The reference he gave was to a précis of a lecture to the Association of Clinical Pathologists in the Journal of Clinical Pathology, but a very much more detailed report, amply illustrated in both colour and black and white, appeared in the British Journal of Surgery in 1962. —I am, etc.,

Eastbourne, Sussex. A. G. SHERA.

#### REFERENCES

<sup>1</sup> Shera, A. G., J. clin. Path., 1953, 6, 327.

<sup>2</sup> —— Brit. J. Surg., 1962, 50, 68.

## Obstetric Forceps or Vacuum Extractor?

SIR,-No doctor would wish to challenge the second sentence of the leading article on obstetric forceps or vacuum extractor (23 September, p. 753): "... the obstetrician must not only exercise his skill to deliver the baby from its obstetric dilemma but he must also protect the baby from any damage or delay caused by the operation itself.' therefore follows that, except in the very experienced hands of either a busy registrar or an active consultant, applying a vacuum extractor, either during the first stage of labour or even in the second stage where the position of the foetal skull is difficult to determine, must be considered as potentially hazardous and decidedly risky to the life or subsequent health of the foetus. Similarly there could be no objection to a widely experienced, active, and well-qualified generalpractitioner obstetrician undertaking this procedure, but of course only in a specialist obstetric unit where every facility is available for readily treating any complication.

The set of the controversy "obstetric forceps or vacuum extractor" in your correspondence columns clearly takes place in the second stage of labour. Presumably no doctor disputes the vacuum extractor's place in the management of delay late in the first stage of labour, and forceps should always be banned at this time. In the second stage there are two situations where instrumental assistance is needed.

If delay occurs in the mid-cavity of the pelvis it is always likely that the foetal skull is in the occipitoposterior or transverse position, such a malposition as is probably the very cause of the delay. Where assistance is needed, either for delay or foetal distress, and before the instrument of choice is applied, the position of the occiput must be accurately determined-that is, if the skull and brain are to be protected from any damage. The delivery is clearly potentially difficult, so in fairness to the child (and mother) only a very experienced obstetrician should be performing. The final decision as to instrument should depend on the obstetrician's experience, enthusiasm, and considered results with its practical use. I can well understand and admire the excellent results obtained by Mr. J. A. Chalmers and others (4 November, p. 292) with the vacuum extractor, but, on the other hand, I firmly suggest that the majority of specialists, at least in this country, would prefer to use the obstetric forceps in order to achieve their best results in these

A vast proportion of instrumental deliveries take place when the foetal skull has advanced beyond the mid-cavity and has now reached the pelvic floor; and again the indications

obstetric situations.

for their use are either foetal distress or delay in progress. It is in this group that bony disproportion is no longer a problem, and so instrumental damage to the skull and brain is unlikely, but only provided the requisite care with the application is undertaken-and this applies whatever instrument is used. It is such a case which a general-practitioner obstetrician might easily be coping with in his general-practitioner unit, and in this situation he would naturally opt for the instrument with which he is most familiar, and, perhaps even more important, the overriding consideration he would take in making his choice would be his wish for a delivery with the least possible delay. Surely, therefore, the majority of doctors would choose the obstetric forceps, because most of us would agree with Mr. W. H. Laird (4 November, p. 292) who says: "... most obstetricians would agree that forceps delivery is preferable to vacuum extraction in cases where rapid delivery is required in the second stage of labour when foetal distress occurs with the foetal head fully engaged, because there is no doubt that the application of, and delivery with, the vacuum extractor does take longer than forceps delivery." I suggest he should continue his argument to include the potential as well as the recognized foetal distress, and, in mentioning potential foetal distress, I naturally refer to the longer second stage of labour. It is interesting that the well-known advocate of the vacuum extractor, Mr. Chalmers, also finds that forceps delivery is quicker.

