immunisation on the attack rate or severity of the disease cannot be estimated. Sixteen per cent of those immunised received the course of injections after having had whooping cough or in the same year as the attack. It was noted several times by the health visitor that children were immunised as a treatment for whooping cough at the beginning of an attack or in an attempt to shorten a protracted illness. This practice was not unknown, particularly in the first half of this century,¹⁴ but by 1949 it was no longer considered an effective treatment¹⁵; and in 1955 Spock wrote: "Whooping cough vaccine is sometimes given in severe cases but it has not been proved that it does much good."¹⁶

Conclusion

The National Survey of Health and Development is an excellent study in which to investigate the long term consequences of whooping cough contracted at a time when immunisation was not widely available and when medical care for the condition was less effective than it is today. Thus we would have expected the consequences, if present, to have been more pronounced than in a more recent cohort. Despite the unexpected results found for the relation between whooping cough and chronic cough, however, there were in general no long term detrimental consequences of whooping cough as manifested by a reduced peak expiratory flow rate or respiratory symptoms at the age of 36. This study is not suitable for investigating the neurological consequences of whooping cough because of their low prevalence, and for the same reason we could not exclude the possibility of long term respiratory deficit in the few most severely affected cases; nevertheless, from such evidence as was available this hypothesis was unlikely.

We could find no evidence that long term respiratory deficit associated with whooping cough was a factor which could play a part in the continuing controversy over the balance between risks associated with whooping cough and those associated with immunisation against it. The nub of the argument, therefore, still remains

the need to protect young children from the risk of death or neurological damage from whooping cough weighed against the small but measurable risk of neurological illness associated with vaccination.

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SHORT REPORTS

Hand infection with *Mycobacterium chelonae*

I report on four patients with hand infection due to *Mycobacterium chelonae* after receiving steroid injections.

Case reports

A 72 year old woman (case 1) had complained of stiffness of both middle fingers for one year and had received multiple steroid injections to both fingers with only transient relief. Tender fusiform swellings had gradually developed. In October 1983 an incisional biopsy was performed on the left middle finger. Chronic granulomatous tissues with acid fast bacilli were observed. The patient started treatment with streptomycin, isoniazid, rifampicin, and pyrazinamide, but her symptoms persisted, and the swelling and pain in the right middle finger worsened. She was admitted to hospital in December 1983.

On admission there was gross swelling over the proximal and middle phalanges of the right middle finger with extension of induration and erythema proximally to the metacarpophalangeal joint. A discharging wound was present over the radial aspect of the proximal interphalangeal joint. The left middle finger had swelling over the proximal interphalangeal joint only. The right middle finger was debrided, and extensive granulation tissues with necrosis of the extensor and flexor tendons were found. As the finger was beyond saving an open ray amputation was performed one week later. On culture *M chelonae* grew, which was sensitive to kanamycin, amikacin, and doxycycline; partially sensitive to sulphamethoxazole; and resistant to streptomycin, isoniazid, and aminosalicylic acid. The patient started treatment with kanamycin. Four weeks later the wound was sutured, and the patient showed gradual improvement. Kanamycin treatment was stopped in April 1984.

In August 1984 the patient presented again with recurrence of swelling in the

left middle finger over the dorsum of the proximal interphalangeal joint. An excisional biopsy was performed that showed a fleshy granulomatous tissue over the extensor tendon. Culture again yielded *M chelonae*. She was given amikacin and doxycycline. The wound healed, and chemotherapy was stopped after six weeks. On follow up in December 1984 there was no evidence of recurrence, and she could grip fully with both hands.

The table summarises the clinical details of three other patients with *M chelonae* infections. Initially, all patients had symptoms attributable to stenosing tenosynovitis, post-traumatic arthritis, or osteoarthritis. They were treated with steroid injections, and subsequently chronic swelling, pain, and stiffness developed. One patient (case 3) also had infection of the knee. In another patient (case 2) incision and drainage was performed: yellowish pus was drained, but on culture pyogenic organisms did not grow. Owing to the delay in diagnosis and treatment ray amputation was necessary in two patients. Recurrence of infection occurred in two patients requiring further debridement and chemotherapy. Two patients (cases 1 and 4) had received steroid injections from the same general practitioner. A contaminated water supply was subsequently identified in the clinic.

Comment

Although all four cases noted above occurred in Hong Kong, *M chelonae* infection has been reported in England,¹ the Netherlands,² Sri Lanka,³ and the United States.^{4,5} It is an uncommon but potentially serious infection that can cause pulmonary disease, osteomyelitis, disseminated infection, and death. Mycobacteria should be considered when faced with an unusual infection.

