is made to help him not to repeat the offence, perhaps with another sibling, when he is released.

But what of the victim? Horrifying though the results of sexual assault by a stranger may be, at least the child should retain the support of the family. When the father is the assailant her story is likely to be disregarded as fantasy[4 7]; and even if she is believed efforts to help may totally disrupt her environment. She has first to face a physical examination, often frightening, in the unpleasant surroundings of a police station. The removal of father may break up the family—even to the extent of the children being taken into care—and mother and siblings are then likely to blame the victim. Unhappily, the opposite solution—a conspiracy of silence—may not help the child either. In cases of incest efforts to help without the authority of the law to back them up are likely to be ineffectual, and the offences often continue.[2 8]

Here in Britain we are only just beginning to realise the extent of the hidden problem of child sexual abuse, though for a few years now we have accepted the high prevalence of physical abuse. In the United States sexual abuse has been discussed openly for far longer, and in some parts of that country police and social services are cooperating well. Instead of breaking up the family by sending the assailant to prison they arrange deferment of prosecution or sentencing, or a probation order may be made so that help may be given to offenders, victims, and their families.[9] Several schemes have been set up to offer treatment, which usually consists of a mixture of individual, marital, family, and group therapy.[10]

Is treatment needed, and is it effective? The long term effects of sexual abuse on a child are very difficult to assess. Most studies have been retrospective.[11] Reported effects range from none[12] to drug abuse, behavioural, interpersonal, and psychological symptoms of all kinds, even psychosis, various types of sexual dysfunction,[11 13 14] and both adult and child prostitution.[15 16]

An attempt at treatment certainly seems justified, but the outcome also needs careful assessment. Good results have been reported from a very intensive programme in California, where police and other agencies combine to offer immediate help as soon as cases are reported.[17] Treatment is then offered to offenders, victims, and their families, usually at first on an individual basis but later using marital, family, and group therapy. Much use is made of self help groups, some of whose members carry on to support new families joining the programme. The public has come to trust the scheme so that families are now even seeking help voluntarily. The children can usually return home—92% of girls after 90 days in one sample—and most of the family relationships improve substantially with treatment.

A few groups are trying to offer this sort of help in Britain.[2] Unhappily, however, most treatment is still fragmentary, and, if they do uncover cases of abuse, few professionals have any means of referring offenders, victims, or their families for expert help. More coordinated effort is urgently needed.

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1 Mrazek PJ, Lynch MA, Bentovim A. Sexual abuse of children in the United Kingdom. *Child Abuse and Neglect* 1983;7:147-54.
2 Furniss T, Bingley-Miller L, Bentovim A. Therapeutic approach to sexual abuse. *Arch Dis Child* 1984;59:865-70.
3 Finkelhar D. Risk factors in the sexual victimisation of children. *Child Abuse and Neglect* 1980;4:265-73.
4 Anderson D. Touching, when is it caring and nurturing or when is it exploitive and damaging. *Child Abuse and Neglect* 1979;3:793-4.
5 Manchester AH. The law of incest in England and Wales. *Child Abuse and Neglect* 1979;3:679-82.
6 Kempe RS, Kempe CH. *Child abuse*. London: Fontana Open Books, 1978.
7 Summit RH. The child sexual abuse accommodation syndrome. *Child Abuse and Neglect* 1983;7:177-91.
8 Furniss T. Mutual influence and interlocking professional-family process in the treatment of child sexual abuse and incest. *Child Abuse and Neglect* 1983;7:207-14.
9 Fraser BG. Sexual child abuse: the legislation and the law in the United States. In: Mrazek PB, Kempe CH, eds. *Sexually abused children and their families*. Oxford: Pergamon Press, 1981:55-73.
10 Forseth LB, Brown A. A survey of intrafamilial sexual abuse treatment centers. Implications for intervention. *Child Abuse and Neglect* 1981;5:177-86.
11 Mrazek PB, Mrazek DA. The effects of child sexual abuse. In: Mrazek PB, Kempe CH, eds. *Sexually abused children and their families*. Oxford: Pergamon Press, 1981:235-45.
12 Lukianowicz N. Incest I. Paternal incest II. Other types of incest. *Br J Psychiatry* 1972;120:301-13.
13 Bender L, Grugett AE. A follow-up report on children who had atypical sexual experience. *Am J Orthopsychiatry* 1952;22:825-37.
14 Burton L. *Vulnerable children*. London: Routledge and Kegan Paul, 1968.
15 James J, Meyerdling J. Early sexual experience and prostitution. *Am J Psychiatry* 1977;134:1381-5.
16 Sereny G. Lost innocence of the rejected children. *The Times* 1984 Sept 21:9(cols 2-4).
17 Giarretto H. A comprehensive child abuse treatment program. In: Mrazek PB, Kempe CH, eds. *Sexually abused children and their families*. Oxford: Pergamon Press, 1981:179-97.

