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Safety and health in the construction industry

Poor and, in Britain, badly served by occupational health

Construction is one of the most visible of our industries and now the most dangerous. In 1986-9, 439 men died at work and 11 270 were seriously injured.[1] Fatality rates are about 10 for every 100 000 employed a year and have remained constant over the past 10 years while those in other industries, especially manufacturing, have declined progressively.

Within the construction industry certain types of project are more dangerous—dam building, demolition, and tunnel boring—and certain tradesmen are at higher risk of major injury and death—steel erectors, roofers, and tunnel miners. Thus a construction worker has over twice the chance of dying or having a major injury at work than any other industrial worker. Moreover, the ratio of major injuries to fatal injuries is low, at 27 compared with 37 for all industries, indicating a greater than average chance of not surviving a major accident. A recent spate of deaths at the British end of the channel tunnel (eight since the start of the project compared with two on the French side) has recently thrown this embarrassing record into prominence. Before examining the reasons behind it we need to ask how bad Britain's record is compared with that of other nations.

Comparisons are not easy because modes of reporting accidents vary. There is also the problem of defining a construction worker (as opposed to simply a worker in the construction industry) and even counting them. The industry is forever expanding and contracting and relies for its labour force on large numbers of migrant subcontracted and self employed workers, not to mention those who escape registration altogether. Even comparisons between the British and French companies building the channel tunnel (both keep scrupulous accident statistics) have been difficult for this very reason. In general, however, and compared with Britain, major injuries certainly seem to be more common in developing countries, are probably more common in some developed countries such as France and the United States, and are probably less common in other developed countries such as those of Scandinavia.

The reasons for the high accident rate in construction work are not difficult to ascertain. The industry is incoherent and can daunt the most experienced of managers, including health and safety managers. Each of the many separate trades has its own hazards. Construction is labour intensive, and that labour tends toward the itinerant, unskilled, foreign, and, above all, subcontracted. Nearly three quarters of the injuries suffered by self employed people occur in construction work. Small contractors are the norm and have most of the accidents. In 1978 only 13% of deaths occurred in the 50 largest companies simply because they are better organised.[2] Lack of inspection on small, short lived sites is inevitable, but in 1987-8 the Health and Safety Executive made 8272 "blitz" visits to 10 000 contractors, half of whom had never seen an inspector before. Enforcement action had to be taken at one in every five sites for immediate improvement.

There are also technical reasons why construction is dangerous: it inevitably breaks new ground, there is plenty of movement horizontally and vertically, and because of the short duration of most projects the learning curve is steep or, more usually, non-existent. Construction work entails hard physical work and exposure to the elements, the site ethos is usually rancorous, and working conditions are moderately foul. There is some evidence that the industry attracts feckless men who enjoy an irregular, physical outdoor life and danger.[3]

Apart from accidents, other risks to health are high in the construction industry, although their profile is much lower. Even when corrected for social class, standardised mortality ratios are high for cancer of the lung and stomach, respiratory disease, and diseases of the circulatory system.[4] Construction workers are exposed to many harmful substances: unpleasant dusts, fumes from burning processes, gases from combustion processes and geological formations, and large numbers of toxic chemicals. There are also biological and physical hazards, particularly from noise but also from heat, vibration, inflammable materials, and compressed air. These are real hazards, but much of the excess morbidity is thought to be work related rather than directly occupational and must be exacerbated by the stresses of site work, low pay, long hours, poor food, and the abuses that an all male work force living away from home seems to indulge in.

When the channel tunnel breaks into France this year British tunnellers will be surprised to see an occupational health service in place there by law, employing hundreds of doctors specialised in construction—compared with three employed in Britain. The best known and most effective occupational health service for a construction industry, however, exists in Sweden, where since 1967 a coalition of employers, trade unions, and the government has created an institution called Bygghalsen, which employs doctors, paramedics, engineers, occupational hygienists, occupational safety managers, and experts in rehabilitation to provide occupational health to construction workers throughout the country. If only the employers' federations and the trade

unions in Britain lobbied the government to create a similar institution here this article would be redundant.

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1 Health and Safety Commission. *Annual report 1988-89*. London: HMSO, 1990.
2 Health and Safety Executive. *Construction health and safety 1981-82*. London: HMSO, 1983.
3 Barry T and Associates. *Behavioural analysis of workers and job hazards in the roofing industry*. Cincinatti, Ohio: Department of Health Education and Welfare, National Institute of Occupational Safety and Health, 1975.
4 Office of Population Censuses and Surveys. *Occupational mortality. Registrar General's decennial supplement for England and Wales 1970-72*. London: HMSO, 1978.

Acid suppression: how much is needed?

Adjust it to suit the condition

Many treatments are now available for acid related disorders—and there is sharp competition among their manufacturers. Mucosal protective drugs are much less popular than treatments that neutralise or suppress acid secretion, the most successful of which have been the H_2 receptor antagonists. Even more potent acid suppressing drugs have been developed, among which the proton pump inhibitor omeprazole stands out. Unlike the H_2 receptor antagonists, which compete for the receptor on the parietal cell, omeprazole inhibits hydrogen-potassium ATPase, the enzyme responsible for secretion of acid.[1] This is a clinically important difference because once the hydrogen-potassium ATPase is inhibited no acid secretion will follow no matter how vigorously the receptors are stimulated. As omeprazole is long acting achlorhydria can be sustained throughout 24 hours, whereas because the existing H_2 receptor antagonists are competitive the inhibitory effect at the receptor can be overridden with a powerful stimulus, such as a meal.

