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Do brachial plexus injuries occur at initial impact in motor-cyclists?

Some 77% of traction injuries to the brachial plexus occur in motor-cycle accidents.¹ Although this injury is uncommon, amputation may be necessary when the resulting paralysis is extensive and permanent.^{2,3} The mechanism of injury has been regarded as mutual separation of the neck and shoulder as the motor-cyclist hits the ground, producing a traction injury to the plexus.⁴

We report here two motor-cyclists who collided head-on, with their right-hand handlebars contacting at an impact speed of about 130 kph. Both sustained severe brachial plexus injuries.

The accident and the injuries

The collision occurred on a winding country lane as the two cyclists were cutting the same corner when travelling in opposite directions (fig). Cyclist no 1, who was fully conscious after impact, remained in the saddle and travelled a further 90 metres until he came to rest against a hedge on his left side. Cyclist no 2 was thrown to the ground after impact and was found unconscious with multiple injuries 27 metres from the site of the collision.

Diagram to illustrate the accident, with the mechanism of the brachial plexus injury.

The damage to both motor-cycles was consistent with a head-on handlebar collision.

On admission to hospital cyclist no 1, aged 18, was found to have an upper brachial plexus lesion with complete loss of C5-C8 motor and sensory functions. There was no soft tissue or bony injury to the arm or shoulder. Findings on cervical myelography one week after injury were normal. Neurophysiological studies performed five weeks after injury suggested a widespread postganglionic brachial plexus injury affecting C5-C8 fibres.

On admission to hospital cyclist no 2, aged 18, was found to have extensive injuries to the right arm: fractures of the neck of scapula, mid-shaft of the

humerus, radius, and ulna (compound), and a complete division of the hand in the 4th web space. There was complete loss of C5 to T1 motor and sensory functions. Cervical myelography ten days after injury showed pseudomeningoceles on the right C6, 7, 8 nerve roots, suggesting traumatic avulsion of the nerve roots.⁵ The results of neurophysiological studies six weeks after injury were consistent with a widespread total brachial plexus injury.

Mechanism of injury—In cyclist no 1 the plexus must have been damaged at the moment of collision. The mechanism of injury was a longitudinal force transmitted up the right arm from the handlebar to the shoulder, causing posterior displacement of the right shoulder and thus stretching the nerve roots and trunks close to the intervertebral foramina. This stretching of the nerves would be accentuated by the momentum of the cyclist's chest, head, and neck, which continued to move forward. Cyclist no 2 had injuries consistent with the initial collision—the fractures being caused by a massive force transmitted up the arm from his hand on the handlebar; nevertheless, this mechanism remains unproved since he fell from his cycle.

Discussion

It has not been widely appreciated that in motor-cycle accidents injury to the brachial plexus may occur at the time of initial impact. This might explain why Fletcher¹ noted a "remarkable absence of actual shoulder injury and the clothing has been undamaged in this region." With an indirect force transmitted up the arm to the shoulder girdle no direct injury takes place. If injury occurs at impact a pre-dominance of right arm injuries would be more likely in Britain, and this has in fact been our experience.

In Newcastle we have seen a sudden increase in the incidence of severe brachial plexus injuries—six cases in eight months. Possibly the increased number of motor-cycles on the road is in part responsible, but more likely the new style of crash helmet, with a chin guard (full face), is responsible, partly by preserving life when a patient would have died, and partly by increasing the momentum of the head—as the full face helmet is nearly twice the weight of the old jet type helmet. Sudden rotation of the handlebars may also be partly responsible and this needs further investigation. Should this mechanism be substantiated, it might be possible to incorporate an anti-jack knife mechanism into the front wheel of the cycle, thus protecting the cyclist (at least in one way) from a severely crippling injury.

We thank Mr D D Milne, consultant in charge, accident department, Newcastle General Hospital, for his help and encouragement and Mr Hammersley, medical artist, University of Newcastle, for the figure.

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Ultraviolet light: an effective treatment of osteomalacia in malabsorption

Intramuscular vitamin D has been the mainstay of treatment for osteomalacia in patients with malabsorption, as oral vitamin D is relatively ineffective because of poor absorption of this fat-soluble vitamin. Nevertheless, intramuscular vitamin D BPC has to be given regularly in large doses and may be ineffective because of poor absorption from the injection site.

The more polar metabolites such as 25-hydroxycholecalciferol (25-OHD) or the synthetic analogue 1- α -hydroxycholecalciferol are more effective when given by mouth but are expensive and require extended administration and careful surveillance for hypercalcaemia.^{1,2}

Before vitamin D was discovered nutritional rickets was treated by exposure to sunlight.³ We report the successful treatment of osteomalacia by ultraviolet light in a patient with malabsorption and draw attention to the value of this long-established, safe, and unduly neglected form of treatment.

