

right index and right thumb were preternaturally movable, and distinct grating could be heard and felt. In September the darting pains attacked the left thumb and an enlargement of the metacarpophalangeal joint followed. About this time Dr. Pepper saw her with me and agreed as to the nature of the case. At his suggestion, hypodermic injections of the double chlorid of gold and sodium were given twice daily. Later on I placed the patient on thyroid extract, but almost immediately alarming toxic symptoms, those of thyroidism, developed, and the remedy had to be abandoned. Throughout these months she was taking tonics, cod liver oil, arsenic, syrup of the iodid of iron, etc., and also received inunctions of cod liver oil; occasionally salicylates were given; faradic electricity was also tried. By November the legs had again become flexed and extension was reapplied and kept on for two and a half months. Bone-marrow was administered for a few weeks in 1895, but without practical result. The disease continued to advance—the right shoulder became involved, and atrophy of the shoulder and scapular muscles occurred. The circumference of the right arm at the axilla was 3 cm. less than that of the left. At no time was the patient's condition influenced in the slightest degree by change in the weather.

The use of strontium bromid was now begun and, whether *propter hoc* or *post hoc*, the patient began to improve; in April, 1895, she was able to walk around the bed by holding on to the railing; on July 1 she walked out into the yard, but the following day had a relapse and was again confined to bed, internal treatment being continued and systematic massage being given. Improvement now became continuous and progressive, and in November she ventured out and walked eight city squares the first day. Last year and this spring she again worked at her trade, and recently consulted me as to the advisability of getting a bicycle. She tells me she has almost forgotten her long illness, the vestiges of which are, however, unmistakable, though they do not trouble her any. The heart murmur has entirely disappeared; there is now only slight accentuation of the second aortic sound; the pulse is regular but a little rapid. Motion in the shoulder, knee, and ankle joints is perfect; the deformity of the right wrist has nearly disappeared, and that of the right thumb is greatly lessened. Abduction of the thumb is, however, still restricted, and there is occasional crackling in the knee joints when she rises from the sitting posture. The ankle clonus can no longer be elicited.

To recapitulate: A young, healthy woman, with a family history of rheumatoid arthritis and lithemia, was taken ill after a sea bath with peculiar pains, soon followed by joint enlargement, destruction of cartilage, atrophy of the muscles, and contraction of the joints, all fairly symmetric. A heart murmur was present; the knee jerks were exaggerated, and there was ankle clonus. After continuous confinement to bed for seventeen months, improvement set in and was followed by a practical recovery.

The treatment was so varied and manifold that definite conclusions can not be drawn from it, but I beg to call attention to the following points: 1. The importance of preventing contractures and the efficient value for this purpose of the extension apparatus. I am convinced that had it not been applied the patient, although the disease is arrested, would now be unable to stand or walk. It has also occurred to me that an analogous device for the arms in certain cases might be of great service. 2. From the apparent effects of strontium bromid in this and other cases I am led to the view, still tentative however, that it has: *a*, an influence over the pains; *b*, perhaps a power to limit the progress of the disease.

A Characteristic Symptom of Lesions in the Posterior Cranial Fossa is the peculiar dyspnea whenever seated upright, relieved by resuming the reclining position. The breathing becomes very slow, deep, snoring, and interrupted by frequent pauses half a minute long. Two new observations are described in the *Deutsche Med. Woch.*, of June 10. One was caused by a tumor in the fossa, left of the basis cerebri, the other by thrombosis arteriæ basilaris, both fatal.

THE CARDIO-VASCULAR AND RENAL RELATIONS AND MANIFESTATIONS OF GOUT.

Presented to the Section on Practice of Medicine, at the Forty-eighth Annual Meeting of the American Medical Association held at Philadelphia, Pa., June 1-4, 1897.

BY N. S. DAVIS, JR., M.D.

CHICAGO.

