skin sensitivity test, no reaction. Radiograph of bones normal. Wassermann reaction negative.

Treatment.—The patient suffered very little disability from his oedema and was quite able to enjoy a normal life. He complained of some "tiredness" of the affected limb at the

end of the day, but despite this he would voluntarily indulge in gymnastic exercises after a full day's work. No surgical intervention was therefore advised. Firm bandaging had some effect in controlling the oedema and also gave him a feeling of support.

Discussion

Examples of unilateral or bilateral oedema of the extremities for which no obvious cause can be discovered are not infrequently encountered; cases with a clear hereditary or family history are, however, much less common, and few have been recorded in British medical literature. The question arises whether patients who display the typical oedema of one or both lower limbs as described above, but who lack the appropriate family history, should be regarded as suffering from Milroy's disease. In this connexion we note that Parkes Weber, discussing at a meeting of the Royal Society of Medicine a family of four sisters in whom oedema of the legs began between the ages of 20 and 23 (Weber and Schlüter, 1937), considered that if the other features were present a family history was not essential for the diagnosis, and for that reason preferred the eponym Nonne-Milroy's disease to the term hereditary oedema. However, the heredo-familial factor in the classical descriptions quoted above is so striking that it seems to justify a separate nomenclature for the hereditary and non-hereditary forms. In describing the aetiological factors it was noted that one unaffected person in the family reported by Hope and French (1907) and two unaffected persons in the family reported by Milroy (1892) produced affected offspring. Apart from these gaps, however, the line of inheritance is direct. It appears, therefore, that the responsible gene is transmitted as a Mendelian dominant with occa-

sional skipping of a generation. In view of this possibility of skipping, the fact that the disease has been recognized for the first time in members of a particular generation does not rule out the likelihood that it has been handed down from earlier generations, especially when it is remembered that the manifestations may be minimal, easily missed by patient and doctor. (In the families described above there was considerable case variation in the degree of oedema, and this was also found in the family reported by us.) Alternatively the condition may arise as a mutation, though this is impossible to establish.

Apart from the description by Milroy (1892) of the patient with testicular enlargement and subsequent disappearance of the oedema, we have not discovered any record of a patient who, like the one we have reported, suffered also from hydrocele and testicular abnormality. While there seemed to be definite evidence of diminished testicular function, it is not clear that there was any relation between this and the oedema, nor would it justify in itself any suggestion that an endocrine factor plays a part in the production of the oedema. Although the disorder has been recognized for over fifty years, the ultimate mechanisms responsible for the chief symptoms remain obscure.

Summary

Milroy described a hereditary form of oedema of the lower limbs for which there was no obvious cause.

This condition produces little or no disability.

A typical case, with bilateral hydrocele and delayed puberty, is described.

We wish to acknowledge our thanks to Dr. Hugh Garland, consulting physician to St. James's Hospital, and to Dr. J. A. Fraser Roberts for much helpful advice and criticism. Dr. W. McIntosh, Medical Director of Leeds Municipal General Hospitals, kindly granted permission to publish this case. Our thanks are also due to Miss C. E. Campbell for the photograph.

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TWO CASES OF PERIARTERITIS NODOSA WITH OBSERVATIONS ON AETIOLOGY, DIAGNOSIS, AND TREATMENT

BY

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Periarteritis nodosa was first described by Rokitsansky in 1852. Kussmaul in 1866 related symptomatology to clinical findings, and during the next 76 years some 350 cases of the disease were reported. Here are recorded two further cases.

Case 1

This patient, a Merchant Navy man, aged 43, was admitted to the Western Infirmary, Glasgow, on Oct. 20, 1946. He complained of an aching pain in the muscles of his back and legs, epigastric discomfort, anorexia, and loss of weight for four weeks. This had been associated with a low-grade pyrexia. He had had malaria in 1938, and in 1939 was treated for staphylococcal empyema by rib-resection and with sulphapyridine over a long period. Sulphonamides had also been administered three weeks and again three days before admission to the infirmary.