Sleep helps healing

"Surely," wrote Minerva, "sound, prolonged sleep is essential for optimum healing?[1] The answer must be yes. Bodily tissues are continuously degraded and continuously renewed. Wounds heal through the same processes as make possible the normal renewal, by cell division and protein synthesis, and these do appear to be aided by rest and sleep.

Across the 24 hours there is normally a balance between catabolism (degradation) and anabolism (renewal): the activities of wakefulness enhance catabolism, while sleep shifts the balance in favour of anabolism. Infection, surgical stress, or trauma increases the activity of the sympathetic nervous system and increases secretion of catabolic hormones such as cortisol, glucagon, and catecholamines, while inhibiting anabolic hormones such as insulin and testosterone, so leading to the loss of body nitrogen, indicative of a net loss of protein.[2] That breakdown of muscle throughout the body is enhanced is confirmed by a large, delayed increase in the excretion of 3-methylhistidine.[3 4]

It is not just that cortisol concentrations are low during most of a normal night: sleep positively inhibits the secretion of cortisol,[5] and appears to do the same for that of catecholamines.[6 7] Moreover, deep sleep is the normal stimulus for the release of most of our growth hormone, an anabolic hormone that increases the synthesis of protein and mobilises free fatty acids to provide energy, thereby saving amino acids from catabolism.[8] Growth hormone acts, for example, directly to enhance the synthesis of bone,[9] and, with haemopoietin, to enhance the formation of red blood cells.[10] Given postoperatively, growth hormone improves nitrogen balance,[11] though, unlike sleep, giving it artificially cannot diminish the release of the counteracting catabolic hormones.[11]

In a wide range of animal tissues cell division and protein synthesis reach their maximum values during the hours of sleep and are minimal during wakefulness.[12] When tissues have been damaged, the rate of healing is greater during sleep, whatever the time of the injury.[13-15] Adrenaline, released through wakeful stress, prevents the cell division that is necessary for healing.[16 17]

In man sleep deprivation leads to loss of body nitrogen,[18 19] and in recent carefully controlled experiments Rechtschaffen and his colleagues in Chicago have shown that in rats (which have a high rate of metabolism) sleep deprivation leads to death through widely distributed deterioration of bodily tissue; this happens "in spite of their increased food intake and suggests an increased ratio of catabolism to anabolism."[20] Modern hypnotic drugs prevent

sleepless nights, and the evidence indicates that these drugs would therefore help the restoration of the tissues during sleep.[21]

At the United States Army Institute for Surgical Research Wilmore and his colleagues, having reviewed the mechanisms and effects of surgical stress, concluded that adequate rest periods for uninterrupted sleep should be provided and the duration of sleep always recorded.[22] It remains an obligation on all who work in hospitals to reduce noise, to relieve patients' anxieties, and help them to sleep.

