INTERMITTENT CLAUDICATION.

BY

WILLIAM OSLER, M.D., F.R.S.,
Professor of Medicine, Johns Hopkins, University.

Reprinted from the Montreal Medical Journal, February, 1902.

INTERMITTENT CLAUDICATION.

BY

WILLIAM OSLER, M.D., F.R.S.,

Professor of Medicine, Johns Hopkins University.

In 1877 or 1878, when studying comparative pathology, I went one day to the country with some of the members of the Montreal Veterinary College to see an autopsy on a horse which had had a peculiar form of intermittent lameness. Dr. McEachran said the condition was well recognized, and had been described by the French writers, but it was very obscure. I have forgotten now the details of the autopsy, except that we found verminous aneurisms of many of the mesenteric vessels and of the iliac arteries. At the time I was much interested, and looked up Bouley's paper on *Claudication Intermittente*. He described an affection in the horse, in which, after being driven for fifteen or twenty minutes, the animal stopped, one or both of the hind legs got stiff, and soon it was unable to stir. In from half an hour to an hour it recovered and was able to go on comfortably for another fifteen minutes, when the attack recurred. In such cases, post-mortem, the artery of the affected limb was found blocked with a clot, or, when both hind legs have been involved, the abdominal aorta contained thrombi.

The subject was not brought to my attention again until a few years ago, when working at the subject of angina pectoris. I then looked up Charcot's description of this intermittent claudication in man, and made also the interesting discovery that Allan Burns in his *Observations on Some of the Most Frequent and Important Diseases of the Heart, 1809*, had given an explanation of this remarkable phenomenon.

One or two of his sentences I may quote: "In health, when we excite the muscular system to more energetic action than usual, we increase the circulation in every part, so that to support this increased action the heart and every other part has its power augmented. If, however, we call into vigorous action a limb round which we have with a moderate degree of tightness applied a ligature, we find that then the member can only support its action for a very short time, for now its supply of energy and its expenditure do not balance each other; consequently, it soon, from a deficiency of nervous influence and arterial blood, fails and sinks into a state of quiescence." He puts it very tersely when he says, "the supply of energy and expenditure do not balance each other."

Charcot was the first to describe a condition in man identical with that met with in the horse. His Memoir was presented to the *Société de Biologie* in 1856, and is also to be found in the *Leçons du Mardi, I.* One day a patient in the service told him that he was not able to walk for more than a quarter of an hour without being taken with cramps in the legs. After resting a while he would get better, and would be able to resume his walking, and then a crisis recurred. At the autopsy Charcot found a ball encysted in the neighbourhood of the iliac artery, and a traumatic aneurysm which had obliterated the artery in its lower part. The circulation was carried on by collateral channels, which were ample to maintain the nutrition while the patient was quiet, and for a short period during exertion, but after a time, when the limbs were fatigued by the movements, the quantity of blood which reached them was insufficient, causing a relative ischæmia, with tingling, cramps, and impossibility of walking. He refers to the fact that the condition is often preliminary to gangrene, and narrates a case in which a patient with the affection had his leg amputated for gangrene.

Interest has been reawakened in the subject by the very careful studies of Erb (Deutsche Zeitschrift für Nervenheilkunde, 13), in which he has reported twelve cases, and has called attention particularly to its association with arterio-sclerosis and calcification of the arteries of the legs. The whole subject, too, has been reviewed this year (1901) by Goldflam in the *Neurologisches Centralblatt*, and in this country cases have been reported by Gordon (New York Medical Journal, 1900), and by Riesman (American Medicine, 1901).

Familiar as I had been for years with the disease in the horse and with the early literature on the subject in Burns' work and with Charcot's description, I had never recognized the condition clinically until in the patients whose histories I here give.

Case I. *Vomiting and pain in abdomen—Pulsating tumor in epigastric region—History of syphilis—General arterio-sclerosis—Wiring and electrolysis of aneurismal sac—Marked improvement—Return in nine months with well marked intermittent claudication.**

W. B., aged 21, from Virginia, came first to the hospital in December, 1899, complaining of vomiting and great pain in the upper abdomen. These symptoms had been present for several months. He had lost in weight and had become very nervous. He had been a healthy fellow, but had had syphilis six or seven years before. The radials were sclerotic, the aortic second sound ringing and accentuated, and in the epigastric region there was a wide area of impulse; on palpation an expansile tumor which could be easily grasped in the hand. I urged him to have the sac wired. To this he consented and went home to settle his affairs. He returned early in January, and Dr. Finnie opened the abdomen and found an aneurism of the abdominal aorta, into which he inserted ten feet of wire, through which he passed an electric current for an hour. The patient did well and returned to his home very greatly benefited, particularly in the relief of the pain. He returned in October, 1900, for examination. He had continued free from pain and vomiting. His general condition was excellent, though he was still nervous and apprehensive. The sac was decidedly smaller and the area of pulsation much less.

He volunteered the statement that there was an additional symptom which had disturbed him not a little; namely, after walking for a certain distance his legs would, as he expressed it, give out completely; so that he could not move another step, and had to sit down. After resting a few minutes he could then go on again. This was more particularly noticeable when he walked on the street. He had to go very slowly and could not go for any distance. There was no paralysis accompanying the loss of ability to walk. He could move his legs, but there was an uncontrollable feeling that he could not take another step. Accompanying this there was a sensation of dead, heavy weight in the legs, but no cramps. Walking about in the house (and in the yard) did not bring on the condition, but he had had it very frequently in the past few months, and he had learned to ward it off by walking very cautiously and slowly and resting at intervals. The femoral arteries and the dorsal arteries of the feet were distinctly sclerotic.

