our respondents. Secondly, we based our 50-item questionnaire on verbatim attitude and opinion statements derived from interviews with members of the various hospital staff groups under study.

While we would agree that it is important to stress the positiveness of attitudes to psychiatry, we consider it equally if not more important to define the areas of prejudice that still exist and which at least up to date defy change. For such prejudice might seriously hamper the further integration of psychiatry in the general hospital and yet go undetected. The fuller analysis of our findings should help to clarify what attitudes still require change and hence lead to a more rational teaching programme for all general hospital staff. Finally, we would hope our results will encourage research into methods of teaching by which such prejudices can be overcome.—We are, etc.,

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Renin Activity in Acute Renal Failure

SIR,—Dr. J. J. Brown and others (31 January, p. 253) have recently reported a significant increase in the renin concentration of peripheral venous blood in most of their patients with acute renal failure. The highest levels were found during the first two weeks after onset, while lower levels were detected during the diuretic phase.

We wish to record our experience with nine patients (five men and four women) with acute renal failure of diverse aetiology: surgery on the gastrointestinal or biliary tract, four; eclampsia, two; and crush injury, heat exhaustion with hypotension, and acute gouty nephropathy, one case each. Acute tubular necrosis was proved by kidney biopsy in four patients. Renin

patients. Renin activity ranged from 2.0 to 8.9 ng./ml. in four of the five patients studied during the oliguric phase, and from 2.0 to 12.2 ng./ml. in five of the six patients observed during the diuretic phase. There was no correlation between the levels of renin activity and serum sodium, potassium, systolic, diastolic, and mean blood pressure. Our findings are summarized in the Table along with results reported by other investigators.

Although the clinical material summarized above included a variety of aetiologies, and the methods of assay employed were dissimilar, it is apparent that during the oliguric phase increased levels of renin or renin activity were found in most instances. We, as others, found no correlation between the levels of renin activity or renin and of serum sodium, potassium, urea, or creatinine. As regards blood pressure, Tu² found a slight positive correlation with renin activity, Dr. Brown and colleagues found a negative correlation, while we and Kokot and Kuska³ observed no correlation. These findings strongly suggest that the stimulus for the increase in renin secretion in acute renal failure arises within the kidney itself, rather than extrarenally.

The possible pathogenetic significance of increased renin in acute renal failure has been exhaustively discussed by Dr. Brown and his colleagues. We believe that their provocative interpretation has considerable merit. Recent studies by Hollenberg *et al.*⁴ have shown that in acute renal failure precipitated by nephrotoxic agents as in that caused by other aetiologies, the outstanding feature is marked reduction in cortical blood flow, with decrease in filtration pressure and filtration rate.⁵ These observations lend support to the concept that in the pathogenesis of acute renal failure activation of a vasomotor intrarenal pathway plays a primary role. In 1945 Goormaghtigh⁶ drew attention to the prominence of the juxtaglomerular apparatus in the crush syndrome and suggested that excessive release of renin and production of angiotensin might be responsible for the spasm of the glomerular afferent arteriole leading to reduction in glomerular filtration rate. To date evidence in support of or against this hypothesis is still lacking. If the increase in the blood levels of renin or renin activity found in clinical and experimental acute renal failure is coincidental or simply the result of the intrarenal disturbance, then the cortical ischaemia of acute renal failure is due to other vasoconstrictor substances. Alternatively, although in acute renal failure renin is markedly increased, lack of adequate amounts of substrate and/or of converting enzyme prevents the formation of angiotensin II within the kidney. Another possibility might be that angiotensin II is inactivated as soon as it is generated.

Oparil *et al.*⁷ have found that direct infusion of angiotensin I into the lung of the anaesthetized dog resulted in 54% conversion to angiotensin II. By contrast, infusion of angiotensin I into the renal artery yielded only 7 to 9% conversion. Whether these experimental findings are applicable to clinical renal failure in man remains to be seen. None the less, even assuming that the kidney has a smaller capacity for converting angiotensin I to angiotensin II, under the appropriate circumstances the angiotensin II produced

may well have pathogenetic importance, as Dr. Brown and his colleagues have suggested.—We are, etc.,

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REFERENCES

- Pickens, P. T., Bumpus, F. M., Lloyd, A. M., Smeby, R. R., Page, I. H., *Circulation Research*, 1965, **17**, 438.
- Tu, W. H., *Circulation*, 1965, **31**, 686.
- Kokot, F., and Kuska, J., *Nephron*, 1969, **6**, 115.
- Hollenberg, N. K., Adams, D. F., Oken, D. E., Abrams, H. L., and Merrill, J. P., *New England Journal of Medicine*, 1970, **282**, 1329.
- Earley, L. E., *New England Journal of Medicine*, 1970, **282**, 1370.
- Goormaghtigh, N., *Proceedings of the Society for Experimental Biology and Medicine*, 1945, **59**, 303.
- Oparil, S., Sanders, C. A., and Haber, E., *Circulation Research*, 1970, **26**, 591.

