all cause facial pain, but there is a bewildering group of patients with episodic or persistent facial pain who have no evidence of bad teeth or chronic sinus infection and no motor or sensory abnormalities to explain their symptoms. Some patients with episodic and sometimes unilateral lower facial pain may have vascular phenomena such as flushing or abnormal heat sensitivity. Occasionally ergotamine gives pain relief.

Other patients complain of a constant dull aching deep-seated pain, often in the lower face. These symptoms may be psychogenic and many, but not all, patients have alterations in mood, attitude, or behaviour. The use of potent antidepressant drugs in high dosage is occasionally successful in the treatment of this variant of atypical facial pain.

Phantom limb pain

Many different types of pain follow injury to nerves. The immediate treatment of painful peripheral nerve lesions with analgesics and sedatives may prevent the later development of

painful phenomena in the limb. Phantom limb pain is one of the most terrible of these, and occurs in about 30% of patients after amputation, being permanent in 5-10%. The most vivid phantom may be associated with the most severe pain. Sympathetic ganglia contribute to this pain in some way, but, although sympathectomy will relieve the constant severe burning pain of causalgia, this operation rarely causes lasting relief of phantom limb pain. If a tender stump is present modification of sensory input by anaesthetic block or intense stimulation occasionally causes pain relief.

The effects of drug treatment are variable, although major tranquillisers (chlorpromazine 50-500 mg daily) may be partially effective. As in the case of postherpetic neuralgia, continuous or intermittent transcutaneous electrical stimulation is occasionally successful in treating this kind of pain, as well as that due to malignant disease with root or nerve involvement. The results of surgery such as chordotomy, thalamotomy, or even cortical ablation, are inconstant and there is probably a widespread central disturbance of pain mechanisms in these patients so that at whatever level pain is attacked it often returns.

Clinical Topics

The forgotten nodule: complications of sacral nodules in rheumatoid arthritis

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Summary

Nodules commonly occur in rheumatoid arthritis and occasionally give rise to complications. The sacral nodule is easily missed and may ulcerate to produce extensive sacral sores which may lead to serious and even fatal complications in patients with rheumatoid arthritis. Seven cases are reported which illustrate some of these features.

Introduction

Most doctors are aware that subcutaneous nodules may be found over the extensor surfaces of the joints and areas of the body prone to minor trauma in patients with rheumatoid arthritis. Rheumatoid nodules over the sacrum, however, are seldom looked for, and yet in our experience these may lead to pressure sores, act as a nidus for infection, and even cause fatal

septicaemia in patients debilitated by severe rheumatoid arthritis.

Recently we have seen seven patients who have suffered serious complications resulting directly from sacral nodules. The main features of the cases are outlined below, and four representative case histories (cases 1-4) are described in more detail.

Case Reports

Case 1—A 67-year-old woman with severe rheumatoid arthritis of 30 years' duration was admitted to the Centre for Rheumatic Diseases in February. Her functional capacity was Steinbrocker grade IV,¹ and she had been bedridden for four years. She was admitted with multiple nutritional deficiencies. On examination she had end-stage, destructive, nodular rheumatoid arthritis with two large ulcers over the sacrum penetrating to bone. These had begun as two small nodules on the sacral area. Haemoglobin was 10.2 g/dl, erythrocyte sedimentation rate (ESR) 92 mm, albumin 2.8 g/l, globulin 3.9 g/l, and rheumatoid factor (R3) titre 1/16. At first the ulcers were treated conservatively, but ultimately they required skin grafting, and the patient was in hospital for just over a year until her ulcers had healed.

Case 2—A 57-year-old woman, who had had rheumatoid arthritis for nine years (functional capacity grade II), was admitted for a right Macintosh knee arthroplasty, and on routine examination she was noted to have a small sacral nodule. Haemoglobin was 10.3 g/l, E.S.R. 29 mm, and rheumatoid factor titre 1/16. The sacral nodule discharged on the ninth day after operation, which resulted in a sacral sore which became infected with proteus. Blood cultures were negative and the sacral ulcer gradually healed with topical antibiotics. Her total inpatient stay as a result of this complication was three months. The sacral sore before skin grafting is shown in the fig.

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Case 2—Development of sacral sore before skin grafting.

Case 3.—A man of 78 with only an eight-month history of rheumatoid arthritis (functional capacity grade IV) was admitted with an unhealed sacral ulcer after surgical excision of a painful sacral nodule. Examination showed a large infected sacral ulcer with pus tracking subcutaneously up his back. A mixture of pseudomonas, klebsiellae and coliforms was cultured from the ulcer but blood cultures were negative. Haemoglobin was 9.4 g/l, white blood count (W.B.C.) $19.2 \times 10^9/l$, E.S.R. 68 mm, and rheumatoid factor titre 1/64. He was treated with prednisolone and indomethacin and then transferred to the plastic surgical unit for débridement of the ulcer, but he died of a terminal bronchopneumonia.

