any observer in the neonatal period to have a click should be treated with prophylactic abduction splintage.

(4) It should be routine practice to observe carefully the hips of any children who present with a congenital foot deformity, whether this be calcaneus or equinus, as often these children have associated hip dysplasia.

(5) We consider that some system of screening during a child's first 12 months of life should be established by public health and infant welfare clinics. We appreciate there may be administrative difficulties, but nevertheless the detection of an unstable hip at six months as compared with 16 months greatly influences treatment and prognosis.

May I stress that, notwithstanding the points raised, von Rosen and Barlow's pioneer work on early screening must receive full acknowledgement. Our concern now is to tighten the net further during the first 12 months of life.—I am, etc.,

ROBERT OWEN

Robert Jones and Agnes Hunt Orthopaedic Hospital, Oswestry, Salop

SIR.—I must congratulate Mr. Geoffrey Walker on his important and timely article on "Problems in the Early Recognition of Congenital Hip Dislocation" (17 July, p. 147).

Having had personal humbling experience of an eight-month-old child referred to me with frank dislocation of the hip, after finding out that at birth I had examined this child for a mild calcaneo valgus deformity of her foot, I am certain that there are two aetiological types of congenital hip dislocation. My colleague, Mr. Robert Owen, brought this to our notice.¹ In this area we have certainly seen cases present after six months who had been examined by skilled medical personnel at birth and found to have normal hips.

As Mr. Walker states, the memorandum published by the Ministry of Health in 1966 has led most people to believe that full hip examination in the neonate would eliminate any cases of congenital dislocation of hip presenting later. I am glad that Mr. Walker's paper has again brought to our notice that this is not completely so and that all cases with any suspicion of even a mild congenital foot deformity should be followed through until they are walking, to make certain that acetabular dysplasia is not present.—I am, etc.,

P. H. CORKERY

Prince Edward War Memorial Hospital, Rhyl

¹ Owen, R. *Journal of Bone and Joint Surgery*, 1968, 50B, 453.

Tuberous Sclerosis and the Lungs

SIR.—I would like to point out a misconception in your otherwise interesting and informative leading article on "Tuberous Sclerosis and the Lungs" (10 July, p. 64). Although various mesodermal tumours are seen in patients with this rare disease, it is not true that "Liposarcomata are almost exclusive to tuberous sclerosis." In neither the study of 103 cases of liposarcoma by Enzinger and Winslow in America¹ nor in

our own series of 85 cases from the Westminster Hospital² is the association of liposarcoma and tuberous sclerosis seen. These papers represent the largest series of liposarcomata on both sides of the Atlantic. Liposarcomata are extremely rare in childhood³ but the associated mesodermal tumours of tuberous sclerosis commonly present in children.

Liposarcoma is one of the most important of the soft tissue sarcomata both because it is one of the most common and because the chances of cure are often good with a five year survival of 64%. It may occur in association with tuberous sclerosis but it is much more commonly found without it.—I am, etc.,

MARGARET F. SPITTLE

Meyerstein Institute of Radiotherapy, Middlesex Hospital, London W.1

¹ Enzinger, F. M., and Winslow, D. J., *Virchows Archiv für pathologische Anatomie und Physiologie und für klinische Medizin*, 1962, 335, 367.
² Spittle, M. F., Newton K. A., and Mackenzie, D. H., *British Journal of Cancer*, 1970, 24, 696.
³ Kauffman, S. L., and Stout, A. P., *Cancer (Philadelphia)* 1959, 12, 912.

SIR.—In your recent leading article on tuberous sclerosis and the lung (10 July, p. 64) you state "Liposarcomata are almost exclusive to tuberous sclerosis."

These are uncommon tumours but neither my colleagues nor I have noticed or heard of any such an association. The most recent authoritative text on soft tissue tumours¹ states "they (liposarcomata) are not related to trauma or any other recognized aetiological factor." Other leading authorities on tumour pathology also fail to mention any association.

Your statement appears to be a quotation from a recent paper by Dwyer *et al.*² and as they make clear it refers to a paper on liposarcomata of the kidney by Fish and McLaughlin.³ Renal liposarcoma is an extraordinarily rare tumour and, in fact, the great majority of liposarcomata arise in other sites.¹ Tuberous sclerosis may well be associated with the renal liposarcomata³ but does not appear to be associated with extrarenal liposarcomata.

I trust you do not think this is too much of a pathologist's quibble. Your leading articles carry considerable authority and it may well be, as a result of the one on tuberous sclerosis, that the relatives of patients with these tumours are subjected to needless investigation of, and much anxiety about, the possibility of a concealed hereditary disease.—I am, etc.,

R. G. M. LETCHER

Bland-Sutton Institute of Pathology, Middlesex Hospital Medical School, London W.1

¹ *Atlas of Tumour Pathology*, Second Series, Fascicle I, 1967, p. 116.
² Dwyer, J. M., Hickie, J. B., and Garvan, J., *Quarterly Journal of Medicine*, 1971, 40, 115.
³ Fish, G. W., and McLaughlin, W. L., *Journal of Urology*, 1946, 55, 28.