That gout and its varied complications are due to toxemia, possibly to uric acid or to other substances which vary in the blood as it does, is generally admitted. Interstitial nephritis, the common renal complication of gout, is so well understood that I need not dwell upon it in this discussion. There are a few points, however, in the course of development of the lesion to which I wish to call attention, for in most text-books and monographs sufficient emphasis is not placed upon them.

It is, of all renal diseases, the most insidious in onset, and it is intermittent in progress. The kidneys are attacked now and again by uric acid or the toxic agent of gout in microscopic areas, which produce active lesions of almost microscopic size. It is true when we examine a kidney which illustrates interstitial nephritis that we find almost the whole parenchyma involved to a greater or less extent, but this extensive lesion has been slowly developed and is due to the confluence of minute changes which have been wrought intermittently. I have spoken of active lesions of microscopic size. These result in a destruction of glomeruli, tubules and arterioles, and when this destruction has been wrought the scar tissue which is left is inert and represents only a loss of renal function.

If the renal changes of gout are looked at in this way they will explain in part the symptomatology of the affection. As the glomeruli are attacked one after the other, not all of them or most of them simultaneously, and as those attacked today may atrophy and become useless before others are involved, the filtration of water from the blood will be modified only by the very few Malpighian bodies actively affected at the time. Therefore only traces of albumin appear in the urine, in marked contrast to the large quantities which appear when the kidneys are involved in parenchymatous inflammation. The atrophic and destructive changes which involve the renal epithelium cause a degree of renal insufficiency to slowly develop. The endarteritis which often obliterates the caliber of the arterioles and the destruction of capillaries in the diseased areas increases blood pressure. This causes the elimination of abnormally large amounts of urine by those glomeruli and renal tubules which are as yet unaffected and healthy.

The insidious and intermittent development of these lesions explains fully the pathognomonic symptoms; polyuria, with traces of albumin, few renal casts, diminished elimination of urinary solids and high arterial tension.

High arterial tension in gout is due in part to uric acid or other toxic substances in the blood which increase the tonus of the arterioles. The destructive agent, whatever it is, that attacks the kidneys produces also arterio-sclerosis or atheroma in the arteries of other tissues. The thickened and rigid walls of the arteries impede circulation, which stimulates the heart to contract more forcefully. This is a second factor producing increased blood pressure. Arterio-sclerosis in the kidneys, in the central nervous system, in the

various tissues of the body, often produces serious or fatal complications in those who have a gouty diathesis. Because the walls of arteries are weakened by disease and stretched by blood under unusual pressure aneurysms sometimes develop in the smaller or larger arteries. The smaller ones are frequently the cause of apoplexy.

The commonest and most characteristic cardiac change associated with gout is hypertrophy. The left ventricle is chiefly thickened, although when the heart is hypertrophied both ventricles are affected more or less. The cardiac hypertrophy is, without doubt, due to increased work which the heart has to do because of the destruction of some arteries and capillaries and diminished caliber and rigidity of others. These changes in the arteries generally necessitate compensatory cardiac hypertrophy and as the arterial changes become more widely diffused, little by little and intermittently, the cardiac hypertrophy slowly increases so long as the nutrition of the individual is good. Ultimately cardiac compensation becomes impossible, cardiac fatigue, dilatation and exhaustion develop. Endocarditis and pericarditis almost never complicate gout. In this respect the contrast with articular rheumatism is striking. It is true that deposits of urates have been found on the valves in gout, but such cases are extremely rare and exceptional.

These various changes in the kidneys, arteries and heart may occur in podagra or characteristic gout, but are more frequently seen independent of it and often themselves constitute the most marked manifestations of a gouty diathesis.

