

DR. C. R. McCLURE, Portland, Ore.: At present through the courtesy of Dr. Coffey's office in Portland we are having observations made on this region of the lower back, and I hope at some future meeting to make some reports concerning our study of this region. We see a great many of these cases, and, like many of the men here, I sometimes doubt the value of the Roentgen ray in a diagnostic way because of the frequency with which these anomalies are found in people who have no symptoms one way or the other.

DR. ROLAND MEISENBACH, Buffalo, N. Y.: That the pelvic joints, especially the sacro-iliac and sacrolumbar, are so frequently involved in the sciaticas and the backaches which are met with, can no longer be doubted. The results of the treatment for these conditions, with the relief given the patient, have spoken for themselves. I believe that the most important matter for our consideration in diagnosis is to differentiate between the sacro-iliac and the sacrolumbar lesions. It is true that in some we have both these joints involved; it must also be remembered that occasionally we may have backache due to the strain or injury to the sacrolumbar joint in a person who may have other conditions present; as, for instance, a very heavy person with leukemia may sustain an injury to the sacrolumbar joint due to the actual weight of the abdomen, and in relieving the strain we are naturally relieving but one of the symptoms. Another type of sacro-iliac and sacrolumbar lesion which is more frequently met with is one of trauma, in which there also exists pelvic inflammation, or glandular inflammation, which has attacked the pelvic joints or has weakened them so that they are subject to trauma. One has only to remember his anatomy and note the variations in the sacrolumbar joint, both as regards the articular processes and also the variations in the transverse process in different individuals. I have met many times a type of sacrolumbar lesion in which the patient has had recurrent backache, which I am sure has been due to a very shallow sacrolumbar articulation. In this type it has been sometimes difficult to illustrate the condition by means of a roentgenogram. In regard to the roentgenogram, I cannot agree with Dr. Langnecker. If he had used the word "occasionally" instead of "usually," I think it would have been more to the point. In cases with a long or thickened transverse process of the fifth lumbar vertebra, which may or may not articulate with the sacrum or ilium, or in cases in which the sacro-iliac joint has opened and the sacrum has dropped or tilted, it is not very difficult to illustrate by means of the roentgenogram; but those cases (and they are frequently met with) in which the sudden movement of the body may quickly incapacitate the individual, causing such a severe pain that the person is unable to take another step, are rather difficult to illustrate by means of the roentgenogram. I believe this type of case to be that of the shallow sacrolumbar joint, in which the joint articulations are of such construction that they easily become dislodged. Many of these do not seek relief at the first attack, but usually after having rested or having had several attacks. Also in the type of loose sacrum it is difficult to demonstrate by means of the roentgenogram; that is, the single flat plate will show nothing, but the stereoscopic roentgenogram will often show the sacrum slightly tilted. Dr. Langnecker has covered the ground of symptoms very well. One of the best illustrations of the severe sciatica which was caused by motion in the sacro-iliac joint is that of metastatic carcinoma of the sacrum following, as in the case I have presented, a removal of the breast for carcinoma. In this case, the slightest motion of the ilium on the side in which the sacro-iliac joint is involved causes a severe sciatica.

DR. C. L. LOWMAN, Los Angeles: We have a skeleton which shows an anomaly of the first sacral—just a small lateral process on one side, like a lumbar vertebra, the full length and width of ala on the opposite side, with a joint between it and the second sacral; an absolutely flat articular process on the right side, and a vertical process on the other. This allowed a great deal of rotation of the sacrolumbar segment forward around this vertical proc-

ess as an axis, with a narrowing of the posterior foramina. Another similar case was presented to me by my radiographer. This case was treated by a chiropractor as a tuberculous spine. The roentgenogram showed an assimilation of the first sacral, which tried to be a lumbar on one side.

DR. H. L. LANGNECKER, San Francisco: In reply to Dr. Ridlon's question, I would say that in a great number of cases, especially those of the lumbosacral region, there seems to be an atrophy of the muscles of the anterior part of the thigh, the atrophy of the other muscles having been consistently noted.

DR. JOHN RIDLON, Chicago: By measurement?

DR. H. L. LANGNECKER: Yes, by measurement. It is one of the signs. These conditions are usually studied by stereoscopic roentgenograms; all the important ones were in my cases. I found that the study of this class of cases was extremely interesting. There is much, as yet, to be said about them. I can only repeat what one of the orthopedic surgeons—a man who has done more, probably, on lesions in the sacro-iliac region than anybody else in the country—told me. He said that there was a great deal yet to be known about lesions in this part of the body, and that he thought when the final verdict was given it would be an anatomic one.