H_2 receptor antagonists therefore do not often achieve complete achlorhydria for 24 hours,[2] even when the dose is increased. Nevertheless, the extent to which acid needs to be neutralised or suppressed varies with the condition being treated: the most potent acid suppressing drugs are not needed in all circumstances.

The aim in clinical practice is to overcome or prevent damage to the mucosa by acid—when that is important. Patients with non-ulcer dyspepsia of the dysmotility type (typically complaining of nausea, distension, and premature satiety) are unlikely to be in danger of such damage. They seem to do well with prokinetic drugs, such as domperidone, metoclopramide, or cisapride.[3,4] Other types of dyspepsia, in which there is some risk of acid damage, may be safely treated without investigation in younger patients (under, say, 45) who have no worrying symptoms.[3] Depending on the severity and persistence of symptoms, antacids or H_2 receptor antagonists may be given as single courses of treatment.

When peptic ulceration or mucosal damage from acid is diagnosed, however, or when there is a risk of it occurring or recurring, a reliable means either of reducing acid secretion or of protecting the mucosa needs to be offered. Peptic ulcers heal well with acid suppression, and there is an excellent correlation between the degree of acid suppression (especially overnight) and the percentage of duodenal ulcers that heal. Modest acid reduction heals about 60% whereas profound reduction heals virtually 100%.[5] Reducing gastric acid concentrations at night, when the acid is not needed, seems attractive, and a single nighttime dose gives virtually identical healing rates to a divided dose and might aid patient compliance.[5,6] Giving the H_2 receptor antagonists with the evening meal may prolong its acid suppressing effect but is not likely to prove practically useful.[7] In gastric ulcers healing shows a better correlation for reduction of 24 hour acidity than with overnight acidity.[8] Mucosal factors pay a larger part in gastric ulcer, which may need 12 weeks' treatment with an H_2 receptor antagonist and endoscopic monitoring.

Clinically there is little to choose between the four H_2 receptor antagonists currently available, all of which are remarkably effective and safe.[9,10] Cimetidine is less potent than ranitidine, nizatidine, or famotidine and has clinically important interactions with some drugs—notably with anticonvulsants, theophyllines, and warfarin—the hepatic metabolism of which it inhibits by binding to cytochrome *P*-450.[11] A meta-analysis comparing cimetidine with ranitidine showed a small advantage for ranitidine of 7% in healing rates over a one month treatment period,[12] but such a difference is undetectable to the individual prescriber and must be offset against cimetidine being much cheaper. The newer compounds nizatidine and famotidine are similar in efficacy to ranitidine and have no appreciable advantage, both being more expensive than cimetidine.

The H_2 receptor antagonists heal about 75% of duodenal ulcers with four weeks' treatment, rising to over 90% at eight weeks. A healing course should probably be six weeks for non-smokers and eight weeks for smokers.[13] Changing the antagonist is unlikely to heal a resistant ulcer, although increasing the dose may help.[14]

About 5-20% of duodenal ulcers do not heal with an H_2 receptor antagonist and need more profound acid suppression, such as can be achieved with omeprazole. Omeprazole heals almost all duodenal ulcers in two to four weeks, swiftly relieves symptoms, and is now the treatment of choice for resistant duodenal ulcers—until eradication of *Helicobacter pylori* becomes more simple and effective.

Concerns have been raised, however, over the long term safety of such profound and sustained acid inhibition. Firstly, about a third of female rats taking very high dose omeprazole over most of their lifespan developed carcinoid-like tumours in the stomach. These tumours may be related to high concentrations of circulating gastrin consequent on achlorhydria, as antrectomy prevented an increase in the density of enterochromaffin-like cells,[15] which is much greater in the rat stomach than in humans. Carcinoid-like tumours are uncommon in pernicious anaemia, where gastrin concentrations are much higher than in patients treated with omeprazole.[16] This then is unlikely to be an important cause for concern. Secondly, bacterial overgrowth in the stomach due to the achlorhydria is feared to lead to production of carcinogens from food contaminants. This hypothesis was originally raised with cimetidine, with which it has now been largely discounted,[17] but it may be different with profound acid inhibition over a long period. Thirdly, a recent suggestion is that omeprazole may itself be genotoxic—that is, be capable of leading to the development of cancer. Glaxo laboratory scientists have published their own technique for screening for potential carcinogenicity of acid inhibiting drugs.[18] They claim that omeprazole had a genotoxic effect in their test whereas ranitidine did not. Their technique has not been validated by any other group and has been heavily criticised[19-21]—appropriately, in my opinion. Until more information is available this claim should be discounted. None the less, omeprazole is a new compound, and until more is known of its longer term safety the aim should be to use it in resistant ulcers and only for short term treatment. Longer