On admission the abdomen showed slight generalized distension. It was "doughy," and on palpation the epigastrium and

both lumbar regions were tender and rigid. The muscles of the legs were tender on pressure; no oedema was observed. No other physical abnormality was detected. Temperature was 99.2° F. (37.3° C.), pulse 105, and blood pressure 150/90. During the next 17 days his condition deteriorated. The temperature was intermittent, ranging from 97 to 101.5° F. (36.1 to 38.6° C.). The pulse rate varied from 80 to 115. Muscular and abdominal pains and increasing general weakness accompanied these changes. On Nov. 8, and for several days subsequently, painful subcutaneous nodules were present on the abdominal wall. One such nodule was removed for histological examination.

After Nov. 15 his general condition improved, although two further exacerbations occurred. On Nov. 22 he complained of stiffness of the neck muscles and pain in the right testicle. On Nov. 19 pain in the lower left chest and left hypochondrium was accompanied by splenic enlargement. During this time the temperature continued to range between 97 and 101° F., and the pulse rate between 70 and 120. The erythrocyte sedimentation rate varied between 37:70 and 75:106 (Westergren).

These acute manifestations subsided within a few days, and the patient continued to improve generally. On Jan. 11, 1947, he was afebrile and symptom-free, and was discharged from hospital.

Investigations in Hospital.—*Blood*:—There was no evidence of anaemia. The white cell count varied from 27,200 to 11,000 per c.mm. The highest eosinophil reading reported was 5%. On Dec. 6 the serum albumin was estimated at 4.6% and total protein at 6.4%. The Wassermann reaction was negative.

Urine:—No abnormal constituents were found. Despite repeated investigations, tests for albumin and red blood cells were uniformly negative. *Fractional test meal*:—A normal acid curve was obtained. Blood was present in the fasting juice, and a test for occult blood in the faeces was positive.

X-ray examination:—Radiologically the lumbar spine, gall-bladder, chest, and arms revealed no abnormal features. Barium meal and enema investigation of stomach and bowel did not disclose any evidence of disease. *Ophthalmoscopic examination* (Nov. 23):—Prof. W. J. B. Riddell reported that no abnormality was detected in either fundus. An *electrocardiogram* (Nov. 23) showed slurring of QRS in leads I and III of a degree which is proof of myocardial change. *Proctoscopic examination* (Dec. 12) revealed no marked change in rectal mucosa. Excess mucus coming down from sigmoid was present; examination of this showed no abnormality. *Histology of subcutaneous nodule* (Nov. 14):—The report on the subcutaneous nodule removed from the abdominal wall was furnished by Dr. G. B. S. Roberts. It read: "The larger blood vessels show changes characteristic of periarteritis nodosa. There is fibrin present in the media and a gross collection of inflammatory cells, mainly polymorphs, in the adventitia."

Progress since Discharge from Hospital.—Since the patient was discharged from hospital (Jan. 11, 1947) three episodes of significance have occurred. (1) On Jan. 24 the patient's urine contained albumin, red blood cells, and granular casts. (2) On March 21 the patient complained of severe headaches, maximal in intensity over the vertex. On March 25 retinal haemorrhage in the left eye was reported, and on April 15 it was suggested that a further haemorrhage had occurred and that the vessel associated with this haemorrhage was raised from the retina. (3) On March 27 he complained of the passage of blood in his stools. This complaint persisted for three weeks. Further proctoscopic examination did not reveal any abnormal feature with the exception of a congested rectal mucosa. No specific pathogenic micro-organisms were isolated from a rectal swab. Bleeding time, clotting time, and blood film were within normal limits.

During this period of out-patient surveillance the condition of the myocardium improved, as shown by repeated electrocardiographic tracings. On Feb. 28 the electrocardiogram showed that slurring of QRS was not then of diagnostic significance.

Comment on Case 1.—The two main points of interest are: (1) the diagnosis was made during life, and (2) some thirteen months after discharge the patient is well enough to be in employment. The average duration of the disease

is given by Leishman (1937) as between three and four months. Some cases, however, have been known to recover.

