

DIABETIC COMA SUCCESSFULLY TREATED BY SALINE TRANSFUSION; NO RELAPSE FOUR WEEKS AFTERWARDS.

BY THOMAS OLIVER, M.A. DURH., M.D. GLASC.,
F.R.C.P. LOND.,

PHYSICIAN TO THE ROYAL INFIRMARY, NEWCASTLE-UPON-TYNE.

It is the general experience of those who have had to treat diabetic coma by saline transfusion that while consciousness may be restored this is of short duration, for the individual after a few hours lapses back into coma, from which a second saline injection fails to arouse him. In putting the matter thus I believe that I am only stating what has been the experience of most physicians. There have of course been exceptions and I venture to record the following case because the notes relate rather an unusual experience—viz., that at the time of writing the patient has remained perfectly intelligent during the twenty-eight days which have elapsed since the transfusion.

A man, aged thirty years, was on June 25th, 1898, admitted into the Newcastle-upon-Tyne Infirmary under my care suffering from diabetes of fully eight months' duration. Nine months ago he became very thirsty and began to drink large quantities of water. He would frequently drink three large bottlefuls of herb beer at a sitting and his night's rest was broken by his being obliged to get up to assuage his thirst. Eight months ago, as he found himself getting weaker, he gave up work. For the previous eleven weeks he had been very constipated, a condition hardly relieved by aperient medicine. Since his thirst became established he had passed large quantities of urine and he had lost weight to the extent of 4 st. There was nothing of importance in the patient's family history. Beyond having had pleurisy five years ago and two mild attacks of influenza since, he had been a very healthy man, and although he had lost flesh he was still well covered. His skin was dry and lemon-coloured, his tongue was dry and covered with a white fur, and the saliva, which was acid, did not contain any sulphocyanic acid. Upon his thighs there were several brown patches, the remains of pustules which came out a few months before. The knee-jerks were absent, the heart and lungs were healthy, and the skin of the abdomen was at places chocolate-coloured. The liver dulness was normal and the splenic dulness was slightly more pronounced than usual. The patient was placed upon diabetic diet and codeia, and on the whole appeared to make fair progress. On the afternoon of July 12th he left the infirmary in order to go home by rail, a distance of thirteen or fourteen miles, but he was brought back to the infirmary the same evening in a state of coma. He had, it appears, reached the station all right, but there he must have become confused, for a few hours afterwards he was found in a railway carriage at Tynemouth in a state of unconsciousness and on a different line of railway from that by which he should have travelled. Dr. Rowell, the house physician, recognising the patient who had left the infirmary only a few hours previously and finding him comatose, at once transfused him with two and a half pints of saline solution, passing it slowly into the right median basilic vein, and also freely purged him. The saline solution was composed of one drachm of chloride of sodium to one pint of boiled distilled water, the temperature of which was reduced to 112° F. During the operation the patient became sufficiently revived to complain that he felt pain. Before the transfusion his pulse was beating at the rate of 45. By degrees consciousness was regained and has since been retained. For the first three days after the transfusion a very small quantity of urine was passed daily—only 21 oz. instead of from 140 to 200 ounces when he was first in the infirmary. As the patient was known to be suffering from diabetes it was unfortunate that his urine was not examined on his readmission during and immediately after the coma, but the day afterwards it was found to be free from albumin and acetone, although containing sugar. Since then the amount of urine passed has gone up by leaps and bounds until he passed daily 190 oz. On July 15th the skin on the back of his hands was rather more bronzed than usual, the pulse was 52, markedly dicrotic and compressible, the tongue was dry, and the saliva was acid. The pupils were slightly dilated. The

urine on July 15th was acid and its specific gravity was 1025; it contained sugar, but neither albumin nor acetone. On July 18th the patient complained of slight numbness in the right hand, but while there was slight anæsthesia there was no analgesia, although there was quite distinct paresis of the extensor muscles of the wrist compared to those of the left hand. There was very slight wrist-drop.

