gation and have emphasized matters often forgotten—such as that a lesion identified is not necessarily a lesion bleeding. However, we should like to make three points which we believe require further debate.

First, the 96% of successful visual diagnoses when the final diagnosis lay in the endoscopy area is slightly misleading, for in this series, as in others, 15% of the patients were discharged without any diagnosis having been established. The success rate should in our view be judged against this so that comparisons with other strategies can be made. When we began to use endoscopy as part of a systematized approach to upper gastrointestinal bleeding we had hoped that the “discharged without a diagnosis” rate would fall as a result of the identification of more patients bleeding from superficial lesions in the oesophagus and stomach. However, in our admittedly small series of 68 consecutive patients admitted to a combined medico-surgical service this has not been the case, though we have seen a shift in diagnostic pattern towards these lesions as the cause of the bleeding.

Secondly, because the patients of Dr. Cotton and his colleagues are a referral series and in spite of their failure to find other patients alive or dead in the hospital records we are doubtful if others will succeed in producing the same low mortality figures. Alternatively, the patients studied represent a selection reaching a London teaching hospital group and the application of the conclusions elsewhere may not be straightforward. Certainly our own results in Melbourne, where selection of admission was biased towards an elderly population of poor means, have been far less good for in spite of early identification and energetic treatment of the bleeding point six of our 68 patients died, only one from a preventable surgical error.

Thirdly, we are concerned that the authors seem to feel that endoscopy and radiology are necessarily in conflict and that one or other must be seen to emerge as better. Our experience with selective investigation does not confirm this, nor does it suggest that clinical histories as determinants of an investigative sequence are valueless. The best results—and by this we mean not only in mortality but in economy of time and number of investigations required to reach a diagnostic decision—are achieved by co-operation between clinician, endoscopist (if he is a separate person), and radiologist. We should not like to see this diagnostic team broken up by arguments over the best first buy for the most patients.

Lastly, may we be permitted to chide Dr. Cotton gently for discounting the value of clinical statistics in determining what to do next in a paper which, to the best of our reckoning, contains a percentage about patients or their investigations once every five and a half lines through 360 lines of text.—We are, etc.,

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Hazards of Laparoscopy

SIR,—I would like to draw attention to a further hazard of laparoscopy. In the course

of a recent laparoscopic tubal diathermy operation I had an unusual accident involving the Palmer's diathermy forceps. While taking a grip of the right Fallopian tube and isolating it from bowel I saw the terminal claw of the forceps detach from the rest of the instrument. After failing to remove the detached part with a second pair of forceps I was forced to proceed to a laparotomy in order to remove the detached part, to inspect the bowels and omentum for damage, and to complete the operation.

On subsequent examination the cause of the detachment was found to be a break of the soldered joint, which was easily repaired with silver solder. The instrument has since given no further trouble. I would thus suggest that a full check of all the joints of the laparoscope be made prior to operation as otherwise a laparotomy for “metal fatigue” may result.—I am, etc.,

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SIR,—We wish to report a case of severe retroperitoneal haematoma complicating laparoscopy.

A 29-year-old para 4+0 was admitted four months after delivery for laparoscopic sterilization, which was performed under general anaesthesia. The Verres needle was inserted just below the umbilicus at the second attempt and 3 l. of carbon dioxide introduced. The laparoscope itself was inserted at the same site and diathermy and division of the Fallopian tubes performed.

When the laparoscope was about to be removed a large retroperitoneal haematoma was noted over the bodies of the lower four lumbar vertebrae and estimated to contain 1,500 ml of blood. An immediate laparotomy showed a small puncture mark in the peritoneum of the posterior abdominal wall over the haematoma. But there was no further increase in the size of the haematoma and the abdomen was closed. The patient had a five-pint blood transfusion and made a good postoperative recovery.

We now consider that the safest site for introduction of the Verres needle is halfway between the symphysis pubis and the umbilicus, with the patient in the lithotomy position and with 30 degrees of head-down tilt. We continue to introduce the laparoscope itself at the lower border of the umbilicus.

The annual reports of the Medical Defence Union for 1971 (p. 51) and 1972 (p. 17) refer to two deaths and nine serious complications following laparoscopy, including a diathermy burn of the small bowel causing peritonitis. Only one retroperitoneal haematoma complicating laparoscopy has been previously reported and this was associated with fatal air embolism.¹ A review of 20,872 cases by Siegler² included 19 deaths due to haemorrhage, peritonitis gas embolism, and anaesthesia. One thousand cases of diagnostic laparoscopy reported by Duignan *et al.*³ and 910 cases of laparoscopic sterilization reported by Jordan *et al.*⁴ were attended by a total of 22 complications and no fatalities.—We are, etc.,

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Disability, Survival, and Coalworkers' Pneumoconiosis

SIR,—It is very pleasant to get from Dr. A. I. Cochrane another episode (2 June, p. 532) in the pneumoconiosis saga from the Rhondda Fach. Sadly, it sounds like one of the final chapters, which is a great pity when so much could be learnt by another clinical and radiological survey at this time.

