Chief Inspector of Factories stressed the association in 1947[19] and R. S. Doll and his colleagues have published follow-up and cohort investigations[20 21] which suggest that in at least one factory the attack rates from bronchogenic cancer in men who entered the industry after 1933 are not higher than would be expected in the general population. But in the United States E. C. Hammond and his colleagues[22] reported 53 deaths from lung cancer in 300 consecutive necropsies of insulating workers, and found the death rates from pulmonary and pleural cancers seven times higher in asbestos workers than in American white males.

Since J. C. Wagner and his colleagues[23] first described the occurrence of pleural mesotheliomata in the crocidolite mining districts of South Africa many cases have come to light elsewhere. In Britain most cases have been discovered in London[24] and at the larger ports where much asbestos is handled in the shipbuilding industry.[12 25 26] Diffuse peritoneal tumours associated with asbestos exposure have been reported.[27] There seems to be little doubt that these tumours—which as recently as 1960 were regarded as "rare curiosities"[28]—are of occupational or environmental origin. Wagner[29 30] produced pleural neoplasms in animal experiments, and his findings have now been confirmed by other investigators.[31] The important question of whether only crocidolite or other types of fibres also, especially the widely used chrysolite, have carcinogenic properties has not yet been unequivocally answered.[14]

Two aspects of the asbestos–mesothelioma association are very disturbing. In some cases extremely short exposures have been reported.[12 24] There is also growing evidence that residents in asbestos mining or manufacturing districts and those at risk by the brushing and washing of contaminated working clothes can develop mesenchymal tumours or the bizarre pleural fibrosis and calcifications which seem to be associated with exposure to asbestos.[32 33]

The scope of the problems yet to be solved has been clearly set out by J. C. Gilson in his summing up of a symposium held in New York[34] and in the report of the subcommittee of the International Union against Cancer.[35] The most important of these problems are: the type of asbestos as a factor in the risk of asbestosis and malignancy; the excess risk of malignancy from asbestos in the absence of clinical, physiological, and radiological manifestations of asbestosis; the organization of prospective surveys to establish the risk to industrial and residential populations; agreement on the clinical, lung function, and the notoriously difficult radiological criteria for diagnosing asbestosis; agreement on the most accurate type of dust measurements; and the clinical significance of pleural plaques and of asbestos bodies.

In the meantime it is to be hoped that the promised revision of the 1931 Asbestos Regulations[36] will soon be put into effect and that they will cover all workers at risk, including those engaged in loading and transporting asbestos. Present evidence suggests that crocidolite is the most dangerous of asbestos fibres and it may be possible to find suitable substitutes for at least some of its uses. The uncontrolled dumping of asbestos waste[37] should not occur again. Finally, in view of the long induction period of asbestosis and malignancy the present restriction, that a worker claiming disability benefits for mesothelioma under the Industrial Injuries Act must have been employed after 4 July 1948,[38] seems to be entirely unrealistic and should be abolished.

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Status Epilepticus: A Medical Emergency

Despite modern treatment status epilepticus remains a dangerous condition carrying a high mortality. Its consequences remain largely uncharted because in most instances reports of treatment end with the control of the status or death. But, since there is evidence that the longer the status continues the greater the mortality, early treatment is important and status should be regarded as a medical emergency.

Status epilepticus is defined as the occurrence of grand mal convulsions without recovery of consciousness between fits. The recognition of fully developed status is easy enough: not only have fits been multiple and usually frequent, with persisting unconsciousness, but other signs of physiological stress—in the heart, lungs, kidneys, and in fluid and electrolyte balance—may have developed. A practical problem is to decide at what stage the patient needs transfer to hospital and to the sometimes very specialized management that may be required. Many patients in early status respond rapidly to a simple increase in the routine anticonvulsants they are already having. Moreover, when status supervenes in an established case of epilepsy there is often adequate warning in the form of increased frequency of fits, up to several a day, but with recovery of consciousness between—so-called serial epilepsy. In these circumstances a trial may be justi-

fied for a few hours—but no more—of the sort of measures that can be undertaken at home (paraldehyde intramuscularly in adequate dosage being a stand-by). But if status continues then admission to hospital should be arranged at once. When status occurs as the very first manifestation of epilepsy, as is not very uncommon when the causative focus is in the frontal region, the likelihood of an intractable form is much greater, and admission to hospital should certainly be arranged.