The case is interesting mainly on account of the length of time consciousness has been retained since the transfusion, which is a cogent reason why in diabetic coma such a line of treatment should be adopted. Not only was the patient rescued from a condition which in all probability would have proved fatal, but he has been so restored that had he been a wealthy man with a will to make or business matters to arrange he would have been able, with his present state of health and clearness of intellect, to accomplish it. I regret that there is no mention on his clinical chart of the quantity and the character of the urine passed in the twenty-four hours for the two days prior to his leaving the infirmary. In most cases I have noticed that before diabetic coma supervenes the patient passes a very small quantity of urine compared to what he had passed previously and that usually this urine contains albumin. Sometimes, indeed, there is almost complete suppression of the secretion of urine. During diabetic coma if a catheter is passed into the bladder perhaps only a few drachms of urine are drawn off, which contains both sugar and albumin. Then for the first time we learn from the nurse that no urine has been passed for the previous twenty-four hours or more. While therefore I do not deny that diabetic coma may be due to acetonæmia caused by the presence of diacetic acid in the blood, I am disposed to regard it rather as the result of a mixed toxæmia in which uræmia plays not an unimportant part. Professor Bedson found albumin, sugar, and acetone in the urine of one of my own patients, but no trace of acetone or its congeners in the blood, which, considering the supposed relationship between acetonæmia and diabetic coma, is rather an interesting fact. I should like, in conclusion, to draw attention to a fact of therapeutical interest, and that is the influence which cocaine often has in overcoming the obstinate constipation of diabetes. Where aperients fail, cocaine in small doses ($\frac{1}{4}$ gr. twice or thrice daily) will not only brace up the muscular system generally and remove the sense of fatigue so frequently present in these patients, but act so decidedly upon the musculature of the intestine that its peristalsis is increased and constipation is overcome.

Newcastle-upon-Tyne.

THE OCCURRENCE OF ERYTHROMELALGIA IN DISEASE OF THE SPINAL CORD; AN ACCOUNT OF TEN CASES.

BY JAMES COLLIER, M.D., B.Sc., M.R.C.P. LOND.,

JUNIOR HOUSE PHYSICIAN TO THE NATIONAL HOSPITAL FOR THE
PARALYSED AND EPILEPTIC, QUEEN-SQUARE, W.C.

THE association of erythromelalgia with symptoms indicative of lesions of the central nervous system has been so frequently observed that the opinion of recent observers supports the opinion originally put forward by Weir Mitchell¹ in 1878 when he first clearly described the clinical picture of the condition. He said: "I believe the group of cases now described represent an unrecognised type of spinal or cerebro-spinal disease and that they may be found in the future associated with distinct lesions in definite regions." Lewin and Benda,² discussing a series of cases collected from various writers, consider that erythromelalgia was not a disease *sui generis* but generally a symptom of definite disease of the central nervous system or of functional disease. Auerbach and Edinger³ last year published a case of tabes in which erythromelalgia confined to the right foot was a conspicuous symptom. After death degeneration of the posterior roots of the lower lumbar and sacral nerves

¹ S. Weir Mitchell: American Journal of the Medical Sciences, July, 1878.

² Lewin and Benda: Berliner Klinische Wochenschrift, 1894 Band xxxi., pp. 53, 87, 117, and 144.

³ Auerbach and Edinger: Nervenheilkunde, Sept., 1897.

almost confined to the right side was found. The spinal ganglia and peripheral nerves were thoroughly examined and found to be normal. The degeneration principally affected, in the spinal cord, the tract lying close to the septum on the right side usually described as rising from the upper sacral roots, which were in this case found totally degenerated; the erythromelalgia was confined to the skin areas supplied by the fifth lumbar and first and second sacral roots. Very few cases of erythromelalgia have been reported in this country and it is not recognised as a condition occurring in spinal disease. There seems reason to believe, however, that the condition would be found not infrequently if looked for and may be of great importance in the diagnosis and prognosis of nervous disease.