I cannot agree that the vacuum extractor should be used without any anaesthetic at all, but would consider that, as with any obstetric forceps delivery, in fairness to the comfort of the patient a local anaesthetic should be administered. Only in exceptional cases would a general anaesthetic be necessary

Finally, I could never condone the use of the vacuum extractor being applied to a foetal skull above the pelvic brim, even for a second twin.—I am, etc.,

DAVID BROWN.

Postgraduate Institute of
Obstetrics and Gynaecology,
St. John's Hospital,
Chelmsford, Essex.

### Alcoholism

SIR,—Thirty years ago three diseases which were little understood, feared, and therefore not talked about were tuberculosis, insanity, and alcoholism. Enormous strides have been made in conquering the first two, but there still remains the same frightened silence about alcoholism, although it is much better understood by experts if not by the general practitioner like myself. I should therefore like to support Dr. A. Spencer Paterson (23 September, p. 798), when he says that the medical attitude is of paramount importance.

The National Council on Alcoholism states that out of 34 million who drink only half a million are alcoholics, and therefore we need only bother about them. These figures are very misleading manifestly, for it implies that the very great numbers of heavy "respectable" drinkers who are inefficient and who cause unhappiness to their relatives are normal. No public health scheme to

combat disease has ever neglected prevention—as the National Council appears to be doing. If a man is going to fall over a cliff we grab hold of him rather than send for an ambulance to go to the foot of the cliff.

Prevention must be studied, and this means stopping not only direct advertising, which costs £23m., but also indirect advertising. It also means a fearless education of adolescents on the dangers of drink. Marty Mann's Primer on Alcoholism<sup>2</sup> should be compulsory reading, and there should be many more discussions in medical societies and journals.—I am, etc.,

London N.W.1.

J. J. MACSORLEY.

#### REFERENCES

<sup>1</sup> National Council on Alcoholism, 1967. London. <sup>2</sup> Mann, M., Primer on Alcoholism, 1952. London.

# Hypersensitivity Reactions to Phenylbutazone

SIR,—In view of the recent interest in acute leukaemia developing after phenylbutazone therapy, it seems worth while reporting two cases seen during the past 18 months, both of which developed a blood picture with features of glandular fever following phenylbutazone administration. In one of the recent reports Dr. A. Lawrence' described a similar case in which "many atypical mononuclears closely resembling glandular-fever cells were seen" in the blood.

A woman aged 53 suffering from rheumatoid arthritis was treated with phenylbutazone, 600 mg. daily for one week, followed by 500 mg. daily for two weeks. A generalized maculopapular rash and a sore throat then developed, and her temperature was 100-101° F. (37.8-38.3° C.). A large soft lymph gland in the left axilla was noted, but no lymphadenopathy elsewhere, and her spleen was not palpable. The results of blood counts are shown below.

211 6 9 7 E Day of Illness	W.B.C.	Neutrophils	Eosinophils	Lymphocytes
	/cu. m.m.	/cu. mm.	/cu. mm.	/cu. mm.
3 4 6 9 11 13	1,700 2,300 7,000 8,000 8,000 11,000 8,000	170 (10%) 40 ( 2%) 0 640 ( 8%) 1,840 (23%) 5,940 (54%) 4,720 (59%)	323 (19%) 437 (19%) 1,120 (16%) 2,960 (37%) 2,720 (34%) 2,200 (20%) 480 (6%)	1,207 (71%) 1,817 (79%) 5,880 (84%) 3,760 (47%) 2,960 (37%) 2,420 (22%) 2,480 (31%)

On days 3 and 4 a few of the lymphocytes were atypical, and some of these resembled glandular-fever cells. On day 6 about half the lymphocytes were atypical, and about a third of these had the character of glandular-fever cells. These atypical cells decreased in number and had disappeared from the blood by day 13. The haemoglobin level and platelet count were normal throughout the illness. Paul-Bunnell and toxoplasma dye tests were repeatedly negative. A sternal bone-marrow examination on day 7 showed no neutrophil myelocytes or leucocytes but an increase in promyelocytes and in eosinophil myelocytes and leucocytes. The increase in promyelocytes presumably represented a recovery phase rather than a maturation arrest, as neutrophil leucocytes reappeared and increased steadily in the peripheral blood shortly afterwards.