* As I look over this paper for the press this patient has been readmitted to the hospital (January, 1902). He has remained very well since the operation two years years ago. The aneurism can be felt. It is hard and firm. He has no pain, but is still very neurasthenic. He has not had the intermittent claudication for nearly a year.

In aneurism of the abdominal aorta the condition is the same as that which produces the intermittent claudication in the horse, and one can readily understand how, as Allan Burns expressed it, the supply of energy and expenditure did not balance each other. In fact, it is surprising that lameness is not more common in such cases.

The following case is a typical illustration of the more frequent cause; namely, general arterio-sclerosis. The patient had, moreover, the associated vaso-motor and nervous disturbances which are not uncommon with disease of the arteries of the extremities.

Case II. *Mitral stenosis—General arterio-sclerosis—Attacks of intermittent lameness with numbness and tingling in the feet and marked vaso-motor disturbances—Absence of pulsation in the dorsal arteries of the feet.*

Mrs. W., aged 55, admited June 7th, 1900, complaining of pains in the right leg, difficulty in walking, and heart trouble. There was nothing of any special moment in her family history. Her mother died of tuberculosis, and probably one sister. She had had the usual diseases of childhood, and had acute articular rheumatism at sixteen. She had had seven children and five miscarriages. The last child was born seven years ago. She had always enjoyed good health, and had had no serious illnesses. She said, however, that she had had heart trouble all her life, and occasional attacks of shortness of breath.

Present Illness. While at Baden last August she went out for a walk after eating a very hearty dinner, and after going a little distance from the hotel she lost control of her legs. There was no pain, but they simply refused to carry her, and she had to be carried back to the hotel. There was no loss of consciousness. She was very much alarmed about herself, and she was given aromatic spirits of ammonia, which made her very nauseated, and a little while later she vomited. The following day she felt well enough to leave Baden. Prior to this time she had begun to suffer a good deal with dyspnœa on exertion. She stood the journey back to this country very well, and remained quite well until about six weeks ago. Walking rapidly one day to the boat at Norfolk, she got somewhat out of breath. She got on the boat all right, and felt quite well until she reached Fortress Monroe, when she found on attempting to get up she was unable to walk. She had at this time a feeling of pins and needles in her feet, chiefly in the right foot. There was no difference in the color, and no swelling. About three weeks ago it was noticed for the first time that the right foot and leg were slightly blue, and she has had a good deal of pain in this foot and leg, sometimes sufficient to require

morphia. For the greater part of the time since the attack she has been in bed. On attempting to move about the legs give way. The pain in the right leg is much intensified if the foot hangs down. She has been very much worried and disturbed about herself, but her general health has been pretty good. She does not think she has been more short of breath of late. She has had a little palpitation and pain about the heart. The dyspnœa is altogether on exertion.

Present Condition. The patient was a medium sized woman, quite stout and looked nervous. The tongue was clean. She gave a very good account of her history and condition. The radial pulse was regular, 96, vessel wall not sclerotic. No sclerosis of the temporal arteries. The pupils were equal, and reacted to light and on accommodation.

Heart. Point of maximum impulse was visible in the fifth interspace about the nipple line. There was an exaggerated systolic impulse on palpation; no definite thrill. On auscultation there was an extremely sharp, flapping first sound at the apex, almost amphoric in tone, and preceded by a short, rumbling murmur. There was a soft systolic bruit at the aortic area, and the second pulmonic sound was loudly accentuated.

The abdomen was not swollen; liver and spleen not enlarged.

Legs. Both could be moved freely in bed. Power of movement of right toes and ankle slightly impaired. The right leg looked cyanosed from the knee down. There was no œdema. It was extremely tender to the touch. The right calf measured the same as the left—31½ cm. Left leg and foot normal in size and color, and not tender to the touch. Both feet felt cold, the right more so than the left, and she complained very much of the numbness in them. There was no pulsation to be felt in the dorsal artery of the right foot, nor in the right popliteal artery. Slight pulsation to be felt in the femoral artery. No pulsation in the dorsalis pedis or popliteal arteries of the left leg. Pulsation in the left femoral was well felt. Pulsation in the external iliacs could be just felt. There were no patellar reflexes in either leg, and the plantar reflexes were very difficult to obtain as she winced so much from tenderness of the soles.

The patient had warmth applied to the legs, careful friction, and she did remarkably well. On the 11th there was no cyanosis in either the leg or foot. It was still cooler to the touch and tender. No pulsation could be felt in the femoral artery.

I heard subsequently from this patient's daughter that she died a month or two after leaving the hospital.

This case illustrated the good effects of careful treatment as recom-

mended by Erb. With rest in bed, warmth to the legs and careful friction she improved very much. She received great benefit too from the use of full doses of nitroglycerine.

A word as to the name. I think it is very much better to use the term intermittent claudication, though it does not specify the etiology. It expresses well the most characteristic feature of the complaint. Erb's term, *intermittirendes Hinken*, is simply the German equivalent. Other terms have been used, such as *angio-sclerotic intermittent dysbasia* by Charcot, *intermittent muscle paresis* by Erb, and *angio-sclerotic paroxysmal myasthenia* by Higier, the author of a long article on this subject in *Deutsche Zeitschrift für Nervenheilkunde*, July, 1901. As shown in the horse and in the first case which I here report, the affection is not always due to simple arterio-sclerosis, but may be due to aneurism, as in Charcot's case and as in the rule in the horse. Oppenheim has reported instances in nervous individuals in which the condition seems to depend upon vaso-motor disturbances.