Dr. Cochrane states that simple pneumoconiosis is not associated with appreciable disability or loss of expectation of life. Indeed, no other overall conclusion seems possible from the figures. But in my view quite different conclusions may be drawn if the frequency of chronic bronchitis is uneven in the groups of miners. There are good grounds for suspecting that this may be so. In people with chronic bronchitis, with excess mucus secretion and narrowed airways, fewer respirable dust particles reach the alveoli.¹ As the development of simple pneumoconiosis is related to the amount of dust reaching that level, bronchitis probably gives some protection. There may therefore be a considerable excess of bronchitis in miners with category O and a decline in their number with higher categories of simple pneumoconiosis.

The possible effect can be illustrated by considering ventilatory capacity. Take groups of 100 miners with different categories of simple pneumoconiosis and a group of non-miners. Assume that chronic bronchitis occurs in 20% of the non-miners and, overall, in 30% of miners but that its distribution is uneven (see table). Award every man an expected ventilatory function score of 100 and assume that chronic bronchitis reduces this on average by 30, category 1 by 5, category 2 by 10 and category 3 by 15. The total score for the non-miners is then $(80 \times 100) + (20 \times 70) = 9,400$. The totals for the mining groups are shown at the foot of the table and these fit pretty accurately on to table III of Cochrane, Higgins, and Thomas² relating ventilatory function to categories of pneumoconiosis. This theory can go a long way to explain why miners without pneumoconiosis have poorer ventilatory levels than non-miners and it shows how substantial impairment due to simple pneumoconiosis can be concealed.

The conventional test of ventilatory function is the forced expiratory volume (FEV) and this reflects in particular air flow in large and medium-sized bronchi, areas heavily involved in chronic bronchitis. In simple pneumoconiosis, however, any obstructive element can be expected to be in the respiratory bronchioles or beyond, areas very poorly represented by the FEV. The current method of assessing obstruction in small airways is by demonstrating abnormalities in closing volume and frequency-dependence of compliance. Using the latter, small-airways disease has been demonstrated in simple pneumoconiosis³ and this may produce imbalance in alveolar ventilation and

perfusion, and consequent disability, without reducing the FEV. Therefore disability produced by simple pneumoconiosis may not only be masked by an unequal distribution of chronic bronchitis, but the tests that have been relied on to look for it are particularly insensitive.

This argument may be rejected because there is little or no evidence that chronic bronchitis is less common in miners with higher categories of simple pneumoconiosis than in those without. Because, during life, chronic bronchitis has to be diagnosed on the presence of symptoms and because it has been convincingly shown⁴ that simple pneumoconiosis itself produces the same symptoms (even when smoking habits are taken into account) there are enormous difficulties in determining the distribution of chronic bronchitis in miners. The same investigation⁴ showed that the presence of simple pneumoconiosis was associated strongly with a complaint of breathlessness. This again would be expected with small-airways or alveolar disease and this seems a more logical explanation than an assumption that the men complained of breathlessness because many were aware that they had pneumoconiosis.

Satisfactory information about the distribution of chronic bronchitis in miners can probably come only from careful postmortem bronchial histology. Some attempt at this was made by Lyons *et al.*⁵ but their series did not include enough subjects without pneumoconiosis to achieve valid conclusions. I have a very strong clinical impression that severe chronic bronchitis, characterized by the "blue bloater," is seldom if ever seen in a man with category 3.

Unequal distribution of chronic bronchitis may also conceal a reduction in life expectancy due to simple pneumoconiosis. Dr. Cochrane's table III gives a little support for this view. Below the age of 45 there is somewhat higher mortality in men with simple pneumoconiosis. In these younger men there should be less bronchitis and thus less "protection" from the development of pneumoconiosis. Mortality would then be a better reflection of the effect of simple pneumoconiosis. Between 45 and 65 years mortality is considerably higher in men with category O than in those with simple pneumoconiosis and this may be due to an excess of bronchitis in the former group.

I believe Dr. Cochrane's conclusions should be qualified by adding that the distribution of chronic bronchitis in miners is probably uneven. Because of the marked effect this has on conventional ventilatory tests and on mortality, uneven distribution may well mask substantial disability and mortality produced by higher categories of simple pneumoconiosis. As someone who tries to avoid being emotionally stirred by a number of dots on x-rays, I still find it impossible, when looking at microscopical and paper-mounted sections of lungs with extensive simple pneumoconiosis, to believe that it has no significant effect on a man's capacity to breathe comfortably and to live to an old age. There are, of course, other ways in which simple pneumoconiosis can produce disability⁶⁻⁷ which have not been touched on here or by Dr. Cochrane.—I am, etc.,

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	Non-Miners	Miners			
		Cat. 0	Cat. 1	Cat. 2	Cat. 3
Number	100	100	100	100	100
No. with bronchitis	20	45	40	25	10
Ventilatory score for non-bronchitics	8,400	5,500	5,700	6,750	7,650
Ventilatory score for bronchitics	1,400	3,150	2,600	1,500	550
Total ventilatory score	9,400	8,650	8,300	8,250	8,200

¹ Lippman, M., Albert R. E., and Peterson, H. T., in *British Occupational Hygiene Society, Symposium on Inhaled Particles*, vol. 1, p. 105. London, Unwin Bros. Ltd., 1971.

