been treated for an acute episode of anterior uveitis with atropine and prednisolone eye drops. There was no relevant history or family history. He was married with three children and had retired from grocery work three years before presentation. He was a non-smoker. He showed appreciable soft tissue swelling and deformity of his distal interphalangeal joints together with dystrophic, pitted nails. Some of his toes were swollen and sausage-shaped but his toe nails were normal. His sacroiliac joints were tender to percussion and he could get within only 15 cm of his toes on bending forward. His spine was stiff. Elbows, shoulders, knees, and ankle joints appeared unaffected. There were no rheumatoid nodules, gouty tophi, or neurological abnormalities. His right eye showed changes consistent with healing anterior uveitis. His glans penis was ulcerated with patches of keratinisation. There was no other rash. Skeletal x-ray films showed an erosive bilateral distal interphalangeal arthropathy with no osteoporosis and normal joints of his feet, although a calcaneal spur was visible. Both sacroiliac joints were eroded with underlying sclerosis. His sedimentation rate was persistently raised at 40–50 mm in first hour. There was no anaemia, and the results of tests for rheumatoid factor (Rose Waaler); antinuclear factor; antibodies to mitochondria, smooth muscle, parietal cells, and thyroid microsomes and colloid were all negative. His serum uric acid concentration was 0.41 mmol/l.

He was treated conservatively with indomethacin, 25 mg thrice daily, which rapidly improved his joint symptoms. Six months after presentation he could stop taking regular indomethacin, and two years later he noted only occasional joint pains. His finger swelling had disappeared and his nails were now normal. There were no changes in the x-ray appearances of his interphalangeal or sacroiliac joints and no new erosions could be seen. There was no recurrence of his urinary symptoms, genital ulceration, or eye problems.

Tissue typing—The family was tested for all antigens designated at the WHO Nomenclature Meeting after the VII Histocompatibility Workshop. The patient was A2, B27, CW3; his mother A2, B27, CW3/AX(2), BW39; and his three offspring A2, B27, CW3/A1, B7 (see figure). Since his father was

at the D locus. It is intriguing to speculate that the pathogenetic mechanisms in these related diseases were triggered by the interaction of an external factor, such as infection with a common virus, and his immunogenetic background.

We thank Dr Eva Wolf and Janet Pritchard for the HLA serological studies and the patient and his family for their co-operation.

¹ *HLA and Disease*, ed J Dausset and A Svejgaard. Copenhagen, Munksgaard, 1977.

² Brewerton, D A, *et al*, *Lancet*, 1973, **1**, 907.

³ Schlosstein, L, *et al*, *New England Journal of Medicine*, 1973, **288**, 704.

⁴ Robinette, M, *et al*, *Histocompatibility Testing*, p 482. Copenhagen, Munksgaard, 1975

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Family tree of patient showing the relatives studied and their HLA A + B locus antigens determined serologically. The patient is represented by the crossed square.

dead it was not possible to establish definitely that he was homozygous for A2, B27, CW3 on serological typing, although the likelihood of having an unidentified antigen at both the A and B locus is very small. If the three offspring who had inherited the same haplotype from the mother had also inherited the same haplotype from the father, their cells should not have stimulated each other in culture.⁴ Two of the siblings did not reciprocally stimulate each other but were strongly stimulated by the third. This suggested that the latter had inherited a different paternal haplotype from the other two siblings, consistent with the view that the daughter had inherited one A3, B27, CW3 haplotype and her two siblings had inherited the other A3, B27, CW3 haplotype with a different D-locus specificity. This view was confirmed by the observation that the father's cells were *not* homozygous for the D-locus as they stimulated his offsprings' cells. Possibly, however, the positive MLC test result among the siblings could be due to the fact that all the offspring had inherited the same paternal haplotype but that a cross-over between HLA-B and D had occurred in the one offspring, who would then differ for a D locus antigen from the other two. From these results it was concluded that the patient was homozygous for A2, B27, and CW3 and heterozygous at the D locus.

Discussion

This patient presented with several of the syndromes known to be associated with HLA B27 (anterior uveitis, ankylosing spondylitis, psoriatic arthropathy with nail changes and features of Reiter's syndrome (urethritis and a keratinising balanitis)). In addition, he had linear shadows in his chest x-ray film and reduced gas transfer suggesting pulmonary alveolitis. These syndromes appeared over a short time and resolved within a year after symptomatic treatment alone. The patient was HLA A2 B27 homozygous but heterozygous

Mononeuritis multiplex due to haemorrhage

Congenital, acquired, and iatrogenic haemorrhagic disorders may cause bleeding into and around peripheral nerves.¹ This case shows the role of a haemorrhagic diathesis in the development of complications of bony and soft tissue injuries.

