Experience shows that a systematic approach to safety is possible. Deming teaches that when any variable is outside an accepted norm the system is at fault; blaming employees for deviations is unhelpful.[2] The system should acknowledge the constraints on it and work to accommodate them. Statistical analysis of accidents enables realistic targets to be established and continuing improvement to take place. This provides a framework for transforming an unacceptable system and then leads to the ability to monitor for early signs of deterioration.

Alcohol, poor physical fitness, and adverse social conditions will play important parts in many accidents.[3] Doctors can have an important role in these aspects, as well as acting as a "conscience" for their companies. It is also our responsibility to bring health problems to society's attention. The argument for safe work could be seriously diluted if it appears as a plea for more jobs for doctors.

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Asbestos diseases and compensation

SIR,—Professor Anthony Seaton asserted that the diagnosis of mesothelioma is not always straightforward and that recommended reporting and inquest procedures are not always being observed.[1]

Our study in Leeds supports these assertions.[2] A diagnosis of mesothelioma during life may not lead to it being recorded on the death certificate.[3] Even when mesothelioma is recorded not all cases are reported to the coroner, although registrars have a responsibility to do so in all cases of death due to industrial disease.[4] And not all cases reported to the coroner result in an inquest, although necropsy may be performed (table). In four of our patients (two men, two women) no inquest was held despite the fact that the deceased people had worked in an asbestos factory.

We believe that the inquest is important. One of its functions surely is to examine the circumstances giving rise to the mesothelioma, and it may be the first occasion when relatives become aware of a possible link between the disease and previous exposure to asbestos.

Professor Seaton discussed the problems of obtaining financial compensation through industrial injuries benefits or civil litigation. The system for awarding industrial injuries benefits has been described as lengthy and obscure.[5] A useful source of advice about litigation is the Society for the Prevention of Asbestos and Industrial Disease (38 Drapers Road, Enfield EN2 8LU).

A further problem arises when a person develops mesothelioma because of indirect exposure to asbestos from a relative or close associate who had worked with asbestos. Such people are currently not entitled to any industrial injuries benefit. Fourteen out of the 180 cases of mesothelioma in Leeds fell into this category. Surely equity demands that such people should also be entitled to some form of benefit.

Finally, may we draw attention to recent legislative changes affecting industrial benefits. These changes[6,7] mean that any industrial benefits received as a result of injury or disease will in future be deducted from any subsequent award or out of court settlement made in compensation above the sum of £2500. These changes will apply to asbestos related diseases.

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Unawareness of hypoglycaemia

SIR,—Dr R E J Ryder and colleagues have suggested that no direct relation exists between hypoglycaemic unawareness and autonomic neuropathy in patients with insulin dependent diabetes.[1] This conclusion is in general agreement with the results of our cross sectional survey of a diabetic population performed in Edinburgh[2] but contrary to the traditional view that hypoglycaemic unawareness is a feature or manifestation of autonomic neuropathy.[3]

Both autonomic neuropathy and hypolgycaemic unawareness become more common as the duration of diabetes increases. In our study 37% of patients with normal awareness of hypoglycaemia had abnormal autonomic test results, whereas one third of those patients with hypoglycaemic unawareness had no objective evidence of cardiovascular autonomic abnormalities. Analysis of the results from those patients who had had diabetes for more than 15 years showed that 54% of those with normal awareness of hypoglycaemia had evidence of cardiovascular autonomic impairment compared with 59% of those who had hypoglycaemic unawareness. Although autonomic neuropathy and hypoglycaemic unawareness were fairly common, their association was not invariable.

During physiological studies of acute hypoglycaemia Hilsted reported that the diabetic patients with autonomic neuropathy were fully aware of the onset of hypoglycaemia, experienced classical symptoms of hypoglycaemia, and were seen to sweat.[4] We recently studied six diabetic patients with autonomic neuropathy, five of whom were aware of the onset of hypoglycaemia and who showed objective evidence of an autonomic reaction (including sweating). The intensity and number of autonomic and neuroglycopenic symptoms were similar to those of the symptoms experienced by patients who had had diabetes for a similar time but who had no evidence of autonomic neuropathy. These studies suggest that the relation between diabetic autonomic neuropathy and hypoglycaemic unawareness is much less definitive than has been stated previously.[5]

Dr Ryder and colleagues showed impaired glucose counterregulation in the diabetic patients with a history of hypoglycaemic unawareness and an apparent association between loss of symptomatic awareness and deficient counterregulation. Although concentrations of few counterregulatory hormones were measured, the impaired blood glucose recovery may have resulted from a failure to lower the blood glucose concentration to the threshold that triggers the counterregulatory response, so that a maximal effect was not achieved. Thus the glycaemic thresholds both for autonomic activation and for counterregulation have been altered so that a greater fall in blood glucose is required, which, coincidentally, causes severe neuroglycopenia. This premise is supported by a study that showed that patients with hypoglycaemic unawareness developed an acute autonomic reaction at a lower blood glucose concentration than did the normally aware subjects. The autonomic reaction occurred after the development of severe neuroglycopenia, which probably obtunded the perception of the autonomic warning symptoms.[6] This phenomenon may represent a form of central hypothalamic dysfunction,[7] which has been implicated in other studies of insulin dependent diabetic patients.[8,9]

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Action taken after deaths due to mesothelioma in Leeds 1971-87

	No of deaths	No who had mesothelioma recorded on death certificate	No of inquests held*	No who had coroner's necropsy but no inquest	Other
Men	125	109	91	5	13
Women	55	45	30	6	8
Total	180	154	121	11	21

*It could not be determined whether an inquest had taken place for one woman.

SIR,—Dr R E J Ryder's and colleagues' suggestion with regard to the nature of the defect in hypoglycaemic counterregulation[1] is similar to the proposal we published in 1979—that is, that there is a central regulatory abnormality, probably in the hypothalamus.[2] Our conclusion was based on studies in 20 diabetic and 14 normal subjects: the diabetic patients with counterregulatory impairment also had lower cortisol and growth hormone responses than the other subjects (both normal and diabetic). Subsequent studies by other groups may not have taken the prevailing blood glucose concentrations sufficiently into account when assessing the adequacy of cortisol and growth hormone responses.