Case report

A 42-year-old mentally defective man presented in February 1976 with a one-year history of diarrhoea and loss of weight. He was found to have steatorrhoea and a jejunal biopsy specimen showed partial villous atrophy. Although the steatorrhoea improved on a gluten-free diet (fat excretion falling from 83 mmol/24 h to 27 mmol/24 h; normal <18 mmol/24 h), complete recovery was never achieved, partly (at least) because of dietary indiscretions. In May 1977 the patient complained of soreness over the ribs and both tibiae for two months. The serum concentration of calcium was 2.08 mmol/l (8.32 mg/100 ml), phosphate 0.67 mmol/l (2.07 mg/100 ml), and alkaline phosphatase 720 IU/l (normal <92 IU/l). A bone biopsy specimen showed wide osteoid seams and a defective calcification front and the plasma 25-OHD concentration was low at 3.9 ng/ml.

Whole-body treatment with ultraviolet light was begun in May 1977 using four Westinghouse lamps FS40. The patient was treated three times per week, with increasing exposure times. Within one week of starting treatment (total exposure 20 min) his bone pains had been relieved and after two weeks' treatment (50 min) the plasma 25-OHD concentration had risen to 18.3 µg/l. Serum concentrations of calcium had risen to 2.31 mmol/l (9.24 mg/100 ml), phosphate to 1.33 mmol/l (4.12 mg/100 ml), while alkaline phosphatase had fallen to 590 IU/l (figure).

Effects of treatment with ultraviolet light.

Normal concentrations: calcium 2.2-2.6 mmol/l (8-10 mg/100 ml), phosphate 0.8-1.4 mmol/l (2.5-4.3 mg/100 ml); and alkaline phosphatase 30-92 IU/l.

Conversion: SI to traditional units—Serum calcium: 1 mmol/l $\approx$ 2 mg/100 ml. Serum phosphate: 1 mmol/l $\approx$ 3.1 mg/100 ml.

After four weeks' treatment (113 min) the serum calcium concentration was 2.45 mmol/l (9.80 mg/100 ml) and alkaline phosphatase 288 IU/l. Treatment was discontinued after seven weeks (212 min). Eighteen weeks after beginning treatment the plasma 25-OHD concentration was 21.5 µg/l and a bone biopsy specimen showed healing of the osteomalacia.

Comment

Our data show that a short exposure to ultraviolet light (20 minutes) results in a rapid rise in the plasma 25-OHD concentration and quick resolution of symptoms. A similar resolution of symptoms and rapid rise in the plasma 25-OHD concentration has been found in a patient with postgastrectomy osteomalacia after only five minutes' (50 joules) exposure to ultraviolet light.⁴ This suggests that a shorter course of ultraviolet light might have been equally as effective in our patient. An advantage of ultraviolet light treatment is that hypercalcaemia,

which may occur with the more polar metabolites of vitamin D, is not a problem. To prevent further relapses patients should be instructed to go out in the sunlight, but a yearly course of treatment (in winter) may be needed if malabsorption persists.

We thank Miss Lane for giving the ultraviolet light and Dr D E M Lawson for estimating plasma 25-OHD.

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Relative chloroquine resistance of *P falciparum* in Zambia

We have suspected that some strains of *Plasmodium falciparum* in Zambia show resistance to chloroquine, but because the patients are ill we have been unable to prove this according to the WHO criteria.¹ We report a case that apparently fulfils these criteria.

Case report

A 2-year-old child was admitted on 4 May 1977 for treatment of a lower respiratory tract infection. The child came from Lusaka and had not been outside that area. A diagnosis of tuberculosis was entertained but subsequently discarded in favour of that of bronchiectasis. While in the ward she had several episodes of fever, some with convulsions, and malaria parasites were seen in the blood film. On 20 June she was given a course of chloroquine but the blood film remained positive for parasites so testing for resistance was instituted. Between 25 June and 7 July she received 1.3 g of chloroquine (130 mg/kg)—that is, an oral dose of between 5 and 10 mg/kg/day (figure). During the course the Haskins's test in the urine was repeatedly positive, proving that she was absorbing the drug. Despite this parasites were still present in the blood film on 12 July. Subsequently the infestation cleared with a 10-day course of doxycycline, 5 mg/kg/day—that is, 50 mg daily by mouth—to which chloroquine was added. On 24 August parasites were no longer present in the film.

Comment

The dosage of chloroquine between 28 June and 7 July is more than adequate by the WHO criteria and the degree of resistance would

Summary of treatment and findings.