LYMPHOID MARROW AND TUBERCULOSIS

AN EXPERIMENTAL STUDY *

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In a paper read before this section at its meeting in 1914, Mr. John Fraser made the statement that red marrow was immune or practically immune to tuberculosis. This statement was based on the results of attempted inoculation of the long bones of laboratory animals, whose marrow is of the red or lymphoid variety. The question is of great importance, and we have been conducting a series of experiments in the laboratories of the Stanford School of Medicine to test the accuracy of Mr. Fraser's conclusions.

Dr. John F. Cowan and I conducted one series, on which we shall report later. Dr. Jean Oliver and I conducted another series which I shall report here.¹

The animals used were rabbits and guinea-pigs. With the former, bovine tubercle bacilli were employed (Cutter Laboratory bovine strain, K 9), with the latter, human bacilli from a virulent strain supplied by Dr. Hirschfelder. The animals were anesthetized with ether, and the limb to be operated was shaved. Strict asepsis was observed, a small incision was made in the leg, a trephine opening was made into the tibia, and with a hypodermic needle a few drops of a suspension of bacilli in normal salt solution were injected directly into the marrow. The wound was then sewed up with catgut and was painted with collodion.

At varying intervals before the operation and sometimes after it, a 1 per cent. solution of trypan blue was injected into the animals, 20 c.c. into the ear vein of the rabbits, and 2 c.c. subcutaneously into the

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¹ Read before the Section on Orthopedic Surgery at the Sixty-Sixth Annual Session of the American Medical Association, San Francisco, June, 1915.

² Owing to lack of space, this article is abbreviated in THE JOURNAL by omission of protocols of animal experiments. The article appears in full in the Transactions of the section and in the author's reprints. A copy of the latter will be sent by the author on receipt of a stamped addressed envelope.

1. After the main question of the vulnerability of the marrow had been determined, the work was divided. Dr. Oliver continued his investigation on the histologic features of the tubercle.

guinea-pigs. The animals were killed at varying intervals after the operation.

Our intention was usually to inject into the metaphysis of one tibia and into the central canal of the other, but in point of fact the canal extends so high in the tibia that practically all injections were into it.

The specimens were hardened in 10 per cent. formaldehyd solution or in alcohol, dehydrated in alcohol, mounted in paraffin, and stained with hematoxylin and eosin, with the Van Gieson stain, and for tubercle bacilli with the Ziehl stain.

A complete description of the findings is appended. The results will be summed up here.

EXPERIMENTS ON RABBITS

Rabbit 41 0: Killed fifty-one days after injection. Right tibia, tuberculosis present macroscopically and microscopically. No tubercle bacilli found. Left tibia kept for gross demonstration. Tuberculosis demonstrated macroscopically in it.

Rabbit 42 0: Killed forty days after injection. Both right and left tibias showed tuberculosis and tubercle bacilli present.

Rabbit 43 0: Killed fifty-four days after injection; blocks lost.

Rabbit 44: Killed on day of operation. Right and left tibias showed neither tuberculosis nor tubercle bacilli.

Rabbit 45: Killed thirty-three days after operation. Both right and left tibias showed tuberculosis macroscopically and microscopically, but no tubercle bacilli in either.

Rabbit 46: Killed thirty-two days after operation. Both right and left tibias showed tuberculosis macroscopically and microscopically; tubercle bacilli in neither.

Rabbit 47: Killed thirty days after operation. Both right and left tibias showed tuberculosis macroscopically and microscopically; tubercle bacilli were found in the right, none in the left.

Rabbit 49: Killed one day after operation. Right and left tibias showed neither tuberculosis nor tubercle bacilli.

Rabbit 75: Killed two days after operation. Right and left tibias showed no sign of tuberculosis or tubercle bacilli.

Rabbit 76: Killed seven days after operation. Both tibias showed neither tuberculosis nor tubercle bacilli.

Rabbit 77: Died fifteen days after operation. Both tibias showed neither tuberculosis nor tubercle bacilli.

Rabbit 78: Killed twelve days after operation. Right tibia showed tuberculosis microscopically and a few tubercle bacilli.

Omitting Rabbit 43 0 whose marrow specimens were lost, we have here 11 rabbits. In 6 tuberculosis was demonstrated, in 5 it was not. The 5 in which it was absent were killed respectively the day of operation, and one, two, seven, and fifteen days after operation. All animals killed after a greater interval than fifteen days, and one after an interval of twelve days showed distinct marrow tuberculosis. In other words, in every animal allowed to live over one week, save one, tuberculosis was demonstrated.

Tubercle bacilli were recovered from both tibias of Rabbit 42 0 (forty days), from the right tibia of Rabbit 47 (thirty days), and from the only operated tibia in Rabbit 78 (twelve days).

Others have noted the difficulty of recovering bovine tubercle bacilli from the marrow. In two tibias of our series they could be found only by diligent search. Typical giant cells are rare. Friedrich² made this observation.