Erythromelalgia is characterised by acute vaso-dilatation over a definite area of the body. The skin assumes in marked cases an intense purple-red colour, swells a little and becomes tense and shiny. The superficial veins of the affected region stand out prominently. The skin can be felt to pulsate and the surface temperature is raised often as much as two degrees above that in the non-affected regions. Severe burning pain, often described as being like a strong galvanic current applied to the skin, accompanies the redness. When the affected area is analgesic patients experience intense tingling, but no pain. The attacks may appear spontaneously or may be induced by exertion, warmth, or emotion. The effect of position upon the affected region is remarkable, so that when the foot is affected an attack may be often brought on at once by merely allowing the foot to hang down, and during the attack that position always increases the pain and the vascular phenomena. Many cases have been reported in which patients have been entirely unable to stand on account of the pain the erect position produced. Elevation of the affected part always causes the pain and the state of vaso-dilatation to pass off so long as the elevated position is maintained. During the attack the application of warmth to the part affected increases the pain and cold greatly relieves it. The surface temperature of the erythromelalgic skin is raised when the part is dependent sometimes to a remarkable degree and lowered when the part is raised. In a normal limb the reverse is the rule. This effect of position upon the vaso-motor state and temperature of the limb is highly characteristic of erythromelalgia. Tender points are frequently observed over the affected area and hyperæsthesia is common. Localised sweating of the skin during the attack is common and thinning of the skin, with brittle, reeded nails, sometimes occurs. The distribution of erythromelalgia is generally peripheral upon the limbs. Rarely the face and trunk have been affected. The lower extremities are more frequently affected than the upper; the distribution is not that of nerve-root or nerve-trunk—it is often limited by a sharply marked circular line round the limb. As a rule the limbs are affected symmetrically, although frequently one limb is affected before the other. The condition shows a marked tendency to spread until the whole distal part of the limb is affected. The duration of the attacks varies from half an hour to several weeks⁴; it is usually three or four hours. Hæmoglobinuria has not been found associated with the condition. The patients rarely suffer with chilblains or “dead fingers.”

I have observed ten cases showing erythromelalgia in different degrees of severity at the National Hospital for the Paralysed and Epileptic during the last six months and I am much indebted to my chiefs—Dr. Jackson, Dr. Buzzard, Dr. Bastian, Sir William Gowers, Dr. Tooth, and Dr. Colman—for permission to publish the following notes.

CASE 1. Disseminated sclerosis.—The patient was a woman, aged twenty-nine years, of healthy appearance. The symptoms of spinal cord disease began nine years before admission. On admission examination revealed pallor of both optic discs, slight weakness of both external recti, diplopia, and slight nystagmus. There was no speech defect. There were slight intention tremor of the hands, loss of control of both sphincters, severe spastic paraplegia with some ataxy of the legs, inability to stand, double rectus and double foot clonus, no sensory disturbance, and some hyperæsthesia of the feet. Seven years before admission she noticed attacks of burning pain in both feet associated with great redness and heat.

These occurred spontaneously and whenever the feet became hot. The attacks lasted about three hours and were at once relieved during that time by the recumbent posture, but the pain and redness returned when she put her feet to the ground. A year later the feet gradually became permanently red and the dependent position of the legs became an active factor in producing the attacks. Sixteen months before admission the feet always became purple and painful when allowed to hang down. The redness, up to this time confined to the feet, extended as high as the knees. The feet swelled during the attacks and sometimes subcuticular hæmorrhages occurred. The feet and legs were then constantly red in whatever position they were placed. Since this time up to her admission the attacks continued, but less frequently than before. As she lay in bed the legs showed a red blush limited sharply above by a circular line just above either knee, which below the middle of the calves changed to a deep purple colour and over the feet showed some mottling. The feet were cold to the touch and pressure produced an intensely white mark which slowly became red. When the feet were hung over the edge of the bed or when she assumed the upright posture they became of a much brighter purple colour and became very hot to the touch. The skin became tense and shiny and the feet swelled but did not pit on pressure. The veins stood out and the whole foot and leg below the knee could be felt to throb. She had intense burning pain limited to the region of redness. A surface thermometer was applied to the dorsum of each foot for ten minutes, the feet being raised above the level of the body. A second application was made for a similar period, the legs being allowed to hang over the edge of the bed. The temperature in the raised position was as follows: in the right foot 94·8° F., in the left foot 94·6°; in the dependent position, right foot, 96°, left foot, 95·8°; the difference being—in the right foot, 1·2°, and in the left foot, 1·2°. On two occasions while she was under my care I was called to this patient in the night and found her crying out with pain in the legs, which presented a condition of erythromelalgia even more severe than that just described as the result of hanging the legs down and with similar distribution. She was relieved by the application of cold and by bandaging the feet tightly with Martin's bandage.