Treatment of Anal Fissure

SIR,—I am currently conducting a four-year follow-up of lateral subcutaneous internal sphincterotomy for chronic anal fissure in 98 patients in Colchester, and I am pleased to say that the findings are in accord with those of Mr. D. C. Hoffmann and Professor J. C. Goligher (19 September, p. 673). There appears to be a slightly higher incidence of haematoma formation, but there has been only one case admitting to minor defects of anal continence. There have been no cases of recurrent fissure, and in only one patient was the sphincterotomy slightly enlarged at a later date when a prolapsing pile was excised.

I believe that this treatment for chronic fissure is the most satisfactory, particularly because anal continence is so well preserved, and in my series I have performed the operation for chronic fissure in three cases of ulcerative colitis. These patients are fully continent, whereas I would have suspected a degree of incontinence if sphincter stretching or posterior sphincterotomy had been performed.—I am, etc.,

DOUGLAS MILLAR.

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Osteoporosis and Fluoride Therapy

SIR,—Your interesting leading article (19 September, p. 660) indicates that there is only scanty information, particularly in the British literature, about the long-continued use of sodium fluoride in the management of osteoporosis in man. Because of this, the leading article quite properly advocates a cautious approach to fluoride therapy. In the context of possible long-term toxicity we should like to record our own experience with fluoride.

We began using sodium fluoride in 1962 and have so far treated 25 patients with osteoporosis, as well as 20 patients with Paget's disease. The results during the first five years were communicated to the Liverpool Medical Institution in 1967. Our

Renin and Renin Activity in Acute Renal Failure

Authors	Oliguric Phase* Incr./Total	Diuretic Phase Incr./Total	Recovery Phase Normal/Total
Tu ²	8/18	1/8	
Kokot and Kuska ³	28/33	8/19	
Brown <i>et al.</i>	22/24	4/8	1/1
This Report	4/5	5/6	1/1
Total	62/80	18/41	2/2
% Increase (mean)	77.5	44	

*Daily urine volume less than 1,000 ml.

activity of peripheral venous blood, measured by the procedure of Pickens *et al.*¹ was increased above normal (normal range with subjects supine: 0.1 to 1.7 ng. of angiotensin II per hour per one ml. of serum) on one or more occasions in seven

approach to osteoporosis is to attempt to maintain a positive calcium balance throughout its management, and sodium fluoride is given to promote this, when indicated, in a minority of patients.

The initial dosage of sodium fluoride is 20 mg. t.d.s., or occasionally q.d.s., and treatment is normally continued for 500 days, until 30 g. have been given. During this period and after cessation of treatment progress is monitored biochemically and by balance studies. Further smaller courses of treatment are given, as indicated. We have looked carefully for signs of toxicity, particularly optic neuritis, and with the above regimen have found none, except for gastric irritation. The latter is eliminated in most patients by taking the drug in capsules and with meals.—I am, etc.,

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Coalworkers' Pneumoconiosis

SIR,—Dr. R. Ryder and colleagues (29 August, p. 481) have made use of their unusual opportunities to report on a large group of miners and ex-miners in whom it was possible to study the chest radiographic appearances of pneumoconiosis and ventilatory capacity in life and a grading of emphysema in the lungs at post mortem. They say, "There is no reason why these deaths should not provide a true sample of the experience of men with this disease." We assume by this they mean that the relationship between measurements made in life and death are representative of the general population of miners and ex-miners with pneumoconiosis in the South Wales area they studied. Is this true? It is well established^{1,2} that spurious relationships and correlations are generated when factors which are unrelated in the general population affect the risk of entry into medical records or risk of dying. As an illustration of the way in which the association of two characteristics in the living population may be distorted by differential death rates to give a different association among those selected at post mortem, we may use age and radiological category of pneumoconiosis. In the Rhondda Valley in 1950 (Cochrane, personal communication) the average ages of the men according to radiological category were:

category 0	43.8
1	48.9
2 and 3	46.3
A	51.4
B and C	55.8

As would be expected there was a fairly steady increase of average age with increasing severity of pneumoconiosis. In the series Dr. Ryder and others report this association is lost.

As the death rate is affected by the FEV level,³ the amount of emphysema,⁴ and the presence of P.M.F.,⁵ a correlation will be expected between these variables in a sample selected at post mortem in this way, even if no such relationship existed in life. This important possible source of bias in, for example, their Fig. 3 relating FEV and emphysema grading, is not discussed.

There is another difficulty in constructing Fig. 3 at its face value. The figure does not differentiate between cases of simple and

complicated pneumoconiosis. Complicated pneumoconiosis itself, by reducing the size of the lung, will reduce the FEV. How much of the relation in Fig. 3 is due to the effect of progressive massive fibrosis (P.M.F.) and how much to emphysema? The Tables show that those with low FEV contain a higher proportion of cases of P.M.F. In addition bronchitis, in the absence of emphysema, is itself related to FEV. The authors do not discuss these factors as a possible cause of the association they show in Fig. 3.

