papers I should like to make some points about the management of a persistent occipitoposterior position. Firstly, it is important to provide adequate analgesia. A full epidural service is not available in all consultant obstetric units, and in my case a pudendal block provided totally inadequate analgesia for Kielland's forceps rotation. I am surprised that in view of the evidence of the inadequacy of the pudendal block obstetricians still "make do" with it.[1-4] There is a tendency for some obstetricians to think that "a really good pudendal block" can anaesthetise areas not served by the pudendal nerve. The psychological effects of enduring such pain both in the short term—possible rejection of the baby and depression—and in the long term—bitterness and souring of family relationships because of the painful and humiliating memory—seem to be very often ignored. Perhaps this is because obstetricians do not normally deal with these sequelae.

Secondly, it has been suggested that a senior obstetrician should examine the patient[5] and conduct the delivery.[6] If this is to be the case, the use of the forceps must become even more a declining art.

Thirdly, delivery face to pubes with Kielland's forceps seems to carry a high perinatal mortality—6·3% is the only figure I can find.[7] It is also traumatic for the mother. I therefore feel, as it must sometimes be difficult to predict in advance whether the head will rotate easily or not, that all attempted rotations should be carried out in an operating theatre with an anaesthetist standing by in case caesarean section is required. This method of management would require an on the spot anaesthetic service that is not universally available.

Fourthly, I cannot find any up to date information about maternal morbidity after Kielland's forceps delivery. It should be possible to obtain some retrospective information both on immediate damage such as lacerations and haematomas and on the incidence of subsequent stress incontinence and the need for repair of prolapse.

Finally, the need for rotational forceps might be reduced by earlier use of an oxytocin drip.

A multicentre trial in multigravidas of Kielland's forceps versus caesarean section for midcavity arrest should pose little ethical problem. Results of such a study would provide much needed information on perinatal mortality and maternal morbidity on which future trials in primigravidas could be based.

References

1 Scudamore JH, Yates MJ. Pudendal block: a misnomer? *Lancet* 1966; i:23.
2 Hutchins CJ. Spinal analgesia for instrumental delivery: a comparison with pudendal nerve block. *Anaesthesia* 1980;**35**:376-7.
3 Gottschalk W, Hamilton LA. Paracervical-pudendal blocks in obstetrics. *Clin Obstet Gynecol* 1975;**2**:565-77.
4 Percival RC, ed. *Holland and Brews manual of obstetrics.* 14th ed. Edinburgh: Churchill Livingstone, 1980:374.
5 Drife JO. Kielland or Caesar? *Br Med J* 1983;**287**:309-10.
6 Cardozo LD, Gibb DMF, Studd JWW, *et al.* Should we abandon Kielland's forceps? *Br Med J* 1983;**287**:315-7.
7 Myerscough PR, ed. *Munro Kerr's operative obstetrics.* 10th ed. London: Baillière Tindall, 1982:57-8.

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Lesson of the Week

Leprosy masked by steroids

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Leprosy is often assumed to be rare in the Middle East. In 1975 the prevalence in Saudi Arabia, based on 460 registered cases, was 0·1 per thousand population,[1] a prevalence similar to that in Spain and considerably lower than that in Burma, where it is 8·6 per thousand population. We wish to alert physicians to the hazards when the diagnosis of leprosy is overlooked and steroids are administered indiscriminately. We report on a patient with leprosy receiving oral steroids in whom the disease downgraded to lepromatous leprosy before it was diagnosed. The patient presented with an atypical clinical picture.

Injudicious use of systemic steroids in an undiagnosed illness may obscure the diagnosis of any infectious disease, including leprosy

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Case report

A 24 year old Saudi Arabian man had developed a rash and fever 18 months before admission to Charing Cross Hospital. He had been examined in Jeddah and Cairo, where skin biopsy specimens had been taken but no definite diagnosis made. Shortly after the onset of his illness he had developed generalised mild weakness, and eventually treatment with oral corticosteroids was started, which relieved his symptoms. When he presented in London he had been receiving steroids for eight months and was currently taking fluocortolone

20 mg daily. In addition to weakness, which recurred if he did not take his tablets, he complained of numbness of his hands and feet.