In this case the following points are of interest. The pain and vaso-motor disturbance always appeared simultaneously. The attacks were at first spontaneous only but subsequently they were often brought on by the dependent position of the lower extremities and lastly always induced by the dependent position. When this patient was wearing tightly applied Martin's bandages from the foot to the knee she could sit with the legs down in comparative comfort. The slow supervention of a gradually deepening permanent vaso-motor palsy, uninfluenced by the position of elevation of the limbs, and occurrence of acute spontaneous vaso-motor disturbance in its area of distribution may have a most important bearing on the pathology of erythromelalgia.

CASE 2. Disseminated sclerosis.—The patient was a woman, aged forty years, delicate-looking and sparsely nourished. Ten years before admission she had transient amblyopia of the left eye. Her present symptoms dated from a chill seven months before admission. At the time of admission there were pallor of both optic discs, concentric contraction of both visual fields and slight nystagmus on extreme lateral deviation. Speech and the upper extremities were normal; the lower extremities were very weak in all movements. There was marked wasting of the muscles in the left thigh and calf; dorsiflexion of either ankle was impossible; the gait was peculiar and strongly suggestive of functional disease. There was loss of sense of position below the hips. There was no anæsthesia. The sphincters were natural, the knee-jerks were difficult to elicit, and the plantar reflexes were very faint. Four months before admission she noticed sudden burning pain in the feet as she was walking. The pain lasted some time and occurred on several occasions. Soon afterwards similar pain was noticed at night and she thought a very hot foot-warmer was in the bed and asked to have it removed. These attacks frequently recurred. A month before admission the feet became red and painful when she stood up. The recumbent position at once caused the pain and redness to disappear, as did the application of cold. As she lay in bed the feet presented a natural appearance. Directly the feet were lowered typical erythromelalgia was seen in either foot, limited by a sharply marked circular line just

⁴ Two cases with attacks of very long duration are reported by Cristiani, *La Riforma Medica*, Torino, 1894, vol. iv., p. 4.

above the malleoli. At times for days together no vaso-motor phenomena appeared when the patient stood up and at other times the erythromelalgia would appear in one foot only. When the feet were warm to the touch as she lay in bed the vascular phenomena always appeared if the legs were allowed to hang down or if a ligature were tied round the calves. There was no persistent vaso-motor palsy. The soles of the feet and the calves were very tender on pressure. There were no trophic changes. This patient had several spontaneous attacks when in the recumbent position. Subsequently she developed complete paraplegia, reflex flexor spasm, complete anaesthesia below the knees, and loss of control of the sphincters with cystitis. She became completely bedridden and since then the vaso-motor disturbances have not again appeared.

The aspect of this case at first was strongly suggestive of functional disease. The presence of erythromelalgia at that time was much in favour of organic disease. The diagnosis of disseminated sclerosis was fully confirmed by the subsequent course of the illness.

CASE 3. Disseminated sclerosis.—The patient was a woman, aged twenty years. Her symptoms began three years ago. When admitted to hospital she had slight weakness of the right arm with marked incoördination and intention tremor, slight incoördination and tremor in the left hand, spastic paraplegia with marked ataxy, no anaesthesia, and loss of control of the sphincters. The knee-jerks were exaggerated, there was double foot clonus, and the triceps and supinator jerks were absent. While in hospital she first noticed severe attacks of burning pain in both feet when they became warm in bed at night, lasting a variable time from a few minutes to several hours. There was well-marked erythromelalgia over both feet limited above by a circular line just above the ankle, that on the right foot being more intense. During the attacks the dependent position of the feet increased all the symptoms and raising the limbs above the level of the body alleviated them, but the dependent position did not seem to be a factor in inducing the attacks. A month after the appearance of the erythromelalgia a condition of permanent vaso-dilatation made its appearance over the same area and gradually deepened in intensity as the attacks went on. Nine months later the attacks became much more frequent; she became quite paraplegic. Since this time the vascular disturbances have gradually diminished and have now been absent for some months.