Case 2

A housekeeper aged 52 was admitted to the Western Infirmary, Glasgow, on Dec. 13, 1946, with a complaint of pain in the chest and haemoptysis of ten days' duration and loss of weight and general weakness for some nine months.

Previous History.—She had been under investigation in a county hospital from July to November, 1946. The history and investigations covering this part of her illness were as follows. Subsequent to an influenzal illness occurring over Christmas, 1945, she experienced weakness of her legs associated with pain in legs, arms, and back of neck. No sulphonamide drugs were given at this time. Her left ankle is stated to have been swollen, and anorexia was accompanied by considerable loss of weight. Clinical examination seems to have been largely negative. "Fibrous" nodules were reported to be present in the trapezius. Blood pressure was 120/80. The temperature varied between 97 and 101.5° F. (36.1 and 38.6° C.) and the pulse rate between 80 and 120, until mid-September, 1946, when both gradually settled to average levels. This was maintained until she was discharged on Nov. 9. The erythrocyte sedimentation rate remained elevated throughout this period—July, 93:115; October, 92:120. The following investigations were carried out: (1) *Blood*:—No evidence of anaemia present. The leucocyte count varied between 5,000 and 11,500 per c.mm. The highest eosinophil reading reported was 2%. (2) *Urine*.—Reaction acid; specific gravity, 1026. Albumin, trace. On microscopic examination 8 to 11 red blood cells were present per high-power field. (3) *Fractional test meal*:—A normal free-acid curve was obtained. A test for occult blood in the faeces was positive. (4) *X-ray examination* of chest, long bones, hands, and feet, barium meal, and barium enema revealed no gross lesions.

After being discharged home on Nov. 9, 1946, the patient remained in moderately good health until three weeks before admission to the Western Infirmary. At this time she developed herpes zoster of her right chest, and after a few days haemoptysis occurred. Recurrent vomiting was complained of for six days before admission to the infirmary.

On admission the patient looked very ill and was markedly emaciated. The percussion note over the right apex was impaired, and crepitant rales were present at the right apex, right mid-zone, and left base. The abdominal reflexes were not elicited; both ankle- and knee-jerks were very active; the plantar responses were flexor. The pulse rate varied between 80 and 120, the temperature between 97 and 98° F. (36.1 and 36.7° C.). The blood pressure was 118/75. The patient is stated to have suffered from "chest trouble" in adolescence. Her mother died of "chest trouble." Her father died of rheumatic fever at the age of 27.

A blood count showed: red cells, 2,100,000 per c.mm.; haemoglobin, 47%; white cells, 8,000 per c.mm. The erythrocyte sedimentation rate was 120:150. No abnormality was detected in the urine. Tubercle bacilli were not found in the sputum. An x-ray report of the chest on Dec. 13 was: "Old calcified primary lesions in both hilar regions and in right lower lobe. Mottled opacities right upper lobe. Appearances are suggestive of active tuberculosis. There is increase in transverse diameter of the heart."

On Dec. 17, four days after admission, the patient died suddenly.

Post-mortem Report.—A necropsy carried out by Dr. George Smith in the pathology department disclosed the presence of typical lesions of periarteritis nodosa in kidneys, liver, lungs, spleen, heart, small intestines, pancreas, adrenals, and uterus. While a few of the lesions were reported to be in the healing phase of fibrosis the great majority showed acute necrotising inflammation of the vessel wall with oedema and polymorph infiltration, in which eosinophils were noteworthy. The vessels chiefly affected were the small arteries and arterioles. With the exception of a healed calcified scar at the apex of the left lung no evidence of pulmonary tuberculosis was detected. Both lungs were denser in patches than normal, but the consistency was considered to be not that of fibrosis or neoplasm,

and was due to haemorrhage. Histological examination did not disclose the presence of tuberculous lesions in the lungs. The immediate cause of death was probably the severe damage to the kidneys. The femoral vein blood urea twelve hours after death was 129 mg. per 100 ml.