² Cochrane, A. L., Higgins, I. T. T., and Thomas, J., *British Journal of Preventive and Social Medicine*, 1961, 15, 1.

³ Seaton, A., *Thorax*, 1973, 28, 267.

⁴ Ashford, J. R., Morgan, D. C., Rae, S., and Sowden, R. R., *American Review of Respiratory Diseases*, 1970, 102, 370.

⁵ Lyons, J. P., Ryder, R., Campbell, H., and Gough, J., *British Medical Journal*, 1972, 1, 713.

⁶ Lyons, J. P., Clarke, W. G., Hall, A. M., and Cotes, J. E., *British Medical Journal*, 1967, 4, 772.

⁷ Morgan, W. K. C., Burgess, D. B., Lapp, N. L., and Seaton, A., *Thorax*, 1971, 26, 585.

Prodromal Symptoms of Biliary Colic or Cholecystitis

SIR,—Prodromal symptoms are such as may occur in the time immediately preceding a more serious disease. On 3 and 4 April 1969 I suffered an acute attack of cholecystitis for which the gall-bladder was successfully removed. During the six months preceding this acute attack I had suffered some symptoms which puzzled me and my doctor and which I mentioned in a short article published in the *B.M.J.* (18 July 1970, p. 147).

These prodromal symptoms occurred at intervals of one to three weeks. The first attack was in about September 1968. I had got to sleep about ten o'clock and woke suddenly a few hours later; I had no pain but I was sweating profusely and my heart was beating about twice its normal rate. I sat up in bed wondering what had caused these symptoms, but since they gradually subsided I took little notice of them. But soon they became frequent enough to cause me to have a towel by my bedside to dry myself if an attack occurred. My doctor examined me thoroughly but could find no cause for the symptoms, but on the night of 9-10 February 1969 I had a more severe attack and rang the bell to my sister's room (she is a qualified nurse) and since I was unable to get on the telephone to my own doctor I phoned my sisters' doctor, who gave me good advice, told me to take my usual hypnotic, and the attack passed off. There was no pain at any time, but the awakening was so sudden that it must have been due to some unascertainable cause.

Then on 3 April I had the acute attack of cholecystitis which I described in my published account. The gall-bladder was already necrotic in some places and when it was removed and opened was found to contain 15 pigmented gall-stones (black pigment). There was no stone which could possibly have passed into the cystic duct but all the stones were irregular in shape and rather flattened, and two of them had each of them at one end a smooth rounded quarter-inch that made it quite certain that they had attempted to enter the cystic duct. Neither of them could

have got far into the duct but I came to the conclusion that their attempted entry into the duct may have been the direct cause of the sweating and the increase in heart rate. At the same time I could not with assurance say that they had caused the symptoms and I have waited four years before I give my opinion that my suggestion was correct, for I have not had any similar attack since the operation was performed.

It is well known that reflexes from the gall-bladder can cause symptoms which can call attention to the heart rather than the gall-bladder and copious sweating could easily be caused reflexly through the sympathetic nerves. The absence of pain can be explained by the fact that I sat up immediately when awakened and the stone would at once fall away from the neck of the gall-bladder. I have never seen any reference to similar prodromal attacks, nor have I heard any mention of them, so they are either very rare or have been overlooked. I am writing this note so that the readers of the *B.M.J.* may be on the look out for any such similar prodromal symptoms.—I am, etc.,

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The Problem Oriented Medical Record

SIR,—One of the major benefits of Professor Lawrence Weed's system of problem oriented medical records which was described by Dr. Neil McIntyre (9 June, p. 598) is the structuring of the clinical data which ensues from it. As the structuring of data is one of the more difficult parts of the design of any system for making inter-patient comparisons, particularly if a computer is involved, it is not surprising to find that one has been used as a vehicle for Professor Weed's record.

May I appeal to those interested in this system, however, not to associate too closely the ideas of problem oriented medical records and the use of a computer to collect and process these records. As you say in your leading article in the same issue (9 June, p. 570), a lot of research is still needed in the application of computing techniques in medicine. Professor Weed's ideas can undoubtedly be applied with great benefit to existing medical data; and should a successful system "pencil and paper" methods of recording of computer-based problem oriented records not be achieved as rapidly as we would wish, it is important that the potential benefits of the problem oriented approach should not be discarded because of difficulties inherent in the application of today's technology in medical environments.—I am, etc.,

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Hospital Medicine Sheets

SIR,—It is surprising that after 20 years of the National Health Service no standard form of hospital medicine prescription sheet has become generally acceptable and applicable.

At the present time different hospital groups, and their separate hospitals in some instances, have their own specially designed and printed sheets. This involves unnecessary