Case report

A 57-year-old man was admitted after a road traffic accident, in which he had sustained multiple rib fractures, a fractured right clavicle, and a mild head injury. He had been found to have a prolonged clotting time at the age of 15 after a tonsillectomy. In 1962 he had had a factor XI result assay of 12%, and his last episode of bleeding had been a haematoma in the left thigh in 1965. On admission he was bleeding from the right ear; the facial nerve and other cranial nerves were intact, and x-ray films of the skull, including tomography, showed no fracture. Further x-ray films confirmed fractures of several ribs on the right side with a pneumothorax and a fracture of the right clavicle. Soft tissue bruising was also present behind both knees. On the second day he complained of paraesthesiae in the right arm in the distribution of the C7, C8, and T1 dermatomes and later had detectable sensory alteration over this distribution with sensory loss over C8. On the fourth day he developed a right sided lower motor neurone facial weakness, which became complete over the next 24 hours. The threshold to stimulation of the facial nerve remained the same as that on the unaffected side when tested three, five, and 13 days after the onset of the facial weakness. Electromyography on days 5 and 13 showed no evidence of denervation, and on day 5 a single unit firing in isolation showed that the lesion was incomplete. Facial movements were improving within two weeks and had recovered fully within four weeks. As he was mobilised he developed a bilateral foot drop, due to lateral popliteal nerve lesions. Nerve conduction studies showed evidence of a 50% conduction block at the neck of the fibula. Recovery was complete within three months.

Discussion

After bony and soft tissue injuries this patient developed traumatic facial palsy, evidence of damage to the brachial plexus on the side of the fractured clavicle, and bilateral lateral popliteal nerve lesions at the site of extensive bruising.

Potter and Braakman² considered that the aetiology of delayed traumatic facial palsy might be delayed swelling of the nerve within its perineurium; ischaemia; external compression from progressive oedema within the bony canal; or pressure on the nerve due to an expanding haematoma, which in turn might be intraneural or between

the nerve and bony canal wall. The reduction in the prevalence of delayed facial palsy reported by Briggs and Potter using ACTH tends to support the suggestion that oedema within the canal may play an important part.³ Nevertheless, Fisch found an expanding haematoma in half of explored cases,⁴ and in our patient the known coagulation defect makes it likely that bleeding occurred within the canal. As electrical studies cannot reliably detect denervation in the first 24-72 hours, and since 90% of delayed onset traumatic facial palsies recover completely, an expectant policy with correction of the coagulopathy is to be preferred.

Neuropathies produced by haemorrhage are rare and are usually associated with haemophilia, when femoral nerve lesions are the most frequent.⁵ Our patient developed signs of peripheral nerve damage in three areas. Although he had mechanical trauma to the region of the brachial plexus and the popliteal fossae, the delayed neural damage is more in keeping with haemorrhage around the nerves. The electromyographic evidence suggested that the lesion in both the facial nerve and the lateral popliteal nerves was neuropraxia and the early recovery confirmed this.

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³ Briggs, M, and Potter, J M, *British Medical Journal*, 1971, **3**, 458.

⁴ Fisch, V, *Laryngoscope*, 1974, **84**, 2141.

⁵ Tallroth, A, *Acta Chirurgica Scandinavica*, 1939, **17**, 195.

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Intermittent positive-pressure breathing

There has been some doubt whether the bronchodilator effect of nebulised salbutamol would be enhanced by intermittent positive-pressure breathing (IPPB). We have studied this in a random trial.

Methods and results

Nebulised salbutamol (10 mg) was given by either Wright's nebuliser or IPPB with a Bennett PR2 ventilator to patients admitted to hospital in status asthmaticus. Ten patients (seven men, three women) of mean age 37 years (SD 19) were studied.

On admission patients recorded a self-assessment of breathlessness on a 250 mm line (linear analogue self-assessment scale)¹; the pulse rate was recorded and peak expiratory flow rate (PEFR) measured. A standard corticosteroid and fluid replacement regimen was begun and one of the alternative treatments (randomly selected) was given. Five minutes, three hours, and six hours afterwards the measurements were repeated. The alternative method was then used and measurements made at similar intervals. Thereafter the treatment first used was administered four times daily. On the second and third day the measurements were repeated before and after the midday treatment. Volumes of solution nebulised were adjusted so that the same time was taken by each method. All tests of statistical significance were based on Wilcoxon's Rank Sum test, with the order effect being allowed for in the crossover phase of the trial.