CASE 4. Disseminated sclerosis.—This patient was a married woman, aged twenty-nine years. Her illness began after a confinement three years before admission. On admission she had unequal pupils, transient diplopia, spontaneous rotatory nystagmus, and coarse nystagmus on deviation. Speech was halting and there were slight tremor of intent in the upper limbs and marked incoördination and intention tremor of both legs. All movement of the lower extremities was very weak. Romberg's symptom was present. There were no anaesthesia or girdle sensation. There was reflex incontinence of urine. The knee-jerks were exaggerated, R. > L. There was double foot clonus. The skin over the outer side of the left foot, including the two outer toes, not extending on to the soles and extending an inch above the external malleolus, was of a red colour, sharply marked off from the surrounding skin, which was pale. The whole left leg below the knee showed a curious *tache* not unlike *urticaria scripta*.⁵ At times and for periods of twelve hours together when she stood up the red area above described assumed a condition of typical erythromelalgia and she had severe pricking pain confined to that area. Alteration of position had the usual effect upon the redness and pain. At other times the erythromelalgia could not be brought on by prolonged exertion or by immersing the feet in hot water or by placing a ligature round the limb. The soles of the feet and calves were tender to pressure. She had attacks of severe burning pain in both feet at night, but this occurred only twice while she was under my care and on neither occasion were the feet seen.

The noteworthy features of this case are the distribution of the permanent vaso-dilatation and the *tache*.

CASE 5. Neurasthenia with (?) disseminated sclerosis.—This patient was a single woman, aged thirty-four years. Six years before admission she had double oöphorectomy performed for the relief of dysmenorrhœa. The operation was followed by severe neurasthenia. For two years

she had been unable to walk or sit up and for the last year there had been slight trouble with micturition. All movements of the limbs were executed feebly and languidly. There was no wasting, tremor or ataxy. She could not sit up in bed and when she attempted to stand she fell "all of a heap." There was distinct left hemianæsthesia and occasional slight delay in micturition. The plantar reflexes were absent and the knee-jerks were brisk. There was left foot clonus. The nails of the fingers and toes were thin, reeded, and brittle; the skin of the hands and feet was soft and thin and perspired profusely. Three years ago she began to have attacks of pain in the hands and feet; sometimes in the hands only. They occurred at night, lasting some hours, and she got into the habit of sleeping with the hands above her head to lessen the pain. Soon after this time she noticed that the dependent position of the limbs brought the attacks on and she had been unable to put her feet to the ground for weeks together on account of the pain. The attacks were accompanied by excessive tenderness and profuse sweating of the hands and feet. They were much better in the winter than in the summer; often in the winter she had gone months without any attack. On examining this patient one morning I discovered a slight degree of erythromelalgia in the left foot. This had passed off by the following day and for the next month she showed no sign of the condition, though I repeatedly tried to induce it. At the end of this time I was called to her one morning and found her crying out with pain, a severe condition of erythromelalgia being present, limited in the hands by a line round the forearm an inch above the wrist and in the feet by a circular line just above the ankle. The left foot was more affected than the right. The hands and feet were perspiring profusely and she complained of a pain as if she were being burnt in the balls of the fingers and toes. Pressure over the heads of the metacarpal and metatarsal bones on the ventral aspect gave great pain. The effect of position and of the application of cold was typical. The following was the surface temperature taken on the dorsum of each hand: hands straight up in the air—right 88·4° F., left 88·6°; hands hanging down—right 90·6°, left 90·8°; the difference being, right 2·2°, left 2·2°. During the remainder of her stay in the hospital these phenomena appeared unvaryingly when the feet and hands were dependent and some degree of vaso-dilatation was constantly present over the areas affected by the erythromelalgia. In this case the erythromelalgia was the earliest organic symptom to make its appearance and together with the sphincter trouble and foot clonus of later onset suggest strongly the presence of organic spinal disease. In connexion with the oöphorectomy it is interesting to note that in several published cases in males erythromelalgia has been associated with aspermatism.

CASE 6. Disseminated sclerosis.—The patient was a woman, aged thirty years. Her symptoms had been present for five years. On admission she had concentric contraction of both visual fields, slight left ptosis, some weakness of the right external rectus, nystagmus, slight tremor on execution of fine movements in the upper extremities, severe spastic paraplegia with ataxy, less of muscular sense in the lower limbs, partial anaesthesia of the legs, and loss of control of the bladder; the knee-jerks were exaggerated and there was double foot clonus. For the last two years she had had attacks of a burning and uncomfortable but not painful sensation in the neck and ears, associated with great redness and heat of the skin. The attacks always occurred when she was recumbent and were brought on by motion. The skin over the whole neck as low as the clavicle and over the whole head, excluding the area of distribution of the fifth nerve, during these attacks became of a red colour, very hot and slightly swollen. The attacks lasted about half an hour.⁶