On examination he had cushingoid facies, numerous strias on his thighs and flanks, and acne on his back. He had a fine erythema on the trunk, an erythematous nodule on the back of his right hand, and a less conspicuous flat nodule on his right arm. His fingers and, to a lesser extent, his hands were swollen. He had asymmetrical weakness and wasting of the small muscles of his hands, although the wasting was partly masked by swelling. There were areas of diminished sensation on his arms and legs, especially over the ulnar border of each hand. A diagnosis of systemic lupus erythematosus or polyarteritis nodosa was considered, and he was admitted to the department of dermatology at Charing Cross Hospital.

His erythrocyte sedimentation rate was 49 mm in the first hour, haemoglobin concentration 14·9 g/dl, and white cell count $13·8 \times 10^9$/l with 89% polymorphs, 10% lymphocytes, and 1% monocytes. Chest *x* ray films, results of urine analysis and liver function tests, and plasma protein, blood urea, and electrolyte concentrations were all normal. A *Treponema pallidum* haemagglutination test, Venereal Disease Research Laboratory test, and tests for rheumatoid antibody latex and antinuclear factor yielded negative results. *X* ray examinations of his hands showed soft tissue swelling around the phalanges but were otherwise normal.

Studies of nerve conduction confirmed multiple mononeuropathies. Electromyography showed evidence of a primary myopathy. Skin biopsy specimens from an erythematous area on the chest and from the nodule on the right hand showed moderate numbers of intracellular acid fast bacilli in dermal nerve bundles and in an associated macrophage granuloma; these changes are diagnostic of leprosy and consistent with early lepromatous leprosy.[2]

At this stage careful examination of his peripheral nerves showed mild but definite thickening of both superficial radials at the wrist, both ulnars above the elbows, and both lateral popliteals at the neck of the fibulas. The clinical classification of his condition was early subpolar lepromatous leprosy (lepromatous leprosy that has evolved from borderline leprosy).[3,4] The reaction to the lepromin test was negative. Skin smears from the ear lobes and from four other sites were all positive for acid fast bacilli, the bacterial index (concentration of acid fast bacilli expressed on a logarithmic scale)[5] being 3·5, but nose blows were negative for acid fast bacilli. Acid fast bacilli obtained in small numbers from a fresh tissue biopsy specimen of lumbar skin showed classical limited multiplication of *Mycobacterium leprae* when inoculated into the foot pads of mice (Dr R J W Rees). The relatively low bacterial load in the skin, the histological appearances, the small yield of acid fast bacilli from fresh tissue, and the nose blows negative for acid fast bacilli all suggested that his condition had only recently downgraded from borderline to lepromatous leprosy.

Specific treatment was started with dapsone 100 mg daily, clofazimine (Lamprene) 100 mg every second day, and rifampicin 600 mg daily for 18 days and thereafter 600 mg daily on the first two days of each month. Prednisolone was reduced and then stopped. Within six weeks (an unusually short interval in subpolar lepromatous leprosy[4]) he developed a reversal or upgrading reaction[6] with fever (39°C), exacerbation of his erythematous rash, ankle oedema, and mild hepatomegaly (liver function tests yielded abnormal results.) The reaction was controlled with prednisolone 10 mg daily, and specific chemotherapy was continued unchanged.

Comment

This case illustrates two points. Firstly, in countries where the prevalence of leprosy is low the diagnosis may be overlooked. The finding of a persistent unexplained rash or of anaesthesia, with or without muscle wasting, always indicates the need for systematic palpation of the peripheral nerves of predilection in leprosy, especially the superficial radials, ulnars, medians, and lateral popliteals for possible thickening, hardening, and tenderness.

