recovered fully with minimal support. Ten received more intensive treatment: four regained normal renal function, four had renal impairment when discharged, one was still receiving continuous ambulatory peritoneal dialysis six months after her initial illness, and one died.

This small uncontrolled series included only one patient who had had surgery. In a double blind study, however, 20 patients receiving diclofenac after oesophagogastrectomy had significantly reduced urine output and potassium excretion, with a tendency to hyperkalaemia.[2] One patient receiving diclofenac developed renal impairment requiring treatment with dopamine. Degradation products of prostaglandin were greatly increased on the first postoperative day in controls, but this rise was abolished by diclofenac. Renal perfusion after surgery is maintained by the increased production of renal prostaglandins; this is blocked by the use of non-steroidal anti-inflammatory drugs.

Acute renal impairment during treatment with non-steroidal anti-inflammatory drugs is more likely in elderly patients, patients with hypertension and vascular disease, and patients with hypovolaemia and pre-existing renal impairment. In such cases we suggest that nephrotoxicity related to use of non-steroidal anti-inflammatory drugs may be a serious problem, with increased morbidity and occasional deaths; this cannot be ignored when postoperative analgesic requirements are being considered. Particular caution is indicated in patients with infection.

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Don't dismiss gastric erosion

EDITOR,—Dermot F Murphy states that no information is available regarding short term use of non-steroidal anti-inflammatory drugs.[1] In fact, a considerable number of studies have confirmed that acute gastrointestinal damage occurs in association with short term use of the drugs. Fourteen such studies have been reviewed elsewhere[2-4]; they are not listed in full here because of limitations on space.

Gastric damage has been observed endoscopically within minutes of a single dose of aspirin, and almost all subjects develop erosions within 24 hours if further doses of aspirin are given.[2,3] In two studies in which indomethacin 50 mg was given three times daily all volunteers had endoscopic evidence of mucosal damage in both the stomach and duodenum after 24 hours.[5]

After five days of parenteral administration of ketorolac (90 mg) "invasive" antral ulcers were found in four of five subjects.[3] This study indicates that non-steroidal anti-inflammatory drugs not only produce gastric damage by a topical irritant effect but may also rapidly produce ulcers by a systemic mode of action.

These studies clearly show that short term use of non-steroidal anti-inflammatory drugs may rapidly cause erosive damage to the upper gastrointestinal tract. Murphy asks whether, when they are used short term, these drugs should be accompanied by a prophylactic agent to prevent the gastrointestinal damage. This raises the issue of the clinical relevance of erosive lesions associated with ingestion of non-steroidal anti-inflammatory drugs and their propensity to develop into frank ulcers. This is clinically important because patients are at greatest risk of upper gastrointestinal bleeding or perforation early in the course of treatment with the drugs.[2] A recent study showed that arthritic patients with minor erosive damage can rapidly develop ulcers and that this progression can be prevented.[4] Perhaps, therefore, perioperative prophylaxis should be considered in those patients identified as being at greater risk of developing gastrointestinal damage.

Murphy points out that the results of long term studies with H_2 antagonists do not support their use for short term prophylaxis. But misoprostol is effective in preventing gastric and duodenal ulceration caused by non-steroidal anti-inflammatory drugs[4] and is licensed for this indication.

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The hazards of asbestos

Detailed occupational history is vital

EDITOR,—The study by Helen De Vos Irvine and colleagues adds new and important information to the debate on the relations between exposure to asbestos, asbestosis, and lung cancer.[1] The clinical and pathological consensus seems to be that attributing cancer to exposure to asbestos is possible only if there is other evidence of a pathological response to exposure—either asbestosis or diffuse pleural fibrosis. Epidemiologists, on the other hand, have regularly found a substantial excess of lung cancers in cohorts of workers exposed to asbestos, those excesses having been at least of the same order of magnitude as the number of cases of mesothelioma. Thus there are substantial differences between numbers of cases of "asbestos lung cancer" diagnosed and those predicted from epidemiological studies.

Why does this difference occur? Probably doctors in general take the Humean philosophical view that leads us to ascribe a disease, if possible, to one cause. As smoking is and has been so widespread among industrial workers we rarely look further for additional causes and therefore fail to appreciate the possible importance of industrial exposures in the aetiology of lung cancer. Moreover, it seems pointless to attribute cause in a condition in which knowledge of the cause is likely to be of so little clinical value. The only reason a doctor or patient may be interested in causation is to claim industrial injury benefits or to pursue litigation.

In commenting on this important matter Irvine and colleagues stray into areas apparently outwith their competence and experience and may unwittingly cause harm. In civil litigation causation is attributed on the balance of probabilities, which is a much less stringent test than is required for a sure clinical diagnosis. To suggest that patients seeking redress in the courts might have an open lung biopsy—a painful, expensive, and potentially fatal operation—to improve their chances is to advise doctors to act unethically. Moreover, as epidemiologists the authors should be aware of the great statistical variability of counts of fibres in lung tissue and of the great cost in time and money of the electron microscopic methods required to identify the fibres reliably.

It is a pity that an otherwise excellent paper has been marred by the authors changing their hat from that of scientists to that of concerned citizens. Their final paragraph need only have had its final sentences modified to emphasise the importance of a detailed occupational history in establishing legal causation. If we all took such histories from our patients with lung cancer we would find the cases that the epidemiologists tell us we are missing. Whether such patients would receive redress is a matter for the courts and for parliament.

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Inaccurate diagnoses distort results

EDITOR,—Throughout their paper on asbestos and lung cancer in Glasgow and the west of Scotland Helen De Vos Irvine and colleagues use the term "lung cancer" as if it was synonymous with "primary carcinoma of the lung" as opposed to sarcoma, lymphoma, or metastatic tumour. As 54% of these "cancers" were not verified histologically many of them would have been metastatic cancers or would not have been cancers at all. In fact, the distinction between primary and metastatic carcinoma is inaccurate even with histological examination.

Although 80% of the mesotheliomas were verified histologically, the accuracy of this diagnosis is also suspect as it was made in 1975-84, before the routine use of immunohistochemical examination. If this could have been done the diagnosis would have been different in many cases. The number of dubious dignoses in this study makes it difficult to have complete faith in the results, although I agree that the main conclusion (that many malignancies associated with asbestos are not recognised as such) is probably beyond dispute.

The authors suggest that an occupational history from those with "lung cancer" or mesothelioma should be used to select patients who may have been exposed to asbestos for an open lung biopsy and a count of asbestos fibres. But most manual workers can think of a possible episode of exposure if questioned directly, whereas only a few of these episodes prove to be important. Conversely, it is not uncommon to find patients with mesothelioma with no known history of exposure to asbestos but with a raised amphibole asbestos fibre count on electron microscopy with energy dispersive x ray microanalysis (the most accurate form of count). Thus many unnecessary open lung biopsies and expensive electron microscopic fibre counts (about £400 each) would be done on terminally ill patients, and even then an appreciable number of tumours associated with asbestos would still be missed.

By law deaths due to suspected occupational lung disease should be referred to a coroner (in England and Wales) and a coroner's postmortem examination will usually be performed. Lung tissue can be taken at this time and sent for an electron microscopic mineral fibre count if it is thought to be worth while. The counts are much more accurate when done on large samples, as opposed to biopsy specimens, as asbestos fibres are not evenly distributed in the lung. Counting asbestiform bodies, the authors suggest, is inaccurate as they can be formed by non-asbestos mineral fibres and are not always seen in lungs with a raised asbestos fibre burden. Even when they are present they tell nothing about the type of