studied at this hospital the homosexual proportion of patients was 16·8%, that of penile warts in homosexuals (24) compared to those seen in heterosexuals (482) was only 5%.—I am, etc.,

MICHAEL WAUGH

Department of Venereology,
West London Hospital,
Charing Cross Group of Hospitals,
London W.6

1 Oriel, J. D., *British Journal of Venereal Diseases*, 1971, **47**, 373.

SIR,—In your leading article on "Condylomata Acuminata" (22 April, p. 179) you mention the treatment of soft anogenital warts with podophyllin 10-25% in spirit. The irritant effect of strong podophyllin on the normal skin is of course well known, and the older standard technique of using 20-30% in liquid paraffin or similar bases had some disastrous effects if precautions were not taken to remove the agent after four hours. Although protection of the surrounding skin with soft paraffin may help, severe balanitis is still liable to occur, especially in the uncircumcised.

For many years now I have been treating such warts with 3% podophyllin in propylene glycol. The main advantages of this preparation is that the treatment can be carried out daily (with an orange stick) by the patient himself. The only instructions that I give to the patient are to wash the area first with soap and water, to apply a simple dusting powder after the application, and to stop treatment temporarily if the area is too inflamed.

If the warts are still present after about six applications, they are almost certainly too hard and cautery or diathermy will be necessary.—I am, etc.,

R. W. B. SCUTT

R.N. Hospital,
Haslar,
Gosport, Hants

Chondromalacia Patellae

SIR,—In your leading article on "Chondromalacia Patellae" (15 April, p. 123) you discuss many of the problems of pathogenesis, diagnosis, and treatment of this condition, but you do not mention one important type of patello-femoral degeneration which may follow plaster treatment of lower limb fractures.

In assessing claims for damages following leg fractures I have been impressed with the frequency with which these patients subsequently complain of grating behind the patella. Such grating is not infrequently the most prominent complaint at the conclusion of treatment and it may be very difficult to estimate the significance of the symptoms. However, I am convinced that in the vast majority of cases this sequel is due to the effect of pressure of the plaster cast on the patella. The condition is therefore preventable. Prevention is a simple matter of cutting a window over the front of the patella thus allowing the patella to protrude through a hole in the plaster.

This type of plaster has been found to be more comfortable than the conventional plaster. Cutting the window is facilitated by first applying to the front of the knee a circular piece of orthopaedic felt 6 inches in diameter.

I have been so impressed with the effectiveness of this measure that I have not thought it justifiable to conduct a controlled trial. Such a trial would not be difficult in a large fracture unit, however.—I am, etc.,

A. W. FOWLER

Bridgend General Hospital,
Bridgend, Glam

Fat Embolism in Patients with Fractured Hips

SIR,—As one of the co-authors of the article on fatal fat embolism following replacement arthroplasty for transcervical fractures of the femur (Dr. G. A. Gresham and others, 12 June 1971, p. 617) I was interested to read Dr. I. Sevitt's article (29 April, p. 257). In the catchment area covered by our article, Moore's arthroplasty was not performed and no comparison was possible between the use of a stemmed prosthesis with and without the use of acrylic cement.

During the last 18 months I have had access to six further cases of fat embolism in the East Suffolk area, in patients who died after fractures of the upper end of the femur. All were women. One had an intertrochanteric fracture and the rest subcapital fractures. The former and one of the latter did not have sufficient fat in the lungs to cause death, although they were both aged 87. Another patient died three days after injury from massive fat embolism before operative treatment had been carried out. A fourth patient died within six hours of a Thompson arthroplasty using cement. The remaining two patients died from massive fat embolism after Moore's arthroplasty without the use of cement. Both operations were done through a posterior approach. One was a woman of 74 who was operated on 10 days after her injury and died within 9 hours of operation. The other was aged 90, operated on two days after an injury and died about 14 hours later. The amounts of fat in the lungs in both these patients were sufficient to have caused death.

These are only two instances of fatal fat embolism after an arthroplasty using a stemmed prosthesis without cement, but it does indicate that the complication exists. I believe that it will prove to be more common if the pathological changes are searched for routinely as the overall mortality rate after the various prosthetic procedures is very similar.[1]—I am, etc.,

D. ROSBOROUGH

Ipswich and East Suffolk Hospital,
Ipswich

1 Hunter, G. A., *British Journal of Surgery*, 1969, **56**, 229.

Total Replacement of the Hip

SIR.—Your leading article (22 April, p. 177) reflects the present enthusiasm for total hip replacement, and it is not surprising that an operation which results in up to 90% of patients being completely relieved of their symptoms[1] should be so acclaimed. However, we feel that some caution is necessary when contemplating reconstructive joint surgery involving the use of acrylic cements.

There have been many reports in the British literature[2-6] of acute hypotension and of fatalities associated with the application of methylmethacrylate cement during total hip replacement, and similar reports have now appeared from the U.S.A.[7 8] where the clinical use of this substance has only lately been allowed. A recent investigation[8] showed that the liquid component of the commercially available cements was the cause of the cardiovascular disturbance.

Transient hypotension was shown to be the main effect which was compensated by an increase in cardiac output. Those patients who have a fixed cardiac output are therefore specially at risk if methylmethacrylate cement is used. The liquid component consists of monomeric methylmethacrylate with small amounts of hydroquinone, dimethylparatoluidine, methanol, methacrylic acid, and water. Our recent (as yet unpublished) work demonstrated unequivocally that methylmethacrylate alone is responsible for the hypotension.