Bullous Lesions in Poisoning

SIR.—We have read Dr. C. M. Ridley's paper on "Bullous Lesions in Nitrazepam Overdosage" (3 July, p. 28) with unusual interest. He has performed a valuable service in drawing attention to the variety of causes of these lesions in unconscious

patients and in concluding "that the bullae represent a reaction to pressure and hypoxia in the skin of the unconscious patient."

We note that his patient had bullae (is it no longer permissible to call them blisters?) on "the left side of the face, left hand and wrist, and left breast" (our italics). We found no difficulty in placing a subject in a position in which these parts were subject to considerable pressure (Fig.). From evidence we collected in four cases of CO or barbiturate poisoning that came under our care¹ because there had been ischaemic damage of muscle and nerve, as well as of skin we came to the conclusion that the ischaemic lesions occurred only where there was considerable

pressure. In this we were about 150 years behind the times; Napoleon's surgeon, Larrey, knew all about it. What puzzles us is that Dr. Ridley does not mention blistering over the left great trochanter; hence our question mark. But he did not, almost certainly because he could not, describe the position in which his patient was found. If it had been what we postulate, it was possible that the upper part of the body was lying on a hard surface and the pelvis on something soft, such as a pillow.

We strongly support his view that it is the combination of pressure and hypoxia that does the damage; the poison that causes unconsciousness is in no way specific.

We are indebted to the Institute of Orthopaedics for placing the facilities of its photographic department at our disposal.

—We are, etc.,

H. J. SEDDON
A. J. D. HOWSE

London W.1

¹ Howse, A. J. D., and Seddon, H. J., *British Medical Journal*, 1966, 1, 192.

SIR.—I read with great interest the article by Dr. C. M. Ridley on "Bullous Lesions in Nitrazepam Overdosage" (3 July, p. 28). This raises the question of the origin of these blisters and stimulates me to report a case of barbiturate blistering which I think may shed some light on the aetiology of these lesions.

A 50-year-old man took an overdose of about 25 200 mg amylobarbitone capsules and a few amitriptyline tablets on 5 June. He was admitted to hospital deeply unconscious and remained comatose for just over 24 hours after this. On admission to the psychiatric unit here on 8 June blisters surrounded by an erythematous margin were visible over the left acromion process, opposing aspects of the knees, and the inner aspects of the dorsa of the feet and on the lateral aspect of the left foot. By 5 July the blistering and ulceration had healed on the left acromial region and similar lesions on the outer aspect of the left foot were well advanced in healing. However, the ulcers which had been associated with the blistering on the opposing surfaces of knees and

feet still showed a large central necrotic zone with healing from the periphery proceeding very slowly (see Fig.).

Presumably, the ulcers on the left acromion and the left outer foot were directly related to the pressure of the full weight of the body. These lesions, however, do not appear to have been nearly as severe

as those on the opposing surfaces of the feet and knees where excessive sweating may have played an important role. It is well known that barbiturates are partly excreted in the sweat and there is a substantial literature quoted by Mandy and Ackerman¹ which shows that the maximum necrosis in barbiturate blistering is in the eccrine sweat epithelium. Beveridge² has stated that the eccrine sweat glands play a part in the formation of the blisters by excreting barbiturate, the concentration being increased by local blockage such as when two limbs or fingers are in contact. He points out that these are common sites of the bullae rather than the situations of maximal pressure.

The case here reported would tend to confirm that pressure plays some role in barbiturate blistering, but emphasizes that severe epidermal necrosis occurs particularly when pressure and excessive sweat concentration occur in conjunction with each other as in opposing surfaces of limbs which are in close and prolonged contact.

My thanks are due to the department of medical photography of the Oldham Hospitals Group for the photograph.
—I am, etc.,

N. BERLYNE

Oldham and District General Hospital,
Oldham, Lancs

¹ Mandy, S., and Ackerman, A. B., *Journal of the American Medical Association*, 1970, 213, 253.
² Beveridge, G. W., *Archives of Dermatology*, 1970, 101, 369.

SIR,—You recently published my paper on Acute Poisoning: Some Myths and Misconceptions (6 March, p. 519), in which I drew attention to the ease with which the contents of articles inadequately supported by laboratory evidence might enter the mythology of acute poisoning. A plea was made to editors and their expert advisers not to publish articles where the sole evidence for the drug ingested was unconfirmed statements by patients or relatives. There could hardly be a better example of this uncritical approach than the paper "Bullous Lesions in Nitrazepam Overdosage" (3 July, p. 28). The title firmly states that bullae occur in nitrazepam overdosage, yet the evidence for in-

criminating nitrazepam and excluding barbiturates is no more than vague supposition and conjecture. No mention is made of any laboratory testing for nitrazepam or other drugs.

