

I immediately entered the room where my friend was on the verge of collapse, but he got neither help nor sympathy. I later saw the young houseman again, and he told me the condition was an astrocytoma.

After a course of radiotherapy at another hospital the patient returned home, but deteriorated and was eventually admitted to the little 50-bedded cottage hospital in her home town. From the point of view of general comfort, nursing care, and kindness and understanding, this was by far the best of all the hospitals she had been in. Nothing was too much trouble to make her end as easy as possible, and to support her husband in his distress. This hospital is now in imminent danger of being closed down on grounds of cost.

It would appear that, the larger the hospital, the lower the level of common humanity and vice versa. Too few of our colleagues seem to appreciate that not only does the patient need kindness and understanding as part of therapy, but that in serious cases reasonable explanation to close relatives might well save them from subsequent severe psychological trauma. In this case the husband and child, being basically balanced individuals, faced up to the whole situation very well, but if they had been of less mature personality the consequences might well have been severe and long standing.—I am, etc.,

Dinas Powis,
Glam.

E. LLOYD JONES.

SIR,—I read Dr. David Kyle's Personal View (25 January, p. 246) with considerable interest, having appreciated the difficulties of doctor-relative communication during 18 months' of house jobs. I agree that the houseman can most easily fill the gap. Firstly, he has had most comprehension, however incomplete, of the patient's family and social background. Secondly, he possesses a unique knowledge of consultant and registrar decisions together with the nursing procedures to which a patient may be submitted. Thirdly, he possibly finds it more easy to communicate with the lay public than more senior medical personnel, and, lastly, is more available than the other medical staff.

In my limited experience a specific availability such as Dr. Kyle suggests can be extremely time-wasting. It is the same relatives who day by day request information, generally about dying patients. You can offer these relatives little new medical information, although it is probable that what they want is sympathy and reassurance which (I dare to suggest) can from time to time be given by the nursing staff. Thus I have found that sister or staff nurse can be an excellent "sorting department," deciding from those who ask who should be seen by the houseman, and deciding, perhaps more importantly, from those who don't ask those that should be seen. They must of course know where the houseman can be contacted during visiting hours, and this cannot relieve the houseman of his responsibility to seek out particular relatives where necessary.

While making these suggestions, I must express my admiration for one of my consultant chiefs who arranged to see the next of kin of every patient in whom we diagnosed a neoplastic condition. I have little

doubt that he would have made a personal arrangement to see Dr. Kyle should his father have been admitted under his care.—I am, etc.,

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PHILIP B. WOOD.

SIR,—I feel very sorry for Dr. David Kyle (Personal View, 25 January, p. 246) in what must be the professional isolation of South Wales. Personally I have been very much more fortunate in my dealings with consultants when my relatives have been ill, and find that they are only too willing to inform and discuss.

I can only hope that more people than myself have been startled by the picture that Dr. Kyle and Major C. G. Harper (25 January, p. 256) have presented to us.—I am, etc.,

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A. B. LEYS.

Acute Urography

SIR,—I have read with interest the article by Mr. R. D. W. McLean and his colleagues (18 January, p. 142). There can be no doubt that the performance of urography as an emergency procedure may be of great value in the detection and management of acute lesions of the renal tract. There are, however, several points which appear to me, as a radiologist, to merit comment.

Firstly, while it is true that a diagnostic answer may be obtained within 30 minutes in many cases, there are also many in whom delayed films are essential. This is particularly true in patients with obstructive uropathy, in whom the whole picture may not become clear for upwards of 24 hours. The authors refer to the use of "40 ml. of a 60% iodine solution." By this they presumably mean a 60% solution of one of the iodine-containing contrast media such as "Urografin 60" (Schering). If a routine dose is to be employed many would favour a larger quantity of a medium with a higher iodine content. Personally I believe that there is a case for infusion pyelography in many of these patients. As the authors point out, they are rarely either prepared or dehydrated, and abdominal compression can seldom be applied.

Few would dispute the value of "acute urography" in many patients with renal colic or following trauma to the renal tract. One has some misgivings, however, as to its employment in the investigation of painless haematuria, unless this be of such severity as to merit immediate surgery. The authors themselves obtained a diagnosis by this method in only three of seven cases so presenting. A comprehensive examination employing all departmental facilities is likely to be far more rewarding. I have no wish to discourage the employment of intravenous urography as an emergency procedure. I feel that it is important, however, to emphasize that the technique, as described, has limitations and is not a substitute for an examination carried out under radiological supervision and with all the facilities, including tomography, available.