The "fibromyxomatous" marrow described by Fraser is a practically constant phenomenon in the neighborhood of the tuberculous marrow, but is not

peculiar to tuberculosis. It is probably present in most slow marrow inflammations, and we regard it as Nature's protective reaction against the spread of infection, not as a mysterious change which the marrow undergoes before it can be invaded, and when tuberculosis is in the neighborhood.

Another very suggestive fact that we (Dr. Cowan and I) have observed in another series of experiments (not tuberculosis experiments) on rabbits' cartilages, is the presence of a thin strip of fatty marrow which frequently is present near the circumference of the end of the femur (that is, close to the attachment of the capsule). We believe this band of yellow marrow is responsible for Fraser's contention that the lymphoid marrow of the bone end must change to fatty marrow before it can be invaded by tuberculosis from the synovial membrane.

Conclusion: The lymphoid marrow of rabbits' bones is a good field for the growth of bovine tubercle bacilli.

EXPERIMENTS ON GUINEA-PIGS

Six guinea-pigs were employed, and were killed or died, respectively, four, six, six, nine, ten and fifteen days after operation. All showed tuberculosis of the marrow, and in all tubercle bacilli were demonstrated.

Conclusion: The lymphoid marrow of guinea-pigs' bones is a good field for the growth of human tubercle bacilli.

It is now five years since the theory was first published that the location of tuberculosis in bone was determined by the presence of red or lymphoid marrow. I should like at this time to sum up the evidence to date:

1. Tuberculosis is found only in those bones or regions of bone where lymphoid marrow is present.

2. Marrow tuberculosis is more common in children, whose bones contain more red marrow. As age advances, marrow tuberculosis becomes rarer and less destructive.

3. After resections in adults, if secondary infection be avoided, the disease disappears. The best authority available says that after resections the lymphoid marrow disappears.

4. Experiments show that lymphoid marrow is an excellent field for the growth of tuberculosis.

Two links are missing in the chain of evidence; that is, the demonstration of the particular cells in the lymphoid marrow which are responsible for the disease there, and the proof that tubercle bacilli will not grow in yellow or fatty marrow. It will be instructive also to learn the exact morphologic changes which take place in the marrow after a resection.

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ABSTRACT OF DISCUSSION

DR. JOHN F. COWAN, San Francisco: What Dr. Ely has said on the question of marrow tuberculosis is exceedingly interesting, in view of the paper which was presented to this section at its meeting in Atlantic City last year by Mr. John Fraser of Edinburgh. It is said that it is very difficult, and in many cases, impossible to infect healthy marrow with tubercle bacilli; that in order for the marrow to become a fertile field for the growth and development of tubercle bacilli, it is necessary for it to undergo certain changes, which he describes as a colloid degeneration, or a fibromyxomatous change; that these changes in the marrow are due to an obliterating endarteritis, resulting from a tuberculous toxemia arising from a tuberculous process in some other portion of the body. Now we do not find

2. Friedrich: *Deutsch. Ztschr. f. Chir.*, 1899, liii, 532.

in uncomplicated cases of tuberculosis much toxemia. Tuberculous toxins cause lesions locally; that is, in their immediate presence; not at a distance by diffusion of the toxins. At least, the kidneys never show any effect which could be ascribed to the elimination of a tuberculous toxin through them. In these experimental animals we do not find evidence of tuberculosis in other parts of the body. Postmortem examination shows tuberculosis of the marrow, but no tuberculous lesions in the lungs, lymph nodes, etc., so that it is difficult to ascribe the local vascular changes to a general toxemia. Arterial degeneration is not found as a frequent condition in tuberculosis. In fact, in advanced cases of tuberculosis, instead of finding arteriosclerosis, there is an atrophy of the vessels, as there is an atrophy of the other tissues of the body. However, there are changes in the vessels in the immediate vicinity of the tuberculous process, but these are not due to a general toxemia, but rather to the local chronic inflammation. In tuberculosis we do not find necrosis as an early factor. One may see endothelial leukocytes filled with tubercle bacilli and yet one may not find any degenerative changes in these cells. I have had the privilege of examining Dr. Ely's specimens. In some of them characteristic tubercles are seen, but with slight or no evidence of changes in the blood vessels; and this absence of an obliterating endarteritis is probably due to the fact that these animals were killed at short periods after inoculation. It seems highly probable that the bone marrow of the experimental animals may be primarily infected by tubercle bacilli.

REVIEW OF RECENT STUDIES IN TRICHINIASIS

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Trichiniasis is not a mere medical curiosity; it is a common and important disease and one all too seldom recognized. A disease having an average mortality about half that of typhoid fever, and in some epidemics a mortality rising to 16 or even 30 per cent., as in the Hedersleben epidemic¹ in 1865, is serious. Proof that it is a common disease may be had from studies such as that of Williams,² who in 105 unselected necropsies in the state of New York found trichinella embryos in 5.4 per cent. In other civilized communities, this parasite is found in from 0.5 to 2 per cent. of necropsy subjects. About 6 per cent. of American swine, and a larger percentage of American rats are infected.