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1 Minerva. Views. *Br Med J* 1984;**288**:1012.
2 Brandt R, Fernandes A, Mordhorst R, Kehlet H. Epidural analgesia improves postoperative nitrogen balance. *Br Med J* 1978;i:1106-8.
3 Elia M, Carter A, Bacon S, Winearls CG, Smith R. Clinical usefulness of urinary 3-methylhistidine excretion in indicating muscle protein breakdown. *Br Med J* 1981;**282**:351-4.
4 Long CL, Birkhahn RH, Geiger JW, Betts JE, Schiller WR, Blakemore W. Urinary excretion of 3-methylhistidine: an assessment of muscle protein catabolism in adult normal subjects and during malnutrition, sepsis and skeletal trauma. *Metabolism* 1981;**30**:765-76.
5 Weitzman ED, Zimmerman JC, Czeisler CA, Ronda J. Cortisol secretion is inhibited during sleep in normal man. *J Clin Endocrinol Metab* 1983;**56**:352-8.
6 Townshend MM, Smith AJ. Factors influencing the urinary excretion of free catecholamines in man. *Clin Sci* 1973;**44**:253-65.
7 Åkerstedt T, Fröberg JE. Sleep and stressor exposure in relation to circadian rhythms in catecholamine excretion. *Biol Psychol* 1979;**8**:69-80.
8 Sassin JF, Parker DC, Mace JW, Gotlin RW, Johnson LC, Rossman LG. Human growth hormone release: relation to slow-wave sleep and sleep-waking cycles. *Science* 1969;**165**:513-5.
9 Isaksson OGP, Jansson J-O, Gause IAM. Growth hormone stimulates longitudinal bone growth directly. *Science* 1982;**216**:1237-9.
10 Golde DW, Bersch N, Li CH. Growth hormone: species-specific stimulation of erythropoiesis in vitro. *Science* 1976;**196**:1112-3.
11 Wilmore DW, Moylan JA, Bristow BF, Mason AD, Pruitt BA. Anabolic effects of human growth hormone and high calorie feedings following thermal injury. *Surg Gynecol Obstet* 1974;**138**:875-84.
12 Adam K, Oswald I. Protein synthesis, bodily renewal and the sleep-wake cycle. *Clin Sci* 1983;**65**:561-7.
13 Gololobova MT. 24-hour rhythm of cell multiplication in rat epidermis during healing of skin wounds. *Biull Eksp Biol Med* 1960;**50**:118-22.
14 Saetren H. Diurnal variation of the first mitotic wave released among rat kidney tubule cells by partial nephrectomy. *Acta Pathol Microbiol Scand [A]* 1972;**80**:737-42.
15 Simmons DJ, Cohen M. Postfracture linear bone growth in rats: a diurnal rhythm. *Clin Orthop* 1980;**149**:240-8.
16 Bullough WS, Laurence EB. Accelerating and decelerating actions of adrenalin in epidermal mitotic activity. *Nature* 1966;**210**:715-6.
17 Fisker AV, Østergaard E, Karring T. The effect of environmental stress stimuli on cell division in rat palatal epithelium. *Cell Tissue Kinetics* 1982;**15**:661-6.
18 Scrimshaw NS, Habicht JP, Pellet P, Piche ML, Cholakos B. Effects of sleep deprivation and reversal of diurnal activity on protein metabolism of young men. *Am J Clin Nutr* 1966;**19**:313-9.
19 Kant GJ, Genser SG, Thorne DR, Pfalser JL, Mougey EH. Effects of 72 hour sleep deprivation on urinary cortisol and indices of metabolism. *Sleep* 1984;**7**:142-6.
20 Rechtschaffen A, Gilliland MA, Bergmann BM, Winter JB. Physiological correlates of prolonged sleep deprivation in rats. *Science* 1982;**221**:182-4.
21 Adam K. Do drugs alter the restorative value of sleep? In: Passouant P, Oswald I, eds. *Pharmacology of the states of alertness*. Oxford: Pergamon Press, 1979:105-11.
22 Wilmore DW, Long JM, Mason AD, Pruitt BA. Stress in surgical patients as a neurophysiological reflex response. *Surg Gynecol Obstet* 1976;**142**:257-69.

