will say, that the town is interested in the disposal of garbage, or sewage. Now should our doctor be informed on the crematory method, we will say of New York City, for the burning of garbage; or the septic tank method for handling the sewage of the city of Glasgow—then surely his giving notice that a practicing physician amongst them understood these things would not be amiss. Nor would it be improper should a public meeting be called at the opera house for him, if invited, to speak on such subjects.

Alas! it sometimes happens that when a physician really commences to follow the principles of medical ethics as outlined in Chapter 3, Sections 1, 2, 3, 4, and 5 regarding his duties to the public, that he will be accused of trying to advertise himself.

Indeed, I sadly fear that if a physician really lived up to and carried into daily practice the principles of medical ethics as adopted, recommended and promulgated by the A. M. A. in its three Chapters, six Articles and fifty-three Sections, it might raise from some quarters such a storm of protest and indignation as to endanger his very license.

## THE TOXEMIC FACTOR IN RHEUMATOID ARTHRITIS.*

By CARL C. WARDEN, M. D., Los Angeles.

Briefly summarized, the factors etiologically associated with the variety of arthritic conditions grouped under the name of rheumatoid arthritis, are the nervous, the bacterial and the toxic. The classification of the arthritic troubles themselves is not so easy or so brief. One of the most satisfactory is that of Goldthwaite, of Boston, who mentions:

1. Chronic villous arthritis (dry joint).
2. Atrophic arthritis (rheumatoid).
3. Hypertrophic arthritis (osteo).
4. Infective arthritis.
5. Metabolic arthritis (gout).

Of these Garrod disregards the first and last and adds *spondylitis deformans*.

The term rheumatoid should be disregarded altogether since it is at best a vague and misleading term, and a classification made based on a more comprehensive clinical description. I prefer to adopt that system which disregards gout altogether and groups all the systemic joint troubles under the heads of *hypertrophic* and *atrophic* arthritis and Still's disease of children, which alone seems to fall under neither division.

The hypertrophic varieties are observed in two clinical forms:

(a) The monoarticular type of old age (osteo-arthritic hip).

(b) The polyarticular type, seen past midlife, oftenest in women, with Herberden's nodes common.

The atrophic varieties are subdivided into:

(a) Acute polyarticular type affecting children and young adults.

(b) Chronic polyarticular type of midlife.

If now we relegate Still's disease, which really does not belong to this class at all since its pathology is wholly different, we can exclude it from our discussion, along with gout and the infectious forms, such as gonorrheal and septic (though we are obliged to admit that the infectious forms are occasionally precursors of the atrophic type). We now have to consider merely the hypertrophic and atrophic forms, and when we regard the pathology of these states and the numerous cases which merge one into the other, it becomes a temptation to call all these states various modifications of one pathological condition, a systemic arthritis.

Postmortem examinations, histological and macroscopics show that at various stages of the morbid process the same changes occur in the tissues in both hypertrophic and atrophic forms. The alterations in the soft parts around the joints are practically the same in each form. The alterations in joint cartilage and the articular surfaces of the bones shade from one into the other in both varieties. In one there may be preponderance of atrophy, in another hypertrophy, but in the latter case the overgrowth almost always follows on primary atrophy. The distinction marked by *wet* and *dry* joints does not always hold good, since not infrequently effusions will form in joints in the one case hypertrophic, in the other atrophic. By our term rheumatoid arthritis then I mean to include these two forms of chronic arthritis trouble and I venture to suggest the term chronic toxemic arthritis as one more appropriate than rheumatoid.

The nervous theory of causation advanced by Remak and defended by Senator, Ord, Spender, Ross, MacMahon and others,—and advanced chiefly because of the resemblance of toxemic joints to the trophic joints of Charcot,—flies widest of the mark inasmuch as it does not explain either the majority of the lesions or exclude other possible causes. The bacterial theory of causation has many advocates and comes nearer filling the requirements. Braumler of Freiburg observed the frequency of toxemic joints following the acute joint infections. Schuller in 1893 isolated a bacillus from the joint of effusions. Bannantyne, Wohlmann and Blaxall also found a bacillus. Painter was unable to find it. Poynton and Payne observed a minute diplococcus in several cases. Many investigators have seen this latter organism. I have myself observed it in two cases, but all agree that the organism is not to be found in every case, even when joint effusions exist.