Comment on Case 2.—This case was not diagnosed until a post-mortem examination had been performed. The clinical diagnosis appeared to rest between pulmonary neoplasm and pulmonary tuberculosis. The latter possibility was supported by the result of x-ray examination of the chest, to which reference has been made. The interesting points in the case are: (1) The patient was a female suffering from a rare disease in which males are affected four times as often as females. (2) Haemoptysis is not a common symptom of periarteritis nodosa. It occurred in only 1% of cases in a series reviewed by Bruce Logue and Mullins (1946). (3) In Kline and Young's (1934) three cases the difficulty in differentiating periarteritis nodosa affecting the lungs from pulmonary tuberculosis was observed. Bruce Logue and Mullins (1946), referring to this, suggest that areas of fibrosis due to small infarctions may resemble pulmonary tuberculosis radiologically. In this case the findings both on clinical and on x-ray examination were compatible with a diagnosis of pulmonary tuberculosis.

Aetiology

The cause of periarteritis nodosa is not yet definitely known. Transmission of the disease to animals has been reported by Von Haun (1920) and by Harris and Friedrichs (1922). These results do not appear to have been obtained by other workers, although Muir (1940) considers the pathological picture to be compatible with the activity of an unknown infective agent.

Ophüls (1923) and Neale and Whitfield (1934) suggest that periarteritis nodosa is a manifestation of a streptococcal allergic state. Its association with rheumatic heart disease was noted by Friedberg and Cross (1934) in four of their eight cases, and they considered that the disease may result from hypersensitivity to more than one agent. This view has received the support of Anderson (1944). Eight cases of periarteritis nodosa associated with asthma and eosinophilia are reported by Rackemann and Greene (1939), and Rich (1942) records the development of periarteritis nodosa in cases which had shown evidence of hypersensitivity to serum or sulphonamides, or both. In 1943 Rich and Gregory demonstrated that the disease could be produced experimentally in rabbits by sensitizing them to foreign serum-sulphonamide complexes. In 1946 Bruce Logue and Mullins referred to a possible relationship between acute disseminated lupus erythematosus and periarteritis nodosa.

It would therefore seem that allergy plays a very considerable part in the aetiology of periarteritis nodosa, and, further, that a hypersensitive "soil" may be an essential factor in the development of the disease. In this connexion it is of interest to record that the son of Case 1 has been found to be suffering from Besnier's prurigo-eczema-asthma syndrome. Hypersensitivity to drugs of the sulphonamide series may be implicated in certain instances, and the apparently increasing incidence of the disease may be attributable to this fact. Sulphonamide drugs had been administered to Case 1 on several occasions. On the other hand, all inquiries regarding this aspect in Case 2 have been entirely negative. There can be little doubt that the disease existed before the sulphonamide era, and in some instances hypersensitivity to infective agents may be responsible.

Diagnosis

The possibility of periarteritis nodosa should be borne in mind in a case presenting multiple symptoms referable

to several systems. In Case 1 the diagnosis initially seemed to be that of pyrexia of uncertain origin, with emphasis on the possibility of enteric fever and abdominal tuberculosis. With the appearance of cutaneous nodules, periarteritis nodosa, carcinomatosis, and trichiniasis became possibilities; less probable causes in this case being bacterial endocarditis with multiple emboli, and Weber-Christian syndrome (Weber, 1925; Christian, 1928). The combination of myositis, abdominal pain, loss of weight, tachycardia, and fever, associated with cutaneous nodules, suggested periarteritis nodosa, and this was confirmed by histological examination of a nodule.

The triad of myositis, abdominal pain, and loss of weight has been referred to by Meyer and by Middleton and McCarter (1935). In a review by Bruce Logue and Mullins (1946) tachycardia and fever occurred in 87%, abdominal pain in 56%, myositis in 44%, loss of weight in 44%, and cutaneous nodules in 23% of cases.