MPTP parkinsonism

Our understanding of Parkinson's disease has progressed very substantially since its first description in 1817. The latest advance is the discovery that low doses of MPTP (1-methyl-4-phenyl-1,2,3,6-tetrahydropyridine) selectively destroy pigmented dopaminergic neurones in both man and monkeys and so may mimic the physical and biochemical signs of Parkinson's disease, including its response to levodopa.

In 1976 a 23 year old American addict manufacturing his own pethidine analogue took a synthetic shortcut and injected himself daily with what was later with his help proved to be two closely related byproducts; one was MPTP.[1] On the third day he developed a pure and severe parkinsonian syndrome which responded dramatically to levodopa but persisted (with some spontaneous improvement) until his suicide 18 months later. Results of biochemical studies on his cerebrospinal fluid were consistent with severe disruption of dopamine metabolism, and his brain showed destruction solely within the substantia nigra.

Last year a cluster of identical cases was reported from California of patients who had injected the same substance sold to them as "synthetic heroin."[2] These patients, also young and severely affected, showed no spontaneous improvement. Their symptoms responded well to levodopa, but some soon developed a fluctuating response and hallucinations. That these complications developed so early supports the view that when seen in idiopathic disease they are due to the severity of the disease rather than to any long term toxicity of levodopa.[3] About 100 other persons exposed at around the same dosage were clinically unaffected, perhaps indicating some individual susceptibility factor. Their follow up may answer the question whether Parkinson's disease is caused by a persistently active agent or whether a fixed insult may become clinically manifest later by superimposed aging.[4] The drug abusing community was informed with commendable speed, bringing this epidemic to an end. Subsequently one further probable case has occurred in a 37 year old chemist exposed while doing laboratory work—a warning to others handling this compound.[5]

As soon as the episode became known attempts were made to reproduce the syndrome in animals. This proved easy: low doses of MPTP given to primates produced striking clinical, pathological, and biochemical changes.[6,7] Other species seem resistant, however—though with much higher doses—biochemical evidence of dopaminergic dysfunction occurs in mice.[8] Pathological studies in primates show fewer abnormalities outside the substantia nigra than is usual in Parkinson's disease and Lewy inclusion bodies, long held to be fundamental to idiopathic parkinsonism, are absent. These abnormalities, however, may yet prove to develop in animals kept alive longer. The only human brain examined showed one possible inclusion body.[1] Dopamine cells elsewhere in the brain are not affected.

Analysis of the mechanism of action of the toxin should provide further insights. Already, selegiline, a monoamine oxidase inhibitor concerned with dopamine breakdown, has been found to prevent toxicity, possibly indicating that an oxidation metabolite produces the damage.[8,8a] Another interesting question is whether nicotine reduces toxicity, for smoking is suspected of reducing the incidence of Parkinson's disease. The reason pigmented dopamine cells are susceptible may be linked with the high affinity of MPTP for melanin.[9] Also their high concentration of metal ion might stimulate free radical production during the oxidation of MPTP and dopamine[10,11]; and relatively poor inherent or acquired antioxidant capabilities—for instance, the amount of available glutathione peroxidase—then exaggerate these effects. An inability to cope with unpleasant oxygen species has been advanced as a factor in parkinsonism[12] and in the action of other dopamine cell toxins such as manganese and 6-hydroxydopamine.[13,14] This hypothesis is worth pursuing, as the generation of these products is potentially containable by antioxidants.

The strong evidence against any important genetic element in the aetiology of parkinsonism argues an environmental cause. Postencephalitic Parkinson's disease is so different from the idiopathic form that a toxin is at least as attractive a proposition as another virus. Such a toxin would be ubiquitous, given the lack of any known geographical or occupational factor, and might be modern. Other culprits