M chelonae, unlike most other mycobacteria, grows rapidly in culture (three to five days). The bacilli are pleomorphic, varying from cocci to slender forms and are usually present in scanty amounts. These characteristics contribute to the difficulty in identifying the organism on routine bacterial examination. The bacillus is resistant to most antituberculous

Summary of clinical details of four patients with *M. chelonae* infection

Case No	Sex and age (years)	Initial symptoms	Treatment history	Treatment after admission	Complications	Final hand function
1	F 72	Stiffness of both middle fingers for one year	Multiple steroid injections to both fingers; streptomycin, isoniazid, rifampicin, and pyrazinamide after incisional biopsy	Ray amputation of right middle finger, Hibitane bath, secondary suture four weeks later, kanamycin for four months	Recurrence of infection in left middle finger four months after chemotherapy withdrawn, debridement and chemotherapy given	Right hand satisfactory, good movement, power of grip fair, left hand good
2	F 73	Stiffness and triggering of left middle finger	Multiple steroid injections, incision and drainage	Ray amputation, amikacin for five months, Hibitane bath	Tinnitus, changed to doxycycline and co-trimoxazole for two months, delay of wound healing for six months	Fair, good movement, power of grip weak
3	F 56	Post-traumatic arthritis of left wrist (Colles' fracture), osteoarthritis of right knee	Multiple steroid injections to left wrist and right knee	Kanamycin for six months, synovectomy of right knee	Infected granuloma in left thenar eminence four weeks after chemotherapy withdrawn, treated with complete excision	Good
4	F 62	Stiffness in left middle and little fingers for five months	Multiple steroid injections to both fingers	Synovectomy of both fingers, amikacin for three months		Good, mild triggering in middle finger

drugs¹; drugs that may be useful include erythromycin,⁴ doxycycline, kanamycin, and amikacin. The infection is probably inoculated by contaminated needles. Steroids may perpetuate the infection by suppressing local inflammatory response. As steroid injections are used widely in both hospital and general practice clinicians must be aware that underlying infections may be exacerbated. Disposable syringes should be used whenever possible.

In conclusion, I believe that early surgery (debridement and synovectomy) and appropriate chemotherapy, according to the results of culture and sensitivity testing, are needed for treatment.

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Minimal change nephrotic syndrome presenting after acute decompression

Decompression sickness is a syndrome with protean manifestations, but the nephrotic syndrome has not been reported. We describe a patient who, after decompression, presented with the nephrotic syndrome, which was due to minimal change glomerulonephritis.

Case report

A 19 year old diver had to ascend rapidly from a depth of roughly 30.5 m because he ran out of oxygen during a normal dive 10 days before presenting to us. On surfacing he experienced substernal chest pain, headache, and dizziness and had abdominal cramps and increased bowel motions but no aches in the muscles or joints. The following day he had puffy eyelids and swollen ankles. These symptoms did not improve, and he received recompression treatment (six hours) at a centre for decompression sickness 72 hours after the incident. He defaulted after the first session, believing that he was cured. Six days later he was admitted to our unit with shortness of breath and generalised anasarca. No symptoms of recent upper respiratory tract infection were present, and he had previously been in good health with no history of symptoms suggesting renal disease.

Physical examination showed generalised oedema, more noticeable in the legs than arms, and also ascites and bilateral pleural effusion. Blood pressure was 120/80 mm Hg. Investigations showed a normal haemoglobin concentration and platelet count and white cell count with normal differential. Urine analysis

showed proteinuria but no glycosuria, and microscopy showed normal sediments. Urine protein excretion (24 h) was 18.5 g and endogenous creatinine clearance 125 ml/min. Serum concentrations were: urea 7.8 mmol/l (46.8 mg/100 ml), creatinine 89 µmol/l (1 mg/100 ml), sodium 141 mmol(mEq)/l, potassium 3.5 mmol(mEq)/l, chloride 111 mmol(mEq)/l, bicarbonate 24 mmol(mEq)/l, urate 0.36 mmol/l (6.05 mg/100 ml), calcium 1.44 mmol/l (5.8 mg/100 ml), phosphate 1.47 mmol/l (4.6 mg/100 ml), albumin 11 g/l, globulin 21 g/l, cholesterol 12.5 mmol/l (483.4 mg/100 ml), and triglyceride 2.6 mmol/l (230 mg/100 ml). Immunological variables were: IgG 3.99 g/l, IgA 2.16 g/l, IgM 3.26 g/l, and IgE 2998 IU/ml. A hepatitis B surface antigen test was negative.

A chest x ray film confirmed bilateral pleural effusion, and a film of the abdomen showed normal renal shadows. Percutaneous renal biopsy was performed and the tissue examined under light microscopy, immunofluorescence, and electron microscopy. Light microscopy showed unremarkable glomeruli

Micrographs of glomeruli (above; original magnification ×250), and diffuse foot process fusion of podocytes (below; original magnification ×3600) with no electron dense deposits.