Finally, may I be allowed a plea for the abandonment of line drawing as employed on the radiographs used to illustrate this article? I appreciate that there is inevitable loss of

definition with reproduction and that some points may require emphasis or clarification. It is, however, extremely difficult now to see what was originally displayed on these films.—I am, etc.,

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Self-levelling Venous Pressure Transducer

SIR,—Differential pressure measurement, as Dr. J. P. Blackburn (28 December, p. 825) describes, is convenient when monitoring central venous pressure, particularly when the subject may move. Similarly, although the potential relative error is less, measurement of the pressure difference between the reference point and the intravascular catheter tip is a convenience in measuring arterial pressure in a mobile subject. However, the technique suggested¹ has disadvantages. A membrane manometer is employed which receives pressure signals on both sides of the sensing membrane. The accuracy of such measurement of phasic pressure must be suspect. To produce meaningful measurements of phasic pressure a manometer must have adequate undistorted frequency response. In practice this requires adjustment of damping of the manometer-catheter system to decrease resonance, an adjustment readily obtained with standard strain-gauge manometers.¹ But when two catheter systems interact across the single membrane of a differential manometer this damping is most difficult to achieve.

I have described a simple, inexpensive and accurate means of differential pressure measurement by electrical subtraction of signals from two strain-gauge manometers. This allows damping to be accurately adjusted for each manometer² separately. Further it has the advantage that standard rather than special—that is, differential—manometers are used.—I am, etc.,

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Acidosis in Obese Fasting Patients

SIR,—Though metabolic acidosis is a recognized consequence of starvation, the routine estimation of arterial pH, PCO₂, and base deficit has been omitted from the many published studies of the treatment of obesity by fasting. In twelve patients admitted to a medical unit for treatment of gross obesity we found by regular blood-gas analysis that not only did severe acidosis develop rapidly in starvation but failure to do so or sudden improvement was good evidence that the fast had been broken.

The clinical data concerning our patients are summarized in the Table.

None of the patients had clinical evidence of cardiac, respiratory, or endocrine disease with the exception of cases 6, 8, and 11, who were known diabetics, and case 10, who was found to have a diabetic curve in a routine oral 50-g. glucose-tolerance test. Each patient had a normal chest x-ray, E.C.G., full blood-count,

Case No.	Age	Sex	Weight Before Treatment (in lb and kg.)	Excess Weight (Expressed as % Standard Weight)	Blood-gas Analysis Before Treatment			Minimum Recorded pH Values and Associated Indices					
					pH	PCO ₂ (mm.Hg)	Base Deficit (mEq/l.)	Days After Onset of Fast	Wt. Loss From Onset of Fast. (lb.)	Wt. Loss as % Pre-treatment Weight	pH	PCO ₂ (mm.Hg)	Base Deficit (mEq/l.)
1	16	F	184 (83.4)	60	7.36	45	1	5	9	5	7.32	29	10
2	18	F	230 (104.3)	81	7.38	40	1	10	21	9	7.31	36	8
3	20	F	215 (97.5)	54	7.38	40	2	5	11	5	7.29	33	10
4	20	F	198 (89.8)	41	7.38	40	2	5	10	5	7.30	30	11
5	21	F	166 (75.2)	54	7.37	42	1	13	19	11	7.26	25	14
6	37	F	212 (96.1)	64	7.39	36	3	5	9	4	7.26	33	12
7	40	F	299 (135.6)	103	7.38	36	3	12	21	7	7.28	34	10
8	41	F	227 (102.9)	77	7.39	42	-1	6	16	7	7.28	29	12
9	44	M	229 (103.8)	42	7.40	33	3	10	38	16	7.30	30	11
10	50	F	186 (84.3)	43	7.37	43	1	7	13	7	7.26	33	12
11	55	M	239 (108.4)	56	7.38	47	-2	11	13	5	7.29	34	10
12	58	F	257 (116.5)	74	7.38	40	1	5	16	6	7.30	34	9
Mean values	35	—	222 (100.6)	62	7.37	40	1.25	7.8	16.3	7	7.29	32	10.5

blood-urea, and electrolyte values, and a normal renal ability to excrete an acid load.¹ The pH, PCO₂ and base deficit in arterialized capillary blood samples obtained before and on alternate days during fasting were estimated using the Micro-Astrup method.² During fasting a liberal water intake was allowed, but apart from one cupful of beef extract daily no other foodstuffs or supplements were given. Exercise was limited to moderate ambulation within the ward area.

The acid-base indices are summarized in the Table. All patients developed a primary metabolic acidosis (base deficit > 2.5 mEq/l.) with some degree of respiratory compensation (PCO₂ < 35 mm. Hg). A significant acidaemia (pH < 7.30) was detected in all but cases 1 and 2, and in case 5 the pH fell to 7.31 as early as the second day of fasting. Though weight loss and ketonuria persisted throughout the period of fasting, any marked improvement in the blood-gases following the development of metabolic acidosis could be traced subsequently to surreptitious eating by the patients.

