹⁰ mentions that in dilatation of the heart the second sound may be reduplicated.

Dislocation of the Heart.—Of this there were 2 cases. In one the heart was pulled over by a contracted lung. In the other the apex was pushed up into the fourth intercostal space by the accumulation of ascitic fluid. Both these cases showed reduplication of the first sound. The case of contracted lung also showed reduplication of the second sound, due probably to increased resistance in the pulmonary circulation.

Myocarditis.—There were 8 cases of myocarditis. They showed a marked diversity. In 2 cases there was no reduplication; in 2 cases, one of which was associated with nephritis, there was reduplication of the first sound only; in 2 cases there was reduplication of the second sound only, one of which showed a very slight reduplication during the appropriate respiratory phase; 2 cases showed reduplication of the first and second sound, one of which, already mentioned, was an extreme instance of gallop rhythm. The only explanation of these diverse findings is in the scattered pathology of this disease. The only conclusion we can draw is that in myocarditis the heart sounds are commonly reduplicated, and in those hearts which are doing least well the reduplication is more marked. Bramwell⁷

8. Sibson, *Lancet*, 1874, pp. 438 and 506.

9. Broadbent: "Diseases of the Heart," 1897.

10. Da Costa: "Medical Diagnosis," 1895, p. 481.

mentions reduplication following "disease or degeneration of cardiac muscle."

Mitral Regurgitation.—Of this there were 10 cases. In 8 of the 10 no reduplication was present. In 1 of the 2 positive cases there was reduplication of the first sound. This was in a case of pneumococcus bronchitis, with an old mitral lesion. As soon as the bronchitis cleared up, the reduplication cleared up, leaving the mitral regurgitation. Only one case, therefore, remains which showed a reduplication; in this case, of the second sound. Sansom⁴ reports 2 cases with reduplication of the first sound. Ohastow³ states that in mitral regurgitation reduplication of the second sound is never met with.

Aortic and Mitral Regurgitation.—There were 2 cases; in one there was no reduplication; in the other there was reduplication of the first sound. In the latter case there was doubt as to the absence of myocardial degeneration.

Mitral Stenosis.—There were 3 cases, of which 2 were associated with regurgitation. In no case was there reduplication of the first sound. In two cases there was reduplication of the second sound. In the third case (associated with regurgitation) there was no reduplication. Broadbent,⁹ Bramwell,⁷ Cabot,¹¹ Allbut¹² and La Fevre¹³ agree that mitral stenosis may cause reduplication of the second sound. Hayden reports 63 cases, with 26 instances of reduplication of the second sound. Sansom⁴ reports 37 cases, 11 with reduplication of the second sound.

SUMMARY AND CONCLUSIONS.

Reduplication of the heart sounds is a not uncommon sign.

The cause may be either asynchronous contraction of the ventricles or the auricular sound.

Normal persons with thin chest walls usually show reduplication. Persons with thick chest walls should not.

In persons in whom no reduplication should be present it is a sign of positive value.

Reduplication of the first sound means that the heart is not working properly. This may be due to nervous interference, as in persons with bad habits, or it may mean that the heart is hampered by external agencies, by pressure, or by traction, or, finally, it may mean that the heart muscle is not efficient, either due to systemic disease or inherent conditions.

Reduplication of the second sound alone means an is usually only a more advanced degree of the same condition.

Reduplication of the second sound alone means an alteration in the relative blood pressure of the systemic and pulmonary circulations.¹⁴

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11. Cabot: "Physical Diagnosis," p. 169.

12. Allbut: "System of Medicine," 1897, vol. v, p. 1018.

13. La Fevre: "Physical Diagnosis," p. 314.

14. For further literature reader may consult von Turgensen: "Erkrankungen des Kreislauforgane." Johnston: Lancet, 1876. "Guy's Hospital Reports," vol. I and VI. Gibbs: Lancet, vol. I, 1901, p. 1601. Butler: "Diagnostics of Internal Medicine," p. 885.

THE CULTIVATION OF SPIRILLUM OBERMEIERI.*

PRELIMINARY NOTE.

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We have heretofore applied the term *Spirillum obermeieri* to the organism isolated by Dr. Charles Norris, from a case of relapsing fever which occurred in New York, in the belief that it was identical with that described by Obermeier in 1873. We will continue to use this term for the present, although, as will be shown, there is reason to believe that the eastern relapsing fever is not identical with the American disease. When further evidence is obtained regarding the non-identity of the organisms found in these regions it may be necessary to restrict the use of the term *Spirillum obermeieri* to the organism found in the eastern disease.

On the other hand, the African relapsing fever, known also as tick fever, has been shown conclusively to be due to a different organism, the *Spirillum duttoni*. As far as we know, the first observation on this organism was made by Dr. D. Nabarro, of the Sleeping Sickness Commission, who noted the presence of spirilla in the blood of a patient in Uganda as early as August, 1903, but this fact was not published until 1905. In November, 1904, the presence of spirilla in tick fever was announced by Ross and Milne and also by Dutton and Todd. This organism was brought to England by Todd, and to Germany by Koch, and has since been studied by several workers.

In our preliminary note¹ we pointed out the probability that tick and relapsing fevers were two distinct diseases, due to different species of spirilla, and in the main paper² it was definitely shown that these two organisms were distinct species, and at that time we named the spirochete of tick fever *Spirillum duttoni*. Our conclusion regarding the specific difference of the two organisms was based on a comparison of (1) the animal reactions of the New York spirillum, as ascertained by Norris and his co-workers and by ourselves, with those described by Dutton and Todd and later by Breinl and Kinghorn; (2) the morphologic characteristics presented by our organism with those of the tick fever spirillum in specimens sent by Dr. Todd, and (3) the arrangement of flagella as demonstrated in this laboratory for the New York spirillum and by Zettnow for that of tick fever. Our belief that a full confirmation of this view would be afforded when cross experiments were made with sera of animals immunized to these two spirilla has been realized by the subsequent work of Breinl³ and by still more recent tests made with our serum by Dr. Schilling at the Institute for Infectious Diseases in Berlin.

It will be seen from the above facts that a recent note made by Breinl and Kinghorn⁴ and implying that our conclusion was reached merely from "a study of the two slides sent from these laboratories and of the few experiments given by Dutton and Todd" is not a fair state-

* From the Hygienic Laboratory, University of Michigan. This investigation has been aided by a grant from the Rockefeller Institute for Medical Research.

1. THE JOURNAL, Jan. 13, 1906.

2. Studies on *Spirillum Obermeieri* and Related Organisms, Jour. of Infect. Dis., May, 1906, vol. III, pp. 291-393.

3. The Specific Nature of the Spirochete of the African Tick Fever, The Lancet, June, 16, 1906.

4. A. Breinl and A. Kinghorn: An Experimental Study of the Parasite of the African Tick Fever (*Spirocheta duttoni*), Memoir XXI, The Liverpool School of Tropical Medicine, September, 1906.

Metatarsalgia.—W. E. Blodgett, in the *Physician and Surgeon*, states that the pain in the chronic type of metatarsalgia, as in the ordinary pronated foot, is due to general abnormal tension and pressure. In the acute type, however, the pain is due to such a displacement of the heads of the metatarsal bones that one of the branches of the external plantar nerve is squeezed between two of the metatarsal heads.