The only supporting evidence to the occurrence of bullae in nitrazepam overdose is given as "R. Goulding (personal communication 1970) at the Poisons Reference Service of Guy's Hospital." In December 1969 I had a communication from Dr. R. Goulding stating that bullae could occur in nitrazepam overdosage. Inquiry as to the validity of this information revealed that other substances might have contributed to the lesions attributed to nitrazepam.

It is not as if nitrazepam cannot be identified in the laboratory. Surely positive laboratory identification of nitrazepam, and negative evidence in the blood or urine regarding barbiturates, methaqualone, meprobamate, glutethimide, opiates, tricyclic antidepressants, and carbon monoxide should have been adduced to substantiate what can now remain but a myth.—I am, etc.,

HENRY MATTHEW

Regional Poisoning Treatment Centre,
Royal Infirmary,
Edinburgh

Smoking, Lung Cancer, and Asthma

SIR,—I would like to draw the attention of readers to the possible relationship between smoking, bronchogenic carcinoma, and asthma, because a preliminary survey of a large number of asthma sufferers has suggested some quite fascinating findings on this subject.

After suggestions that certain types of new growth seem to be less prevalent in patients suffering from allergic diseases, an analysis of over 25,000 allergic patients in Australia was started to try to confirm or otherwise this postulate. This group included some 13,000 asthma sufferers of all types seen over the past 12 years.

Every asthmatic at the first consultation is informed in no uncertain terms that he must give up smoking because of the sensitivity of his respiratory tract. Practically all follow this advice if it is put to them firmly enough; in any event most know from experience that they just cannot smoke without bringing on an attack of wheezing. The number of patients with this disease who are smokers therefore, even to a moderate degree, is quite small. Perhaps this is one reason why so few asthma sufferers, even those with severe recurrent disease over many years, develop destructive lung lesions such as emphysema, as opposed to the chronic bronchitic type of patient, who often uses the excuse that he must continue smoking in order to help him cough up sputum.

The main point of this letter, however, is to record the extraordinary fact that the results of observations so far on these 13,000 true asthma sufferers have shown that only one developed primary bronchogenic carcinoma over this 12-year period, and that was a man who had been informed some five years previously that he just had to give up smoking his 30-odd cigarettes a day, but he did not do so.

These findings suggest two possibilities. Firstly, that patients with asthma may have some resistance to the development of new growth, perhaps as a result of their hyper-

sensitive state, or secondly, that patients who do not smoke or give up smoking, even if they have chronic chest diseases, will not develop primary bronchial cancer.

It is hoped to publish the results of this survey in greater detail in the near future, relating them to the number of years each patient has been followed up, etc. It must be stressed at this stage that these findings are preliminary. They would appear to be so significant, however, that they should really be brought to the attention of the medical profession at this early stage because they suggest that further studies of this nature could well be carried out in different areas on an even wider basis.

This letter is largely written because I would be most interested to hear of the opinions of experiences on this subject of any physicians who have had wide experience in the care of bronchial asthma and lung cancer.—I am, etc.,

R. MUNRO FORD

Adelaide,
South Australia

Glutaraldehyde as a Bactericidal Agent

SIR.—Glutaraldehyde, a dialdehyde, is of particular importance in the field of hospital disinfection because, unlike most other commonly used disinfectants, it possesses sporicidal activity. It is used extensively as a chemosterilizer for endoscopes, anaesthetic equipment, and urological and other surgical instruments.¹ A stock 2% solution, when activated with 0.3% sodium bicarbonate, has a limited shelf-life of two weeks. This deterioration in bactericidal activity is thought to be the result of aldehyde polymerization at alkaline pH, the monomer being considered as being the active form.²

Recently, in the course of our studies³ into the nature of the antibacterial action of this dialdehyde against washed suspensions of *Escherichia coli* N.C.T.C. 9001, it was necessary to use different grades of glutaraldehyde obtained from different sources. These grades were: Kodak (25% solution), Koch-Light (25%), Cidex (2%), Polaron (8%) and Sigma (50%). Kodak and Koch-Light are commercial solutions of general industrial application, Cidex is the chemosterilizer used in hospitals, Polaron is a vacuum-distilled preparation containing a high amount of the monomer, and the Sigma preparation is stored over barium carbonate to prevent polymerization. Each was diluted to a concentration of 0.01%, sodium bicarbonate added to give a concentration of 0.3%, and *E. coli* incorporated to give an initial count of 3×10^6 viable cells/ml. Samples were removed at intervals during subsequent incubation for 1 hour at room temperature and viable counts made by the pour-plate method; colonies were counted after incubation of the plates for 48 hours at 37°C.

Of the glutaraldehyde samples tested, the Kodak solution produced a complete kill after 10 minutes, Cidex and Koch-Light after 20 minutes, and Sigma after 30 minutes, whereas with the highly purified Polaron sample complete loss of viability was not produced even after a 40-minute contact period. The samples containing the largest amounts of polymeric glutaraldehyde are thus more bactericidal than the samples containing a higher proportion of the monomer.

Although these results refer to one organism only it is apparent that in order to improve the bactericidal efficiency and shelf