monitor-defibrillator allows a cardiac diagnosis to be made, and if the heart is in ventricular fibrillation it should be defibrillated. Children have been successfully defibrillated at temperatures of 27°C to 30°C, contrary to what has been previously thought.¹ If unsuccessful, further attempts should be made. Laying the patient on a dry blanket protects the operator; arcing does not seem to happen, even with a damp chest. If the heart is in asystole, cardiopulmonary resuscitation should be continued until the victim's temperature is higher than 32°C, the established technique for the management of cardiac arrest being followed.

Victims should never be certified dead until their body temperature and electrocardiogram can be monitored. Resuscitation should continue for however long this takes, for there are recorded cases of full neurological recovery after cardiopulmonary resuscitation for more than two hours.²

Although right heart bypass is an extremely valuable technique in warming the victim, I have seen many who have survived after peritoneal dialysis and continued cardiopulmonary resuscitation, a technique available in every district general hospital.

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2 Orłowski JP. Drowning, near-drowning, and ice-water submersion. *Pediatr Clin North Am* 1987;34:75-92.

Child abuse and osteogenesis imperfecta

SIR,—In reference to the letters of Dr L S Taitz and of Drs Helen Carty and Donald G Shaw (23 January, p 292), I confirm that the 13 patients mentioned in our letter (12 December, p 1561) were all ones in whom there is now no doubt about the diagnosis of osteogenesis imperfecta. We deliberately excluded those, such as those mentioned by Dr Taitz, over whom some doubt may persist. These patients form a worrying group.

I can also reassure Drs Carty and Shaw that the patients we reported on in our recent paper on osteogenesis imperfecta type IVA¹ were collected after patients in whom the diagnosis of osteogenesis imperfecta was doubtful had been excluded, as had been all patients whom we could not, with confidence, allocate to the type IVA variant. Of this group, 64 had been seen personally and 45 had a clearly positive family history. In each case osteogenesis imperfecta had been diagnosed by local paediatricians or orthopaedic surgeons before we carried out our investigations. Skull fractures do occur in this group of patients, as in all other types of osteogenesis imperfecta. In any individual there are of course many more limb fractures than skull fractures, but our figures indicated the proportion of patients who had had at least one skull fracture at any time.

It is not true that osteogenesis imperfecta type IVA is always a mild disease; in our study the mean fracture rate was about twice that of IA disease at all ages. There is even more variation in the severity of the condition than is true of type I disease.

In all the cases I reviewed there were substantial reasons for doubt about the diagnosis of non-accidental injury before I was asked to investigate. Indeed about half were referred not by the parents' solicitors but by the guardians ad litem. The diagnosis in a child with unexplained fractures is seldom easy, and at least four bone disorders—osteogenesis imperfecta, copper deficiency, rickets, and hyperphosphatasia—can cause difficulty. It is incumbent on all of us who are concerned in

these difficult cases to ensure that an incorrect diagnosis of child abuse is not reached and equally that genuine cases of child abuse are identified to protect the patient and any siblings.

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1 Paterson CR, McAllion SJ, Shaw JW. Clinical and radiological features of osteogenesis imperfecta type IVA. *Acta Paediatr Scand* 1987;76:548-52.

**This correspondence is now closed.—Ed, *BMJ*.

Clumsy children

SIR,—I agree with Dr D M B Hall (6 February, p 375) that clumsiness in childhood has many causes but suggest that within the overall group it is possible to identify a specific syndrome of difficulty in learning motor skills (and sometimes cognitive skills as well) for which no structural abnormality in the brain can be shown.^{1,2} At its most severe this syndrome probably represents one end of a spectrum of severity, since minor degrees of difficulty are common when detailed psychological assessment is carried out in school-children.³

I wondered, however, why Dr Hall did not refer specifically to psychometric testing of such children and whether he would agree that the use of the Wechsler intelligence scale for children can be a useful screening test; in most cases where a specific learning defect causes defective acquisition of motor skills it shows a discrepancy between a relatively high verbal IQ and a low performance IQ. I agree with Dr Hall that many of these problems are at least partially overcome in the teen years and that clumsiness of this nature is a talent deficit which, like other learning disabilities, demands specialised and skilled educational requirements for these children.

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1 Walton JN, Ellis E, Court SDM. Clumsy children: a study of developmental apraxia and agnosia. *Brain* 1962;85:603-12.