Treatment, to be of value, must be hygienic. It will aim to prevent the accumulation in the blood of those substances, uric acid or whatever they may be, which produce the vascular, cardiac and renal changes. Necessarily treatment must be continued for a long time. By removing the cause of these complications of gout their progress may be brought to a halt and a patient may be made comparatively comfortable. It is impossible to restore renal tubules and glomeruli which have been destroyed. But it is rare that the renal changes are so extensive that they, of themselves, will prove destructive of life. In almost every instance, when the disease is recognized, enough healthy renal tissue exists so that if its cause is removed the kidneys will continue to do their work fairly well, and maintain a condition of physiologic health even though they are anatomically much deformed. The same can be said of the inhibition of the growing cardiac and vascular lesions by suitable treatment.

CHRONIC INFLAMMATION AND ULCERATION OF THE DUODENUM, WITH RESULTANT REFLEXES.

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BY JOHN M. ALLEN, A.M., M.D.

PROFESSOR OF THE PRINCIPLES AND PRACTICE OF MEDICINE AND PRESIDENT UNIVERSITY MEDICAL COLLEGE, KANSAS CITY, MO.
LIBERTY, MO.

The duodenum is abundantly supplied with arterial blood. Its veins empty into the portal vein, any obstruction of which, either from the lungs, liver or heart, produces passive congestion of this organ. Its nervous connection with the brain is by branches from the par vagum and with the spinal cord by branches

from it, with the sympathetic system by branches from the solar plexus, and with the thoracic plexus by the splanchnic nerve. It has within its structure the plexus of Meisner and Auerbach. It is studded with the glands of Lieberkühn and Brunner, and with solitary glands as well as lymphatics. It is the physiologic receptacle of the chyme which is often perverted by functional disease of the stomach or over-repletion, either of which may be highly irritating to the duodenum. It also receives the secretion from the liver and pancreas and performs, possibly, as important a function in the digestive process as does the stomach, and is as frequently subject to physiologic hyperemia. Indeed, there is no structure in the human body which is so often exposed, both internally and externally, to as many varieties of disease-producing forces as the duodenum. It is directly connected with the brain, spinal cord and sympathetic system as no other portion of the alimentary canal is, which fact makes it the great reflex center of this canal.

Careful analysis of a large number of cases of reflex irritation supposed to have originated at other points of this canal, disclosed the fact they always originated in the duodenum. It is far more frequently diseased than one would infer from the scant literature on the subject.

In my clinic at the University Medical College of Kansas City, Mo., 10 per cent. of the cases had chronic inflammation and ulceration of the duodenum. In my private practice the percentage is about the same. As compared with chronic gastritis—or so-called catarrh of the stomach—it is far more frequent. In 17,000 clinical patients I found 666 of duodenal disease, and only one case of chronic gastritis and one of ulcer of the stomach. In fact, I never saw a case of chronic inflammation of the stomach, excluding chemic and other irritants as causative, that did not proceed by extension from inflammation of the duodenum. It is most often found between the ages of 20 and 40, but it frequently occurs in childhood, the result of enterocolitis or cholera infantum. It occurs in females more often than in males because of their more numerous reflexes.

Typhoid fever is the most frequent cause. Other causes are phthisis pulmonalis, functional disease of the stomach, croupous dysentery by extension, the exanthematous fevers, particularly rubeola, acute inflammation of the organ, passive congestion, burns, acute inflammation associated with pneumonia, which obstructs the bile ducts, producing what the older writers called bilious pneumonia, and reflex irritation, the result of disease of the uterus and appendages.

Brown-Séquard asserted years ago that reflex irritation was sufficient to arrest secretion and nutrition, and produce inflammation. This fact is beautifully illustrated in severe burns of the skin, which, by reflex irritation, result in acute inflammation of the duodenum, and *vice versa*, irritation of the duodenum will produce inflammation of the skin, as in urticaria and eczema. In childhood we have a clear example of the effect of irritation of the intestinal canal, producing reflex irritation of the nerve centers, attended with functionary changes in the blood of these centers, thereby producing convulsions.

Esquirol, Pinel, Cullen and others believed and taught that gastro-intestinal irritation was a frequent cause of insanity, yet they were not able to give a reason why this should be so. In the light of mod-