CASE 7. Tabes dorsalis.—The patient was a man, aged thirty-one years. He had had syphilis nine years ago. The symptoms had been present for two years. He had unequal Argyll-Robertson pupils, incoördination of movements of the upper extremities and complete loss of muscular sense in the three ulnar fingers of both hands, tactile anaesthesia and partial analgesia below the sixth cervical root distribution, absolute analgesia below the third lumbar root distribution, lightning pains, girdle sensation, absolute loss of muscular

⁵ A similar condition occurring in association with erythromelalgia is reported by Senator, *Berliner Klinische Wochenschrift*, 1892, Band xxix., pp. 1125-9.

⁶ A case of tabes with similar distribution is reported by Benda, *Berliner Klinische Wochenschrift*, 1894, Band xxxi., pp. 53, 89, 117 and 14

sense in the legs, Romberg's sign, and absent knee-jerks; there was no sphincter trouble. He had noticed for two months that the feet and legs as high as the knee became red, swelled and tingled all over when he tried to walk or when he was sitting with legs down and that sometimes they became very red and tingled when he was in bed. There was a typical condition of erythromelalgia over both lower extremities, gradually becoming less marked from below upwards and ceasing just above the knee. This varied much from day to day, at times being more severe, at others hardly noticeable or absent. Surface thermometry gave the following results: legs horizontal—right foot 85·6° F., left foot 89°; legs dependent—right foot 86·6°, left foot 90°; the difference being, right foot 1°, left foot 1°. (A Hicks' thermometer was applied to the dorsum of the foot for ten minutes in each case.) There were no trophic changes, no sweating, no tender points, and no persistent vaso-motor palsy. Whilst under observation he had severe gastric crises; during several of them attacks of erythromelalgia in both hands limited sharply at the wrists were seen, but the attacks were accompanied by tingling and not by pain, the absence of which in both hands and feet during the vascular disturbance is to be explained by the fact that both regions were analgesic.

CASE 8. *Tabes dorsalis*.—The patient was a man, aged fifty-two years. He had had syphilis twenty-five years ago and had had two attacks of lead colic. On admission he had unequal Argyll-Robertson pupils, incoördination and loss of muscular sense in the arms and legs, Romberg's sign, marked anæsthesia and analgesia below the sixth cervical root distribution (the anæsthesia splitting the middle finger), which become almost absolute below the tenth dorsal level; lightning pains, girdle sensation, severe gastric crises, and delayed micturition. The knee, wrist, and elbow jerks were absent. There was alopecia over the fifth lumbar and first sacral root areas. The skin over the feet was thin and shiny and the nails were brittle and reeded. A history of erythromelalgia was carefully inquired for with negative result. After he had been under observation a month one morning he had an attack of severe gastric pain and went to bed. Immediately afterwards both hands assumed a deep purple colour and felt as if they would burst. They tingled all over, the veins stood out, and the whole hands throbbed. The skin became very tense and swelled slightly. The redness was sharply limited above by the flexure of the wrist on the radial side and extended for 2 in. above the wrist on the ulnar side. It was quite symmetrical. Raising the hands above the head caused the redness and tingling to disappear; lowering the hands caused the symptoms to reappear and become intense. The following are the surface temperatures. Hands raised above the head—right 89·5° F., left 89·5°; hands hanging vertically—right 92·5°, left 92·5°; the differences being, right 3°, left 3°. The attack lasted three hours and for six hours after it had passed off some degree of erythromelalgia could be produced by allowing the hands to hang down. Up to the present time this patient has had no further attack. In this case, as in the last, the vascular disturbance was situated in an analgesic region and tingling appeared in the place of pain. In both cases the vascular and gastric crises appeared simultaneously.

CASE 9. *Myelitis*.—The patient was a man, aged thirty-five years. His symptoms began gradually in February, 1896. When this note was made he had severe spastic paraplegia with ataxy and reflex spasms, partial loss of sensibility from the xiphoid cartilage downwards and delay in micturition; the knee-jerks were exaggerated and there was double foot clonus. At Christmas, 1897, he first noticed attacks of burning pain in the feet associated with redness and heat. The attack occurred almost every night and he constantly required injections of morphia to ease the pain. There was a condition of marked erythromelalgia over both feet limited sharply at the middle of the calf. It varied greatly in degree at different times. Subsequently this patient recovered such power in the legs as to enable him to walk. The attacks still occur when he walks about much but are much less severe than formerly.