A woman aged 71 suffering from rheumatoid arthritis had been treated with phenylbutazone, 300 mg. daily for three weeks, when a general-

ized maculo-papular rash appeared, which was partly petechial on her legs. There was no lymphadenopathy and the spleen was not palpable. The results of blood counts are shown below.

Day of Illness	W.B.C.	Neutrophils	Eosinophils	Lymphocytes
	/cu. mm.	/cu. mm.	/cu. mm.	/cu. mm.
1	11,000	4,400 (40%)	1,210 (11%)	4,950 (45%)
3	17,000	5,780 (34%)	1,050 ( 5%)	10,030 (59%)
11	12,000	4,440 (37%)	3,480 (29%)	3,360 (28%)
24	9,000	6,390 (71%)	270 ( 3%)	1,890 (21%)

On days 1 and 3 nearly all the lymphocytes were atypical, and about half of these resembled glandular-fever cells. By day 11 the latter had disappeared and only an occasional lymphocyte was atypical. On day 24 the leucocyte picture was normal. The haemoglobin level and platelet count were constantly normal. Paul-Bunnell tests were repeatedly negative, and toxoplasma dye tests gave titres of 256 during and for three months after the illness.

It seems most unlikely that glandular-fever or toxoplasma infection was the cause of the illness in either case. Both infections are rare over the age of 50.23 Lymphadenopathy was not present, as it is invariably at some time in glandular fever<sup>2</sup> and in cases of toxoplasmosis with glandular-fever cells in the blood.3 A toxoplasma dye titre of 256, as in Case 2, is found in 1% of adults, and the titre in the second case did not rise during or after the illness. On the other hand, the skin rashes and the marked eosinophilia, and the agranulocytosis in the first case, strongly suggest that both patients suffered a reaction to the drug phenylbutazone, and that the glandular-fever cells in the blood were part of this reaction. This was the conclusion of Dr. Lawrence as regards his case, which showed an even greater lymphocytosis of 17,820/cu. mm. with many glandular-fever cells present and an eosinophilia of 1,080/cu. mm.

I wish to thank Drs. J. Laurie, E. A. Murray, and I. D. S. Cowie for the clinical details and permission to publish these cases, and Miss J. Hutchinson for assistance.

–I am, etc.,

J. G. SELWYN. Area Department of Haematology, Royal Infirmary, Dumfries.

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- Lawrence, A., Brit. med. J., 1960. 2, 1736.
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### **Elastic Band Injuries**

SIR.—Deliberate production of artifact ulcers by elastic bands around the lower third of the leg is described in the paper by Mr. I. D. Kitchin and others (22 April, p. 218) and in the letter of Dr. K. Dawson-Butterworth (20 May, p. 510). Accidental production of artifact ulcers by elastic bands around the fingers and around the arm is described by Dr. J. G. B. Thurston (6 May, p. 376) and Mr. J. P. Turney (13 May, p. 445), respectively.

A widow aged 77 presented in September 1967 with a deep circumferential ulcer of the lower third of the right leg and a chronic varicose ulcer distal to this. She reported that a tight bandage had been applied to the varicose ulcer; this was postulated as the cause of the circumferential ulcer. The day after admission to hospital an elastic band



was removed from the depths of the ulcer. On further questioning she admitted having used an elastic band to hold a dressing on the varicose ulcer some weeks before admission, and had been perplexed at its disappearance.

The photograph was taken by Mr. A. D. Ring 24 hours after removal of the band.

—I am, etc.,

GEOFFREY GLEW.

Christchurch Hospital, Christchurch, New Zealand.

# Nalidixic Acid and Intracranial Hypertension

SIR,—There have been letters in the B.M.J. this year describing intracranial hypertension in children treated with nalidixic acid (29 April, p. 310; 5 August, p. 370; and 9 September, p. 679).