Case 4.—A 59-year-old man who had been confined to a wheelchair for six years with severe nodular rheumatoid arthritis (functional capacity grade IV) was admitted for bilateral knee arthroplasty. Haemoglobin was 11.2 g/l, W.B.C. $9.4 \times 10^9/l$, E.S.R. 97 mm, rheumatoid factor titre 1/2048, antinuclear factor 1/1000, and DNA binding normal. He underwent a left geometric arthroplasty, and in the immediate postoperative period he was noted to have a small discharging sacral nodule. Seven days later he had a swinging fever and a discharge of pus from his surgical wound. He then developed staphylococcal septicaemia with septic arthritis involving the first left metacarpophalangeal joint, the right knee joint, the right elbow, and both shoulders. Despite vigorous antibiotic therapy with ibuprofen, indomethacin, and naproxen the geometric implant became a nidus of infection, which led to recurrent episodes of bacteraemia with septic arthritis. He ultimately succumbed to a fatal pseudomonas septicaemia.

Case 5.—A 65-year-old woman who had had rheumatoid arthritis for 15 years (functional capacity grade IV) was admitted because of exacerbation of her condition. She had numerous nodules over her scapulae and sacrum. Rheumatoid factor titre was 1/1024. The nodules discharged and cultures were positive for pseudomonas. She was treated with phenylbutazone and the sores healed.

Case 6.—A 64-year-old woman with a 20-year history of rheumatoid arthritis (functional capacity grade III) was admitted for geometric knee arthroplasty. Rheumatoid factor titre was 1/2048. She had a sacral nodule, which discharged and was found to be infected with bacteroides and *Escherichia coli*. She was treated with indomethacin and naproxen, but ultimately needed a skin graft to the sacral ulcer.

Case 7.—A 68-year-old woman who had had rheumatoid arthritis for five years (functional capacity grade III) was admitted because of a necrotic discharging sacral nodule. Rheumatoid factor titre was 1/1024. *E. coli* was grown from the necrotic nodule, which healed after prednisolone treatment.

Discussion

Infections can be a serious problem in patients debilitated by rheumatoid arthritis, and they are often recurrent and present in an unusual manner.² Areas of superficial skin ulceration are a particular problem as they often act as portals of entry of staphylococci, which cause staphylococcal septicaemia and septic arthritis.^{3,4} Rheumatoid nodules are prone to break down

and produce superficial skin ulcers which may be overlooked if they occur in sites other than well-known areas such as the extensor surfaces of the elbows.⁵ Sacral nodules are easily missed on routine examination of a patient with rheumatoid arthritis and a small skin ulcer over the sacral region in a patient who spends most of his time in a chair or in bed may not even be noticed by the patient himself. In the light of our experience we wish to draw attention to the potential hazards of sacral nodules, particularly in patients who may be selected for joint surgery during the course of their illness. Cohen⁶ described one case of staphylococcal septicaemia with multiple pyarthrosis in a patient with rheumatoid arthritis who had a small superficial sacral sore which yielded a culture of coagulase-positive staphylococci. Similarly, Kellgren *et al.*,² (1958) in their series of 12 patients with rheumatism and severe infections, reported one patient who died of multiple pyarthrosis and staphylococcal septicaemia after ulceration of several sacral nodules. Two of our patients died from overwhelming staphylococcal infection, while the others had their hospital stay considerably lengthened as a result of the intensive nursing care required to heal their sacral sores. We would emphasize, therefore, the importance of noting sacral nodules when examining a patient with rheumatoid arthritis, especially before surgery. The presence of a sacral ulcer, however small, must be a contraindication to any surgical procedure on the joints until the breach in the skin has healed. The nursing staff should be alerted to reporting the first signs of any skin ulceration in the sacral area, and when sacral nodules exist intensive measures should be taken to minimize pressure effects in a patient confined to bed with arthritis. Only when both medical and nursing staff are alerted to the potential dangers of the "occult" sacral nodule in patients with rheumatoid arthritis may adequate prophylactic measures be taken to prevent an added complication occurring in patients already burdened with many other problems.

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Are young babies at risk when taken to indoor swimming pools, often before having started their polio immunisation programme and certainly before having completed it?

The risk of acquiring polio at a properly maintained indoor swimming pool in Britain is negligible. The transmission of the polio virus by faecal contamination is mainly through personal contact and rarely water-borne. In countries with poor standards of sanitation and personal hygiene, overcrowding, and very large families, nearly all children are exposed to the polio virus before the age of 3 and immunity in the older children is usual. Children in Britain do not acquire this "natural" immunity and only by continuing the present practice of active immunisation of all infants will protection be maintained.

The recommended course for using Herpid (idoxuridine 5%) lasts four days, which means that a large quantity remains out of the 5 ml container. Can this be kept by the patient, or has it to be discarded?

Herpid (5% idoxuridine BP in 100% dimethyl sulphoxide) should not be used for longer than four days as maceration of the skin may develop. The amount used in this time depends on the size of the affected area. It is thought that some of the decomposition products have reduced antiviral activity and are more toxic than idoxuridine and the manufacturers recommend that any remaining solution should be discarded as soon as the course is completed.