A toxemic theory is most satisfactory for many reasons. In the first place, it does not exclude a primary microbic infection, in fact, it may assume it, the lasting and crippling lesions appearing only when the infection, however slight, has long since subsided; and in the second place, it does not necessarily postulate an infective cause inasmuch as evidences of bacterial damage may be wholly lacking and yet a toxemic cause remain. Llewellyn Jones sees many factors in the disease comparable to the processes at work in Raynaud's disease, tetany,

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migraine, etc. He describes pathological changes in the liver, spleen, kidneys, pancreas, ovaries and arteries, but makes no mention of the digestive apparatus beyond a macroscopic intestinal thickening. Clark of Edinburgh, on the other hand, makes the intestinal canal largely responsible for the production of toxins. Chalmers Watson agrees. Luff assigns the difficulty to an infection from some mucous membrane. Cave mentions many sources of toxin, rectal ulcers among them, but lays greatest stress on pyorrhea alveolaris. Tubby and Payne support this view, the former stating that almost invariably these cases had carious teeth, the latter that joint trouble was frequently observed by him in patients having pyorrhea alveolaris. Tubby cites largely from a valuable report of Andrews and Hoke on the "Relation of Albuminous Putrefaction of the Intestines to Arthritis Deformans and Its Influence on Treatment." In this paper the authors quote Herter's classification of three types of intestinal putrefaction

(a) The Indolic type, occurring in the entire intestinal canal, in which quantities of indol are made and in which the bacillus coli appears.

(b) The Saccharo-Butyric type, occurring in the lower ileum and large bowel, caused by anaerobic organisms for the most part, in which the stools may be acid, and indol is not excessive.

(c) The combined type.

Herter is also credited with the statement that the intestinal flora both as to numbers and character is influenced especially by the quantity of albuminous food taken, the anerobes diminishing with decrease of nitrogenous food and the concomitant decrease in nitrogenous putrefaction. Combe of Lausanne in his recent book on the Diseases of Children lays stress on the train of evils resulting from proteid putrefaction in the intestinal canal. He indexes the degree of putrefaction by quantitative estimations of urinary indican and the higher ethereal sulphates and by the alkalimetric observations of the feces. The theories of Metschnikoff on longevity have gone so far as to reach lay readers and his work on intestinal bacterial putrefaction has stimulated many observations on the question. Metschnikoff contends that albuminous putrefaction is frequently due to the pathological abundance in the intestinal canal of anaerobic, alkali producing organisms, which condition alters the normal acidity of bowel contents, banishes acrobic flora from the intestine and causes an abnormal splitting of nitrogenous molecules into toxic radicals which, once absorbed, lead to systemic toxemia. It is this toxemia which we seize upon as an etiologic factor in rheumatoid arthritis. Metschnikoff and his followers commended as a therapeutic measure the reduction of nitrogenous food in the dietary together with the correction of alkalinity and anaerobic invasion by administering lactic acid producing organisms which in suitable media will generate nascent lactic acid in the intestinal canal and assist in rendering the digestive tract uninhabitable by these noxious bacteria. With this end in view he gives milk soured by various organisms, chiefly the bacillus or streptococcus lacticus and the bacillus of Massol. In the laboratory the sterile milk is prepared by inoculation with cultures, in the homes by the liquid milk starters and compressed tablets now supplied by the commercial pharmaceutical houses.

Tubby reports several cases of toxemic arthritis treated by this method with very encouraging results. We, personally, have under observation at present three cases of toxemic polyarthritis. Two are of the hypertrophic type, in women under thirty, one of the atrophic form in a man of thirty, the bony and periarticular changes showing well in radiograms. These patients at the outset of treatment had indican in the urine in great excess. In all there was a history of digestive disturbances of long standing with constipation and occasional attacks of diarrhea. The feces of each was markedly alkaline in reaction and contained many anaerobic bacteria, the bacillus enteritides sporogenes important among them. The treatment has consisted of daily bowel evacuations by agents best suited to the individual case, the administration of intestinal antiseptics like salol and guaiacol carbonate, and artificially soured milk in quantities of at least one pint daily. All meat in the diet has been interdicted. The custom has been to prepare the milk in our laboratory by inoculating sterilized milk, preferably skimmed, with pure cultures of the streptococcus lacticus and the Bulgarian bacillus of Massol and incubating over night at 37°. The product is smooth with well marked odor and taste of lactic acid. From this supply starters of one ounce quantity are given the nurses with instructions for the preparation of the milk at the patients' homes. A portion of the supply made in this manner may be used as starter for the next day's ration and this process carried on for a week when usually contamination will have begun and fresh pure cultures are required. The preparations sold by the pharmaceutical houses answer fairly well, but they almost invariably contain yeasts which while harmless are apt to make the soured milk unpalatable.

Under this simple treatment the patients have improved, two of them remarkably, the third to some extent only owing to the brief time he has been under care. The chief improvement is shown by the lowering of temperature, the reduction in periarticular swellings and effusions, the greatly increased range of motion, the diminution of pain and a general amelioration of subjective feeling. One case is complicated by a chronic parenchymatous nephritis, but the kidneys have improved *pari passu* with the arthritic troubles as shown by considerable reductions in the number of casts and the quantity of albumen. There has been a steady reduction of the quantity of indican in the urine. In one case it has entirely disappeared. The history of illness in one woman dates back nine years, in the other, two. The improvement in the former case, despite the nephritic handicap is most encouraging. The second woman has in six months' time come from absolute helplessness in bed to her wheel-chair, her fancy work and piano exercises, with entire sub-

sidence of inflammatory trouble about the joints. I believe the most valuable adjunct to the treatment of these cases to be the passive hyperemia of Bier. It has appeared to exert a marked influence on the pain in the joints and to have assisted in the absorption of joint exudates.