We therefore think it of interest to report the case of a middle-aged woman, who in the third week of acute glomerulonephritis, while yet still febrile and slightly oedematous, but with good urine output and blood urea of 24 mg./100 ml., was given nalidixic acid in a dose of 1 g. four times a day for a superimposed coliform infection of the urine. At the time she was normotensive and had no papilloedema. Within 24 hours of commencement of the nalidixic acid she developed, in the night, a toxic confusional state of schizoid type with paranoid ideas and depersonalization. A day later the nalidixic acid was stopped and there was thereafter considerable improvement, although another week passed before she was completely normal. An E.E.G. at this time was essentially normal, but during the record the patient had a hallucinatory episode during which there was demonstrated an increase of theta activity, abolished by opening the eyes. A repeat E.E.G. at a later date was entirely normal, though it is probably significant that the frequency of the alpha rhythm showed an increase of one cycle per second.

It would seem possible that the administration of nalidixic acid during the oedematous phase of acute nephritis precipitated this psychosis. Such an occurrence bears a relation to the previous reports of intracranial hypertension, although in this case there was no direct evidence.-We are, etc.,

L. Kremer. M. WALTON. E. N. WARDLE.

Royal Victoria Infirmary, Newcastle upon Tyne.

# Goodpasture's Syndrome and Dialysis

SIR,—The following case history supports the use of peritoneal dialysis in Goodpasture's syndrome, as mentioned by Dr. J. F. Munro and others (14 October, p. 95).

A 36-year-old woman was admitted in August 1966 with pyrexia. She had previously enjoyed good health and had borne five children with no apparent medical complication. She had noticed haemoptysis for the previous two weeks.

A postero-anterior chest x-ray showed bilateral diffuse mottling in the mid and lower zones. Haemoglobin was 6 g./100 ml. and the R.B.C.s showed the features of an iron deficiency anaemia. There was no other site deficiency anaemia. There was no other site of blood loss. The W.B.C. was 9,600/cu. mm.; the E.S.R. 125 mm. in one hour, and the blood urea 175 mg./100 ml.; serum proteins were 4.6 g./100 ml. with an albumin of 2.9 and a globulin of 1.7. Lupus erythematosus and rheumatoid arthritis screening tests were negative. Her sputum contained many macrophages laden with haemosiderin but no mycobacterium. The urine contained a moderate amount of protein. R.B.C.s and casts could be seen on microscopy.

Despite her sex, these features are in keeping with Goodpasture's syndrome. She was started on prednisone. Over the ensuing days haemoptysis continued and she complained of increasing dyspnoea. Her urine output fell. blood urea slowly rose to 390 mg./100 ml., by which time she had become semi-conscious. Peritoneal dialysis was performed with dramatic subjective improvement and her urea fell to 222 mg./100 ml.

During the next two weeks she ate a low protein diet and her blood urea fell still further to 42 mg./100 ml. Haemoptysis ceased.

She then started to develop considerable oedema despite a reasonable fluid output and was prescribed frusemide. She commenced a normal diet. Average proteinuria was 1 g. per day, using Esbach's method at the time of discharge. Since that time she has remained in reasonable health up to August of this year, although her blood urea has slowly risen to 100 mg./100 ml. Episodes of haemoptysis have been infrequent.

The original peritoneal dialysis was lifesaving during a critical part of her illness. She has recently been readmitted complaining of dyspnoea. Her haemoglobin is 10 g./ 100 ml. The blood urea has risen to 300 mg./100 ml.-I am, etc.,

DEREK BAINBRIDGE. General Hospital, Burton-on-Trent.

# Cowpox and Paravaccinia

SIR,-I read your leading article "Cowpox and Paravaccinia" (November 11, p. 308) with great interest. How does the cow's udder become infected? What is the direct source of infection? Many country folk, and others, have quaint notions about this.