CASE 10. *Traumatic neurasthenia*.—The patient was a man, aged twenty-five years. His symptoms followed a severe fall on to his back fourteen weeks before admission. On admission all movements of the lower limbs were performed feebly and with much coarse tremor; there was no rigidity and no wasting. He trembled all over when he tried to walk and frequently dropped on his knees. His gait was

typically functional. There was intense tenderness over the last lumbar spine but no abnormality in the spine or pelvis. There was absolute analgesia and diminished tactile sensibility over the lower extremities below Poupart's ligament on either side. He had slight difficulty in starting the act of micturition. The knee-jerks were exaggerated and there was double foot clonus. While in the hospital this patient for the period of a fortnight had several attacks of erythromelalgia of slight degree, the distribution being from the knee downwards and quite symmetrical on the two sides. The redness was not sharply limited above but faded off gradually into the normal skin. The attacks, which occurred several times a day and lasted about two hours, became slighter and disappeared at the end of a fortnight. I have since heard that they have returned.

This case was without doubt chiefly one of traumatic neurasthenia, but the existence of slight sphincter trouble and persistent foot clonus suggested that there was some lesion of the spinal cord. The occurrence of erythromelalgia strongly supported the probability of this.

The occurrence of these cases within a short period of time argues that erythromelalgia is not a rare symptom of spinal cord disease. It has without doubt frequently been overlooked. In the absence of symptoms pointing to the disease of the nervous system erythromelalgia has been investigated as an entity and the earlier literature on the subject relates chiefly to "idiopathic" cases. Lannois⁷ in a collection of cases published before 1880 states that nervous symptoms are only rarely coincident. The prevailing opinion at the present time among German writers is the opposite extreme to this. When a case presents as one of definite disease of the nervous system, erythromelalgia, if it occur, may not be recognised as such and may be simply noted as "vaso-motor paralysis," the accompanying sensations being set down to some of the many subjective peripheral sensations occurring in spinal cord lesions. One of its most characteristic symptoms—pain—may be absent if there be analgesia, as was the case in the two tabetic patients cited. Again, the variability of the occurrence of the symptoms—often absent for long periods together, especially in patients who are confined to bed—renders it easy to be overlooked. I would suggest that if the symptom be looked for with a knowledge of the peculiarities distinguishing it from other forms of vaso-motor paralysis it will be found not uncommonly in disease of the spinal cord.

In several of my cases there occurred at first only spontaneous attacks; afterwards the conditions became frequently induced by the dependent posture, and later a condition of permanent vaso-motor palsy of greater or less degree made its appearance; the attacks meanwhile continuing. This sequence suggests an irritative lesion of nerve structures governing the blood-vessels being the cause of the vascular crises and of the progress of this irritative lesion to a partially destructive lesion being the cause of the persistent vaso-motor palsy, these phenomena in vaso-motor nerve elements being parallel with pain followed by anæsthesia in sensory nerve elements and with spasm followed by motor paralysis in motor elements. Weir Mitchell used the term "vascular storm" in reference to this condition and the term "vascular crisis" would, I think, be very apt, occurring as it does in tabes associated with gastric and other sensory "crises." Probably the same fundamental pathological processes underlie both sensory and vascular crises. In all my cases the vascular change was never preceded by the sensory disturbance, but either preceded it or the two appeared simultaneously. It seemed as if the sensory disturbance was a local result of the altered vascular condition of the part. I would lay stress on the fact that erythromelalgia may be the first symptom of organic disease of the cord and may be of great value in diagnosis and especially valuable in the differential diagnosis between functional disease and disseminated sclerosis.

⁷ Lannois: Sur Erythromelalgie, Paris, 1880.

DRAINS AT BODMIN.—At the meeting of the Bodmin (Cornwall) Rural District Council held on July 30th a complaint was made of an open drain at Port Isaac, whereupon a member remarked that personally he did not believe in drains, and added that there was no plague in Bombay before drains were made. Eventually it was decided to instruct the police to report persons throwing slops into the drain.