It is regrettable that no characteristic lesions are observed on ophthalmoscopic examination, but emphasis should be placed on histological examination of cutaneous nodules in suspected cases, or in their absence success may attend muscle biopsy.

Treatment

From a review of the literature no treatment appears to have been of any value. Leishman (1937) confirms this view. Assessment of any particular treatment is difficult because of the infrequency with which the disease is diagnosed during life. Secondly, chronic forms characterized by exacerbations and remissions occur (Arkin, 1930; Macaigue and Nicaud, 1932). Lastly, Boyd (1944) emphasizes the conception of healed periarteritis nodosa with multiple chronic ischaemic lesions.

Penicillin.—As long-continued pyrexia suggested infection in Case 1, penicillin was tried, a total dosage of 4 mega units being given intramuscularly from Dec. 4 to 10. Penicillin in smaller dosage had been administered to Case 2 before admission to the Western Infirmary, Glasgow. No improvement occurred in either case during or immediately subsequent to penicillin therapy. Despite this it is possible that beneficial effects might ensue in cases in which a penicillin-sensitive micro-organism is acting as antigen. On the other hand, according to Suchecki (1946), allergic reaction to penicillin occurs in some 0.5% of cases. In such instances penicillin therapy may indeed be contraindicated.

Anti-histamine Preparations

The drugs used were β -dimethylaminoethyl benzhydryl ether hydrochloride ("benadryl") and histamine azoprotein ("lertigon"). These were employed on the basis of the histamine concept of allergy. Benadryl has histamine antagonizing properties (Loew, Kaiser, and Moore, 1945). It is thought to act by competing with histamine for a given site of action, and thus prevents the effect of histamine combination at this site. On the other hand, lertigon is said to produce antibodies which neutralize histamine before it can enter into combination at the site of action. Benadryl and lertigon are thus synergistic in action, and it would appear rational to use them concurrently.

In Case 1 benadryl was administered initially in doses of 50 mg., four times daily, and its administration coincided with a tendency of the temperature and pulse rate to return to average levels. It would appear, however, that benadryl has not been effective in preventing the retinal and alimentary tract exacerbations (March, 1947). No toxic effects have been observed; but it was noticed that a steady fall in the patient's weight ceased when the dose of benadryl was reduced. No explanation has been found for this

side-effect of benadryl, if indeed the drug was responsible for the loss of weight.

Lertigon therapy was started in May, 1947, being administered subcutaneously in increasing doses every fourth day, up to a maximum single dose of 1 ml. Subsequently the dose has been reduced to 1 ml. weekly. No toxic effects have been observed.

Since beginning this combined therapy no exacerbations of the disease process have occurred. The patient states that he feels well, his weight is stationary, and the erythrocyte sedimentation rate is within average limits. There is some evidence, however, that hypertension may be developing, possibly secondary to ischaemic renal lesions. Whilst no conclusions can be drawn from a single case, it is felt that therapy along similar lines may hold out promise for the future treatment of periarteritis nodosa, and perhaps of other conditions.

Summary

Two cases of periarteritis nodosa are reported. One case was diagnosed during life, and 16 months after admission the patient is in good health. The aetiology of the disease is reviewed. It would appear that periarteritis nodosa is an expression of an allergic state to either micro-organisms or drugs—e.g., sulphonamides.

Observations are made regarding diagnosis. Emphasis is placed on clinical diagnosis, and the importance of histological examination of cutaneous nodules or muscle tissue in any doubtful case is stressed.

The results of treatment with penicillin, benadryl, and lertigon are reported. Encouraging results have followed combined benadryl and lertigon therapy in one case.

I wish to express my thanks to Dr. D. K. Adams for encouragement and advice in writing this paper, and for his permission to publish the cases. My thanks are also due to Prof. D. F. Cappell and Prof. W. J. B. Riddell for their helpful criticisms, and to various colleagues whose assistance I have acknowledged in some instances by name.