Recent years have added much to our knowledge of trichiniasis. The life history of the parasite and its methods of migration and distribution in the body of its host have been quite conclusively worked out. The symptoms have been adequately described and new methods of diagnosis brought into use. It remains to study more extensively certain symptoms of the disease, with a view to a more complete understanding of some as yet unsolved problems.

One of these symptoms is fever. It is interesting that the *Trichinella spiralis* is the only metazoic parasite that, infesting man, causes fever with constancy. Filariasis, hydatid disease, bilharziasis may give rise to fever, but exceptionally. In trichiniasis alone, of the parasitic disease of this class, is fever almost always present. Such fever may be due to secondary bacterial infection, to the toxic products elaborated by the trichinella, or may be a true protein fever from

the parenteral introduction of alien material, either in the form of the embryo of the trichinella itself, or a devitalized body protein formed as a result of the activities of the parasite.

Evidence in favor of the first hypothesis is contradictory. The female trichinella within a week of infection burrows beneath the submucosa of the duodenum, often destroying two or three villi, and deposits embryos in the lymph spaces of the submucosa, thus giving opportunity for micro-organisms to gain entrance to the lymph channels and blood stream (Frothingham³). In this way a bacteremia may arise. Bacteremia in trichiniasis in man is rare. Staübli⁴ mentions a severe case in which there was very high fever, and in which streptococci in pure culture were found in the blood and urine. In the second case, deep intramuscular abscesses containing numberless staphylococci were found. Friedreich⁵ mentions the frequency of furunculosis as a complication of trichiniasis. In one of his cases the embryo was found in the midst of one of the furuncles along with staphylococci.

That meat containing bacteria and trichinella can give rise to a double infection seems fairly well established by a report of Chiari and Zorkendorfer⁶ of an epidemic in which sixty-eight persons were made ill by eating sausage. Twenty-one of these patients showed severe symptoms with notable enlargement of the spleen. Four of the patients died and came to necropsy. The muscles of all these showed trichinella embryos, and the spleen and blood, anthrax bacilli. The *mettwurst* causing this epidemic was made from pork and a variety of other meat some of which was, in this instance, thought to be from an animal infected with anthrax. Staübli⁴ examined the peritoneal exudate and blood from the hearts of two guinea-pigs dying from trichiniasis, and in one found a diplococcus, in the second, streptococcus in both regions. Romanovitch⁷ could find no bacteria in infected tissue on section. However, on examination of the blood of thirty-six trichinous rats, twenty-six showed bacteremia. Of fourteen such guinea-pigs eleven showed bacteremia. The bacteria were of various kinds, streptococci, staphylococci, etc. Gruber⁸ made cultures from the heart blood of fifteen guinea-pigs infected with the trichinella; only three were positive. These were in such small numbers that Gruber thought them probably contamination.

In the search for clinical data on the question of bacteremia in trichiniasis I have reviewed the cases in the Roosevelt and St. Luke's Hospitals of New York observed during the past five years, and have found that of ten cases with blood culture in none were bacteria demonstrated.

The weight of evidence is against the bacterial origin of the fever of trichiniasis. Additional argument is the conclusion of McNeal and Chace,⁹ who found that in the normal duodenum bacteria are few.

3. Frothingham, C. Jr.: A Contribution to the Knowledge of the Lesions Caused by *Trichinella Spiralis* in Man, Jour. Med. Research, 1906, x, 483; The Intestinal Lesions Caused by *Trichinella Spiralis* in Rats, Arch. Int. Med., January, 1909, p. 505.

4. Staübli: Trichinosis, Wiesbaden, 1909, p. 141.

5. Freidreich: Beobachtungen über Trichinosis, Deutsch. Arch. f. klin. Med., ix, 459.

6. Chiari and Zorkendorfer: Ueber die Aetiologie einer Massenerkrankung in Teplitz Schönau nach dem Genuss von Fleisch und Wurstwaren (Trichinose und Milzbrand), Ztschr. f. Heilk., 1894, xv, 435.

7. Romanovitch: Recherches sur la trichinose, Ann. de l'Inst. Pasteur, 1912, xxvi, 351.

8. Gruber: Neue Studien über die Pathologie der Trichinose, München. med. Wchnschr., 1914, lxi, 645.

9. McNeal, Ward J., and Chace, Arthur F.: A Contribution to the Bacteriology of the Duodenum, Arch. Int. Med., August, 1913, p. 178.

1. Cited by Staübli: Trichinosis, Wiesbaden, 1909, p. 16.
2. Williams: The Frequency of Trichiniasis in the United States, Jour. Med. Research, 1901, vi, 64.