Whereas the metabolic acidosis of starvation is probably largely due to ketone production by the liver, the possibility of impairment in the body's mechanisms of dealing with an acid load cannot be overlooked. Though our patients were able to excrete an exogenous acid load beforehand, abnormal tubular function arising during fasting may have contributed to their acidosis.³

Our findings show that in obese fasting patients metabolic acidosis may develop rapidly and become unexpectedly severe. Moreover, failure to develop acidosis, or its sudden improvement during starvation treatment, is good evidence that the fast has been broken despite continued weight loss, ketonuria, and denial by the patient. Since underlying renal or pulmonary disease could aggravate acidosis, it would seem advisable to assess renal tubular and pulmonary ventilatory functions before fasting is commenced, and to assess acid-base status during treatment. It is now generally believed that the treatment of obesity by total starvation is safe, yet some of our patients seemed to find nibbling even safer.—We are, etc.,

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Difficult Case of Gout

SIR,—Gout may be difficult to diagnose, and in the case reported below the presenting history was totally misleading.

A 60-year-old, overweight labourer was referred to the orthopaedic clinic with a five-month history of recurring swelling over the metacarpophalangeal joint of his right middle finger. Though the swelling was episodic, it never subsided completely. At the clinic he described pain and swelling over the joint, which was slightly swollen and tender. There was no history of specific injury, but he performed very heavy labouring, breaking boulders with a hammer. X-ray examination revealed a foreign body, probably metal, lying in the soft tissues between the heads of the second and third metacarpals. It was thought that his symptoms were due to chronic infection arising from this foreign body.

Exploration of the dorsum of the hand was performed under general anaesthesia. A bursa and degenerate tissue were found over the metacarpophalangeal joint of the middle finger, and these were excised. The foreign body could not be found, and it was thought inadvisable to extend the exploration in the presence of peri-arthritis. Hydrocortisone was injected locally, and the wound was closed. The resected tissue was examined by the pathologist, who reported that the tissue was densely fibrotic but contained a few synovial clefts. There were small zones of fibroelastic activity and occasional giant cells. There was no haemosiderin deposition or calcification, and examination under polarized light was negative. The appearances were those of non-specific chronic tenosynovitis.

On the fourth postoperative day he reported with severe pain in his hand. The area around the wound was red and hot, but there was no evidence of sepsis. A plaster supporting splint was applied with marked improvement in symptoms.

At this stage the possibility of gout was considered, and the serum uric acid was found to be 11.2 mg./100 ml. In the presence of a normal blood-urea this was indicative of acute gout. The diagnosis was confirmed by subsequent detailed biochemical studies. X-ray examination three months after exploration showed degenerative arthritic changes in the second and third metacarpophalangeal and interphalangeal joints. The metallic foreign body was again demonstrated. His condition improved with colchicine and probenecid, though his grip did not return to normal.

X-ray examination in this case was quite misleading. A metallic foreign body was found, with no sign of joint damage in the initial films. In retrospect, this is not surprising, for other authors have reported similar findings.^{1,2} It may well be that repeated and unnoticed trauma influences the site of gouty arthritis.³ The high-protein

diet necessary for labourers is a further possible factor in precipitating attacks of gout in susceptible individuals.

Diagnosis is difficult when symptoms and signs are not typical. Initial investigations by radiology, biochemistry, and histology may all be negative in the early stages of gout.^{4,5} Serial investigations in such cases should be performed, and only persistently normal results, together with a failure to respond to uricosuric agents, should be allowed to discredit the clinical diagnosis. In this context patients must be warned that self-medication with salicylates will prevent the beneficial effect of probenecid. Both are uricosuric, but they act as competitors, and as a result neither is as effective in the presence of the other.

I wish to thank Dr. I. A. Anderson and Dr. W. B. Davis for biochemical and pathological details, respectively, and Mr. T. H. Norton for permission to publish.

—I am, etc.,

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Paracetamol Overdose and Liver Damage

SIR,—Paracetamol is widely used as a mild analgesic, and the few reported side-effects suggest a high margin of safety when used in therapeutic doses. The finding of liver necrosis in rats,¹ was followed by two reports of fatal hepatic necrosis in inmates of a mental institution,² a third fatality being mentioned by Thomson and Prescott.³

Case 1.—A 46-year-old male was admitted two hours after ingesting 75 paracetamol tablets. He had not received other medication and there was no history of liver or kidney disease. He was in good general condition, conscious, and apyrexial. The pulse was 80, and the blood pressure 130/90. There was no icterus, and the liver was not enlarged or tender. Gastric lavage was carried out without detectable product, and a diuresis was initiated with frusemide 40 mg. and 5% glucose by intravenous infusion.

On the second day he complained of right upper abdominal pain and vomited altered blood. He was icteric, and the urine output had fallen. The blood pressure varied from 120/70 to