Crossover phase—Six patients received Wright's nebuliser first, and four patients received IPPB first. All patients recorded a considerable improvement in symptom score immediately after the first treatment (table) and thereafter showed little change. There was no significant difference between the results of the treatments. The pulse rate dropped after using the Wright's nebuliser and increased after IPPB. The difference in response to the treatments was statistically significant 5 minutes after treatment ($P < 0.05$). Changes in PEFR showed no statistically significant difference between the two methods.

Second phase—After the crossover patients continued treatment by the method used first. For all patients responses on the second and third days of the trial were similar and have been amalgamated (table). Symptom scores showed a significantly greater increase from the pretrial levels for patients using the Wright's nebuliser ($P < 0.05$). The results from days 2 and 3 again showed a different effect of the two methods on pulse rate. In both groups the pulse rates averaged over days 2 and 3 were lower than the admission rates, but this drop was greater in the patients using the Wright's nebuliser ($P < 0.05$). Only one patient showed major changes in pulse rate immediately after treatment on days 2 and 3: he was in the IPPB group, and the rate rose from 80 to 108/min and from 88 to 104/min, respectively. PEFR changes showed substantial variation among individuals. Although the rise was higher in the group using the Wright's nebuliser, significance at the 5% level was not obtained.

Discussion

Although Webber *et al*² found no difference between the bronchodilator effects of salbutamol given by a Wright's nebuliser and given by IPPB (Bird Mark VII ventilator), they conceded that in more severe asthma IPPB might offer some advantage. The results of our study showed no such advantage for IPPB. After the simple nebuliser the pulse rate fell but after IPPB tachycardia was more pronounced, an undesirable effect in patients with severe asthma. Patients claimed greater improvement with the Wright's nebuliser.

Dolovich *et al*³ have shown that IPPB produced less penetration and peripheral deposition of radiolabelled aerosol particles than did quiet mouth breathing in patients with severe airways obstruction. Cherniack⁴ and Murray,⁵ reviewing the American experience, concluded that the available data failed to show any advantage of IPPB in patients with chronic obstructive pulmonary disease. Our results suggest that the same is true for severe asthma and indicate that expensive IPPB machines are not a necessary part of nebulised bronchodilator treatment.

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² Webber, B A, Shenfield, G M, and Paterson, J W, *American Review of Respiratory Disease*, 1974, **109**, 293.

³ Dolovich, M B, *et al*, *American Review of Respiratory Disease*, 1977, **115**, 397.

⁴ Cherniack, R M, *American Review of Respiratory Disease*, 1974, **110**, suppl, p 188.

⁵ Murray, J F, *American Review of Respiratory Disease*, 1974, **110**, suppl, p 193.

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Symptom score, pulse rate, and PEFR (SE in parentheses)

Index	Order	Day 1—Crossover								Days 2 and 3	
		1st treatment				2nd treatment				Pre-treatment	
		Pre-treatment	5 min	3 h	6 h	Pre-treatment	5 min	3 h	6 h	Pre-treatment	Post-treatment
Symptom score (mm) (higher = better)	IPPB—Wright's	50 (24)	83 (30)	103 (26)	104 (22)	104 (22)	111 (25)	89 (31)	85 (21)	90 (23)	113 (24)
	Wright's—IPPB	53 (16)	146 (16)	150 (17)	153 (18)	153 (18)	146 (16)	150 (17)	153 (18)	196 (23)	215 (17)
Pulse rate/min	IPPB—Wright's	112 (7)	120 (9)	110 (4)	112 (5)	112 (5)	107 (4)	111 (4)	110 (5)	101 (6)	108 (3)
	Wright's—IPPB	118 (5)	112 (4)	107 (6)	93 (8)	93 (8)	112 (4)	107 (6)	93 (8)	91 (3)	90 (2)
PEFR (l/min)	IPPB—Wright's	85 (22)	88 (23)	86 (25)	90 (21)	90 (21)	96 (22)	85 (15)	81 (13)	92 (25)	99 (32)
	Wright's—IPPB	104 (17)	124 (25)	118 (20)	127 (25)	127 (25)	124 (25)	118 (20)	127 (25)	207 (47)	239 (59)