2 Gubbay SS, Walton JN, Court SDM. Clumsy children: a study of apraxic and agnosic defects in 21 children. *Brain* 1965;88:295.

3 Brenner MW, Gillman S, Zangwill OL, Farrell M. Visuo-motor disability in schoolchildren. *Br Med J* 1967;iv:259-62.

AUTHORS' REPLY.—Sir John suggests that clumsy children represent one end of a spectrum. He does not specify whether he means a spectrum of normal function, as I proposed in my article, or of some as yet uncharacterised pathological process.

Clumsiness may, of course, occasionally be the functional manifestation of several distinct pathological processes. For example, it may follow encephalitis or neonatal hypoglycaemia. Perhaps some localised cerebral malformation might be responsible; such lesions have been associated with dyslexia⁴ (though they do not necessarily cause it). In my experience cases with evidence of pathology are rare, whereas some authors claim that clumsiness affects 3-15% of children. It is difficult to believe that they all have damaged or maldeveloped brains.

Since there is no radiological or biochemical marker for clumsiness the condition can be defined only clinically. Sir John implies that clumsy children can be identified through a verbal-performance discrepancy on IQ testing.

There are undoubtedly children of normal or superior verbal ability who not only are clumsy but

also have difficulties with constructional tasks, attention control, and so on. This apparent clustering, which gave rise to the concept of the "clumsy child syndrome" and the closely related idea of "minimal brain dysfunction," has been shown to be largely artefactual.² Each of the many subskills which make up psychological functioning can vary independently and is to some extent under separate genetic control.³ The unfortunate child who has a statistically improbable mix of talents and deficits is not well tolerated by parents, peers, or educationists. If he is referred for medical evaluation he is likely to be diagnosed as a "typical" clumsy child. Clumsiness is a pejorative term and is best avoided altogether, but the concept may have some value if it is applied specifically to motor disabilities. Any associated problems should be assessed and described on their merits.

Some children are undoubtedly helped by recognition that they have real difficulties with particular tasks, but there are also dangers in the medicalisation of normal human variation: "An entire generation has been conditioned to regard its deviation from the narrowing standards of approved norms as sickness."⁴

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1 Galaburda AM, Kempner TL. Cytoarchitectonic abnormalities in development dyslexia: a case study. *Ann Neurol* 1978;6:94-100.

2 Schmidt MH, Esser G, Allehoff W, Geisel B, Laucht M, Woerner W. Evaluating the significance of minimal brain dysfunction—an epidemiological study. *J Child Psychol Psychiatry* 1983;28:803-22.

3 Ludlow CL, Cooper JA, eds. *Genetic aspects of speech and language disorders*. New York: Academic Press, 1983.

4 Illich I. *Limits to medicine: medical nemesis, the expropriation of health*. Harmondsworth: Pelican, 1977:102.

Treatment of prostatic cancer

SIR,—Dr Jonathan Waxman (30 January, p 356) discusses the relative merits of subcapsular orchidectomy and gonadotrophin releasing hormone analogues for treating prostatic cancer.

In Bristol we have had extensive experience over the past three years with depot gonadotrophin hormone releasing hormone analogues. Undoubtedly when we discuss the treatment options most men will select a monthly (or three monthly) depot injection in preference to orchidectomy. Some men, however, do prefer the "once and for all" surgical option. Perhaps more men would choose it if we explained to them that while both provide effective treatment they both have the same physiological effects—impotence, flushes, and reduction in size of scrotal contents. The apparent advantages of avoiding a "mutilating" surgical procedure may well be outweighed for some by the need for regular outpatient attendance and therefore a monthly (or three monthly) reminder of their condition. This has been particularly noticeable in our unit, where patients are reviewed in a specific prostate cancer clinic. Some patients find the obvious deterioration of others, who may have been attending the same clinic on the same day for several months, disturbing.

There is no evidence that men with prostatic cancer necessarily view orchidectomy as mutilating, and we know of no studies into the psychological effects of this procedure in this group of patients. Recent studies in patients with breast cancer have actually shown no difference in psychological morbidity between those undergoing mutilating surgery (mastectomy) and those undergoing conservative surgery.¹ Unless similar studies of attitudes to orchidectomy are undertaken it cannot