Secondly, the injudicious use of systemic steroids in an undiagnosed illness may obscure the diagnosis of any infectious disease, including leprosy. The characteristic signs and symptoms may be masked and the suppression of immunity induced by steroids may lead to widespread dissemination of the infecting organism, so that an atypical picture may result. Our patient lost specific cell mediated immunity, and his condition downgraded to lepromatous leprosy with an unusual rash before the correct diagnosis was made.

We thank Dr D S Ridley, Hospital for Tropical Diseases, London, for the histological reports and Dr R J W Rees, National Institute for Medical Research, London, for inoculating the patient's strain of *M leprae* into the foot pads of mice.

References

1 World Health Organisation. Leprosy. *WHO Weekly Epidemiological Record* 1979;No 3:17-23.
2 Ridley DS, Jopling WH. Classification of leprosy according to immunity: a five group system. *Int J Lepr* 1966;**34**:255-73.
3 Ridley DS. Histological classification and the immunological spectrum of leprosy. *Bull WHO* 1974;**51**:451-6.
4 Ridley DS, Waters MFR. Significance of variations within the lepromatous group. *Lepr Rev* 1969;**40**:143-52.
5 Ridley DS. Therapeutic trials in leprosy using serial biopsies. *Lepr Rev* 1958;**29**:45-52.
6 Ridley DS. Reactions in leprosy. *Lepr Rev* 1969;**40**:77-81.

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Clinical curio: hypothermia caused by treatment of a scald

The first aid for a minor scald is removal of overlying clothing and dowsing with cold water.[1] We report a case in which this treatment was overused.

A 12 month old boy was admitted to the casualty department in mid-winter one hour after pulling a cup of hot coffee on to his right shoulder and neck. His mother, a 16 year old unmarried woman, was alone in the house and treated the scald by sponging with cold water. She then sought help from a neighbour, who advised that sponging was inadequate, ran a cold bath, and immersed the child in it for 20 minutes. After this he was unresponsive and was having episodes of stiffening and shaking of all four limbs. He was then brought to the casualty department. On arrival he had a rectal temperature of 28°C. He was cyanosed and had a high pitched cry. There was generalised hypertonia with frequent extensor spasms of the limbs. No sign of scalding was seen. Peripheral pulses were thready with a rate of 80/min, blood pressure was 70 mm Hg systolic, and electrocardiographic monitoring showed frequent ventricular extrasystoles. Blood glucose concentration was normal. He was rewarmed gradually in a heated room wrapped in blankets. His temperature returned to normal in six hours, by which time he was fully conscious, alert, and hungry. No further problems were encountered, and he was allowed home the next day.

Hypothermia in childhood usually follows near drowning or exposure.[2] Temperatures below 28°C carry an appreciable mortality, mainly from ventricular fibrillation.[3] In this case a minor scald had been grossly overtreated by someone who had misunderstood the correct procedure and had subjected the child to prolonged cold water immersion. We reviewed the first aid manuals on general sale locally; these recommended either flooding with[4,5] or immersion in[5,6] cold water. None contained any warning regarding hypothermia. Scalds to the trunk in infancy are common, and dowsing of this area is difficult. This case, however, shows that immersion of the child is dangerous and should be avoided.

We thank Dr D Burman for his permission to report this case.—
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1 Lissauer T. *Paediatric emergencies*. London: Update Publications, 1982.
2 Thornton ML, Eng GD, Flynn TG, *et al*. Accidental hypothermia. *Paediatrics* 1979;**63**:926-8.
3 Conn AW. Near drowning and hypothermia. *Can Med Assoc J* 1979;**120**:397-400.
4 Readers Digest. *Readers Digest family medical adviser*. London: Readers Digest, 1983.
5 British Red Cross Society and St John Ambulance Brigade. *First aid manual*. London: Dorling Kindersley, 1982.
6 British Red Cross Society. *Practical first aid*. Wakefield: E P Publishing, 1982.