The comparison of emphysema grading in miners and ex-miners and in a non-mining group is interesting, particularly the differences in the age relationships in the two groups. Is it not possible that bias was introduced at the time of assessment because it must have been clear from the coal dust in the lungs which were the miners? Also, is it not possible that the outlining of the spaces by coal pigment itself increases the ease of detecting "abnormal air spaces"? In comparing the emphysema counts in those with "p," and "m" and "n" types of pneumoconiosis, no mention is made of the possible effects of age differences in the groups.

We congratulate the authors on their attempt to meet Fletcher's requirements,⁶ but we are unhappy about the possible fallacies in the analysis of their findings. Perhaps further work on the analysis will permit the measurement of the magnitude of the biases we suggest may be present, and so help to develop methods for dealing with material of this kind.—We are, etc.,

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REFERENCES

- 1 Berkson, J., *Biometrics Bulletin*, 1946, 2, 47.
- 2 Mainland, D., and Herrera, L., *Methods in Medical Research*, 1954, 6, 165.
- 3 Gilson, J. C., *Proceedings of the Royal Society of Medicine*, 1970, 63, 857.
- 4 Jones, N. L., Burrows, B., and Fletcher, C. M., *Thorax*, 1967, 22, 327.
- 5 Cochrane, A. L., Carpenter, R. G., Moore, F., and Thomas, J., *British Journal of Industrial Medicine*, 1964, 21, 38.
- 6 Fletcher, C. M., in *Form and Function in the Human Lung*, ed. G. Cumming and L. B. Hunt, p. 37. Edinburgh, Livingstone, 1968.

SIR,—Dr. C. Ogilvie (26 September, p. 769) and Dr. M. C. S. Kennedy (10 October, p. 115) raised several points concerning our recent paper (29 August, p. 481), many of which we hope to deal with when all the findings arising from our correlation survey have been satisfactorily assessed; in the current paper we were primarily concerned with emphysema.

An important point, however, concerning the 20 category 0 cases may be dealt with at the present time.

As was recorded in the paper, the majority of these cases had, in fact, some radiological evidence of pneumoconiosis, and only four were considered to be unequivocal category 0 cases. In addition it should be made clear that where a majority of the readers of the radiographs preferred an alternative diagnosis to that of pneumoconiosis such as, for instance, fibrosing alveolitis, they classified the film as category 0 although a minority of them might have diagnosed p type simple pneumoconiosis on the same film. Such discrepancies amongst readers are by no means

rare, particularly when the radiological changes are atypical as is often the case in coalworkers' pneumoconiosis—the more so when additional disease is present. It is apparent, therefore, that our category 0 cases, possibly excepting the four referred to above, cannot be regarded as typical since the great majority of them showed some radiological evidence of either pneumoconiosis or other pulmonary disease, and for this reason we do not believe that they support Dr. Ogilvie's conclusions regarding compensation.

We agree with Dr. Rae and others (26 September, p. 769) that to determine whether there is more emphysema in the mining community as a whole than in the general population would ideally require samples representative of both communities, whereas our sample of miners is confined almost exclusively to cases with pneumoconiosis. However, emphysema as defined is essentially a diagnosis based upon the morbid pathology of whole-lung sections and we know of no way of obtaining such representative samples from total populations.—We are, etc.,

R. RYDER.
J. P. LYONS.
H. CAMPBELL.
J. GOUGH.

Cardiff.

SIR,—I agree with Dr. S. Rae and others (26 September, p. 769) that Dr. R. Ryder and colleagues' findings (29 August, p. 481) of emphysema in coalworkers were "derived from a selected minority." I also agree with Dr. C. Ogilvie (p. 769) that it is not justifiable to base what he calls "compensation"—but what in fact are disability benefits—upon radiological categories. I very much doubt, however, whether there will ever be any comprehensive and sophisticated physiological studies which will differentiate between industrial and non-industrial dysfunction with a reasonable degree of accuracy.

All this does not invalidate in any way Dr. Ryder and colleagues' main findings based on their comprehensive and well planned pathological and histological study—that is, that emphysema was much more common among their miners compared with their carefully chosen control group of non-miners. If nothing else their important paper makes nonsense of the special provision in the Industrial Injuries (Amendment) Act of 1967, whereby "If the extent of a claimant's disablement resulting from pneumoconiosis would be assessed at not less than 50%, if his physical condition were otherwise normal, the effects of emphysema or chronic bronchitis from which he may be suffering are treated as if they were the effects of pneumoconiosis for assessment purposes."

I fully support professor C. R. Lowe's view¹ that the whole industrial injuries legislation which is based on antiquated Victorian compensation concepts needs scrapping, and that the social security benefits should be related to family needs rather than to the cause of the wage earner's disablement.—I am, etc.,

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REFERENCE

- 1 Lowe, C. R., *British Medical Journal*, 1969, 1, 463.