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Mr. James Griffiths, Minister of National Insurance, points out in a message he has sent to all members of the staff of his department that 700 of the 1,000 local national-insurance offices ultimately needed are already open and the necessary wide and intimate contacts with the public have already begun. He adds: "We must never forget that for the most part we shall be dealing with people who are ill or in trouble or are perplexed by some point or other. They will need not only the benefits due to them but in many cases help and advice also. There have already been many difficulties and there will be more. We are compelled to go forward in face of shortages of premises, of staff, and of equipment of every kind. Success must depend not only on the initiative and leadership of those in key positions but also on an alert and willing response from all. I have found everywhere the keenest enthusiasm and a high sense of the great social mission with which the Ministry is charged. This is going to be a soldier's battle for us and I am confident that every member of the staff, from the highest to the lowest, will do his best and not spare himself in the critical months that lie ahead of us."

Medical Memoranda

Otitis Media in Infancy

The following brief statistical survey indicates that otitis media in infancy remains a major clinical problem in a large children's hospital. It shows the results of treatment with penicillin and sulphonamides and of the surgical procedures of myringotomy and posterior drainage of the mastoid antrum.

Of 7,806 admissions to the wards of Alder Hey Children's Hospital, Liverpool, during the year 1946, 1,107 were infants under the age of 12 months. This number includes medical, surgical, and orthopaedic conditions, as well as primary aural diseases and cases of feeding error. Of these 1,107 infants 240 had an aural infection on admission; in the remaining 867 the ears appeared normal when first seen but 191 later developed otitis media or mastoiditis—a total of 431 (39%) infants with ear disease. Of these, 268 recovered and 163 (38%) died. The following surgical procedures were carried out:

Bilateral myringotomies in	227 cases (109 died)
Unilateral " "	18 " (8 ")
Posterior drainage also " "	64 " (48 ")

TABLE I.—Relationship of Otitis Media to Parenteral Infections

	Recovered	Died	Total
1. Respiratory:			
Bronchitis or pneumonia with otitis media or mastoiditis	60	43 (42%)	103
Bronchitis or pneumonia without complicating aural disease	168	*48 (22%)	216
2. Alimentary:			
Gastro-enteritis with otitis media or mastoiditis	118	75 (39%)	193
Gastro-enteritis without complicating aural disease	105	45 (30%)	150

* Of these 26 died within 24 hours of admission.

TABLE II.—Results of Treatment of 431 Infants with Aural Disease

Treatment	Recovered	Died	Total
Penicillin alone	32	14 (30%)	46
Chemotherapy alone	105	31 (23%)	136
Both penicillin and chemotherapy (extremely ill cases)	102	104 (50%)	206
No specific treatment (includes both mild infections and those dying within a few hours of admission before treatment had begun)	29	14 (33%)	43
Total	268	163 (38%)	431

These figures are striking in that they reveal the high incidence of otitis media in infants in hospital. Barrier-nursing technique was observed in all cases whether admitted to general wards or individual cubicles. The environment seemed to have no significant bearing on the subsequent development of the disease.

Prolonged clinical observation bears out the extreme difficulty of assessing whether otitis media is primary or secondary in cases in which respiratory or alimentary infections are also present.

CONCLUSIONS

On the question of treatment it is very difficult to draw specific conclusions, one can only generalize. There is no great difference between the results of those treated with myringotomy alone and those having posterior drainage; the mortality rate in both groups is high. Posterior drainage is performed by some surgeons only when symptoms persist after repeated myringotomies, whereas others consider that posterior drainage is indicated if there is no improvement after one adequate paracentesis.

Penicillin appears to have only a small place in the treatment of otitis media in young infants—possibly because of lack of sensitivity of the infecting organism. The mortality rate is high in spite of treatment with sulphonamides, penicillin, and intravenous fluids to combat dehydration.

Thanks are due to Dr. W. E. Crosbie, medical superintendent, Alder Hey Children's Hospital, for permission to publish these figures, to Professor Norman B. Capon for his kind interest and advice, and to Dr. Margaret Rogerson for assistance.

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