In established status the problem of management is threefold: to control the fits, to ensure an adequate airway and oxygenation, and to maintain the biochemical *milieu intérieur* as normal as possible. Monitoring of fluid balance, electrolytes, blood urea, blood pressure, and pulse will be required, and a simple airway may be needed. If status is long continued then tracheostomy with full curarization and assisted respiration may be necessary; in that case monitoring of blood P_{CO_2} and P_{O_2} will also be called for. In some cases these measures are life-saving, and this makes the condition a matter for urgent admission to hospital.

The linchpin of treatment must remain the control of the fits, and the sooner this is achieved the less need there will be for other measures. If a substantial increase in routine anticonvulsants—given by injection rather than by mouth—does not abolish fits and allow return of consciousness within an hour or two, additional drugs must be used. The remedy most widely advocated is paraldehyde given intramuscularly or intravenously in glucose saline or plasma drip. The wide margin between anticonvulsant and serious toxic doses makes this a drug of choice. Other preparations—soluble barbiturates, soluble phenytoin, and inhalation anaesthetics—have been successfully used, but have not in general displaced paraldehyde. Recently, however, a new drug diazepam (Valium), introduced initially as a muscle relaxant and tranquillizer, has been found to give benefit in status. First reports on it came from French neurologists.[1,2] Now Drs. M. J. Parsonage and J. W. Norris in this issue of the *B.M.J.* (page 85) give their experience of its use in nine cases of severe status. They gave it intravenously and in seven patients attained immediate control of seizures. The effect was rapid and obvious both on clinical seizures and on electroencephalographic discharges. Although in seven cases other methods of treatment were also used—including paraldehyde in four—there was no doubt that diazepam was a valuable and at times decisive therapeutic agent. Its rapidity of action and its absence of serious toxic effects must recommend it as one first choice in the treatment of persisting status. Initially it should be given intravenously, and Parsonage and Norris advise a saline drip (100 mg. per 500 ml.), though 10-mg. intravenous doses repeated may be given. Control may later be maintained by intramuscular injection. Once control is established a gradual return to the usual oral remedies should be undertaken. There is not yet any clear evidence that the drug is effective by mouth on its own, though it appears at times to be an effective adjuvant to others.

The addition of diazepam to the treatment of status does not reduce the seriousness of this condition. Parsonage and Norris's series contains two deaths, one patient with persisting severe dementia and one with residual hemiparesis. This morbidity is important and has been little considered heretofore. There is growing evidence that mental and neurological deficit may follow status. This reinforces the argument for rapid control of the condition, for careful attention to the biochemical milieu while it lasts, and for early and if possible preventive treatment.

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Safer Dental Anaesthesia

In July 1965 the Standing Medical and Dental Advisory Committees of the Central Health Services Council set up a Joint Subcommittee at the request of the Minister of Health with the following terms of reference:

> To consider the use of general anaesthetics in general dental practice and to advise:
>
> (1) How far the administration of general anaesthetics for conservative treatment can be justified, and
>
> (2) How far the administration of general anaesthetics for any purpose without the attendance of a second practitioner can be justified.

This report has now been published[1] and the members of the Joint Subcommittee must be congratulated on producing a document which not only will influence the future of dental anaesthesia but will also serve as a model of the way to present authoritative and exact information in a concise form.

The report classifies the main groups of patients whose clinical condition may justify general anaesthesia for conservative work in general dental practice as follows: (*a*) spastics or others who because of physical infirmity would be uncontrollable under local analgesia; (*b*) patients who are known to react adversely or to be resistant to local analgesics; (*c*) mentally subnormal patients; (*d*) those whose psychological attitude to dentistry is such that they would refuse from fear any form of dental treatment not performed under anaesthesia and who, in the opinion of the dental surgeon, are unsuitable for local analgesia. It can be inferred from the report that the Joint Subcommittee thought some dental practitioners were making too little use of local analgesia for conservative dentistry, for the introduction of lignocaine as an analgesic agent nearly twenty years ago has made the employment of local analgesia reliable and certain. For many years the average Briton had an ingrained fear of the dental surgeon and did not consult him until he could request him to take all his teeth out "with gas but not cocaine." Little wonder then that he prefers that such conservative dentistry as he will permit should be performed while he is asleep. In other countries, such as the U.S.A. and Scandinavia, where local analgesia has been the routine form of pain relief for all types of dental surgery, the use of general anaesthesia for conservative dentistry has not been so enthusiastically championed as in Great Britain.

The report is careful to point out that the relatively low mortality connected with dental anaesthesia in the past is related to the short time for which it is administered. However, if longer operations become more frequent, the mortality and morbidity must inevitably rise, especially if practitioners who are not properly trained administer general anaesthetics for them. The report states that the "administration of general anaesthesia and the performance of dental surgery

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