## THE RELATION OF RAT LEPROSY TO HUMAN LEPROSY.*

(With an Exhibit of Gross and Microscopic Specimens.)

By WM. B. WHERRY, M. D., San Francisco.

It seemed worth while, in connection with Doctor Clark's exhibit of cases of human leprosy, to show you some specimens from a very closely related, if not identical, disease which occurs in the rat. Leprosy in rats has been known for a number of years and has been recorded in England, Southern Russia, Roumania, India, and the region about San Francisco Bay.

The disease has usually been found in connection with the examination of large numbers of rats for plague and no doubt further investigation will show that it is quite prevalent among rats all over the world. It is characterized by a granulomatous proliferation of the subcutaneous tissues, leading to a marked thickening of the skin, alopecia, and ulceration. In some cases the peripheral nerves are involved, resulting in the loss of fingers and toes or the tail. Acid-proof bacilli resembling those found in human leprosy occur in enormous numbers in the affected tissues. Upon histological examination the changes in the skin closely resemble those found in human leprosy. The disease may be transmitted by inoculation from rat to rat but not to guinea pigs, rabbits nor monkeys. Such inoculated rats develop the disease very slowly and it is only after several months have elapsed that definite signs of the disease are found.

This disease is of particular interest because it furnishes material for experiments of a comparative nature, whereby we may gain some idea as to how human leprosy is transmitted. It widens the field for research on such problems as the early diagnosis of leprosy—the production of immunity to leprosy—and the treatment of leprosy.

It is generally believed that the bacillus of rat leprosy has become specialized in the rat and differs from the human leprosy bacillus to about the same degree that bovine tubercle bacilli differ from human tubercle bacilli. The recent work of Mezincescu (*Comp. Rend. Soc. Biol.*, 1909, 66, 56), would seem to support this idea. This investigator, working in Roumania, tested the ability of rat lepra bacilli to fix complement according to the Bordet-Gengou reaction. Of the sera from twenty-four cases of human leprosy (tubercular, mixed, and anesthetic cases) twenty gave complete fixation; two slow fixation; and with two fixation was negative. He controlled a certain number of these cases by tests with extracts of the tubercle bacillus and paratubercle bacilli (Timothy-Mist) with entirely negative results. He believes that his observations point to a very close relation between rat leprosy and human leprosy. (A leper rat caught last Saturday at 21st and Broadway, Oakland, is on exhibition. Also a section of skin from a leper rat showing the enormous numbers of acid-proof bacilli present in this disease.)

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## REPORT OF A CASE OF TRANSIENT CYCLOPLEGIA DUE TO GLYCOSURIA.*

By W. HUMES ROBERTS, M. D., Pasadena.

Paralysis of accommodation, due to diabetes, is sufficiently rare to warrant the report of the following case:

Mr. W., aged 51, first consulted me March 30, 1908, concerning a tickling in his throat, which had troubled him for about two weeks. He was coughing a great deal, he felt and looked sick, and he had recently lost flesh.

Examination showed his uvula was thickened and elongated, fauces congested; right cord somewhat immovable, irregular in outline, and reddened near the arytenoid cartilage.

Fearing that a tubercular process might be commencing in his lungs, I advised him to consult a general physician for a physical examination. He placed himself under the care of Dr. Joseph D. Condit. Dr. Condit reported to me that there was no evidence of tuberculosis, but that his urine contained 7% of sugar.

Under appropriate diet, the sugar began to lessen, so that by the 7th of April it was down to 5 5-8%, and his weight, which was 133 pounds, began to increase. By the 29th of April all sugar had disappeared from his urine, and, when last observed by Dr. Condit on the 22nd of July, his weight had increased to 142 pounds.

On the 15th of April, when he had been under Dr. Condit's care for two weeks, his urine showed only ⅜% of sugar. At that time, he came to me again, saying that for a few days past he had been unable to read with his glasses, which had theretofore been perfectly satisfactory, and that he now needed them to see with in the distance. Until he noticed this failure of his glasses for reading, his vision for distance had been perfect; but now he could not see in the distance without the use of his old reading glasses.

I found that he had been using for reading a pair of + sph. 1.75

At that time O. D. V.=6/22.5
O. S. V.=6/15

Manifest examination showed
O. D. + sph. 1.75=6/5
O. S. + sph. 1.75=6/5 1½° esophoria.

For reading at thirteen inches, he required to be added to the above + sph. 2.25.

External examination of the eyes showed everything normal; pupils were of normal size, and they reacted to light and accommodation. Ophthalmoscopic examination showed the media clear and the fundi normal. A test on the following day confirmed this examination, so I ordered the foregoing lenses for him.

On the 18th of May he came in again very much improved in his general health. He said that until within a few days, these last glasses had been perfectly satisfactory; but that now he could not see so clearly in the distance with them, and he found that he had to hold newspapers and books too close to his eyes to read with comfort. His vision with his distance glasses now was only 6/12.

* Read at the Thirty-ninth Annual Meeting of the State Society, San Jose, April, 1909.