There is no place for a complacent attitude when considering those operations during which large amounts of this acrylic cement are applied to vascular bone surfaces, and it is to be hoped that an innocuous cement compound will be developed as an alternative to methylmethacrylate. Until then, as already stressed by Charnley and his colleagues,[9] the most cautious selection of patients is essential so that only those in whom the advantages of the procedure outweigh the dangers of its performance are submitted to operations using large amounts of bone cement.—We are, etc.,

RICHARD H. ELLIS
JAMES MULVEIN

Department of Anaesthesia,
St. Bartholomew's Hospital,
London E.C.1

1 Charnley, J., *Journal of Bone and Joint Surgery*, 1972, **54B**, 61.
2 Powell, J. N., McGrath, P. J., Lahiri, S. K., and Hill, P., *British Medical Journal*, 1970, **3**, 326.
3 Ling, R. S. M., and James, M. L., *British Medical Journal*, 1971, **2**, 404.
4 Thomas, T. A., Sutherland, I. C., and Waterhouse, T. D., *Anaesthesia*, 1971, **26**, 298.
5 Phillips, H., Cole, P. V., and Letting, A. W. F., *British Medical Journal*, 1971, **3**, 460.
6 Peebles, D. J., Ellis, R. H., Stride, S. D. K. S., and Simpson, B. R. J., *British Medical Journal*, 1972, **1**, 349.
7 Cohen, C. A., and Smith, T. C., *Anesthesiology*, 1971, **35**, 547.
8 Newens, A. F., and Volz, R. G., *Anesthesiology*, 1972, **36**, 298.
9 Charnley, J., Murphy, J. C. M., and Pitkeathly, D. A., *British Medical Journal*, 1971, **3**, 474.

ABO Blood Groups and Sex Ratio at Birth

SIR,—In a series of 264 ABO blood-grouped white newborn babies and their mothers reported in 1925 Hirszfeld and Zborowski[1] observed that the sex ratio—that is, the ratio of males to females—was higher for babies of mothers of blood group AB than for babies of mothers of the combined blood groups A, B, and O ($P<0.05$). They declared that "if this finding were confirmed it would have far-reaching theoretical significance," and in view of this it is interesting that, as the Table shows, the difference they observed obtains ($P<0.01$) in the aggregate, totalling 45,645 cases, of all 15 of the white series reported in 1924-72.[1-4] (This aggregate includes a personal series of 14,451 cases, of whom 5,612 were reported previously.[3]) Further, in a series reported in 1951 Sanghvi[2] observed that the sex ratio was higher for B babies of B mothers and O babies of O mothers than for A babies of A mothers, and the Table shows that the same difference obtains in the 1924-72 aggregate.

The Table shows also a new finding, in that the difference observed by Sanghvi is, in this aggregate, accompanied by a differ-

Sex Ratio of White Newborn Babies, by ABO Blood Group, in the Aggregate of the 15 Relevant Series of 1924-72[1-4]

Mothers' Group	Babies' Group	Number of Babies		
		Male	Female	M./F.
AB	A or B	824	647	1·27
	AB	141	135	1·04
A	O or AB or B	3496	3083	1·13
	A	5633	5560	1·01
O	A or B	3291	3194	1·03
	O	7520	6839	1·10
B	O or AB or A	1497	1467	1·02
	B	1243	1075	1·16
		23645	22000	1·07
AB Mothers		965	782	1·23
A + O + B Mothers		22680	21218	1·07
Mothers and Babies	Different	9108	8391	1·09
	The Same	14537	13609	1·07

ence which is reciprocal to it—that is, by a *lower* sex ratio for *non*-B babies of B mothers and *non*-O babies of O mothers than for non-A babies of A mothers. That is to say, the 1924-72 aggregate presents a clear-cut difference in sex ratio between the babies of two contrasted types of mother—namely, mothers whose babies are of the *same* ABO blood group as themselves, and those whose babies are of *another* group from themselves—the difference being significant for B mothers ($P<0.05$), O mothers ($P<0.05$), and A mothers ($P<0.0005$). A difference parallel to the one observed in A mothers is seen in AB mothers—it is evident in the Table, and is present even in Hirszfeld and Zborowski's series—but here the difference is *not* significant ($P>0.10$), whatever may eventuate when the aggregate is added to. A detailed account and discussion are being prepared in which the idea will be considered that the phenomena are the result of unknown effects of steroid hormones.

I am grateful to Professor D. F. Kerridge and Dr. H. B. M. Lewis for their stimulating comments.

—I am, etc.,

T. M. ALLAN

Blood Transfusion Centre,
Royal Infirmary,
Aberdeen

1 Hirszfeld, L., and Zborowski, H., *Klinische Wochenschrift*, 1925, **4**, 1152.
2 Sanghvi, L. D., *Nature*, 1951, **168**, 1077.
3 *Cited by* Allan, T. M., *British Medical Journal*, 1959, **1**, 553.
4 Krauss, A., and Zimmermann, B., *Zentralblatt für Gynäkologie*, 1970, **92**, 12.

Hypotension after Verapamil

SIR,—In view of Dr. M. E. Benaim's report of asystole after verapamil (15 April, p. 169) we should like to report another instance of an adverse reaction associated with the use of this drug.

A 46-year-old man in congestive cardiac failure resulting from fast atrial fibrillation probably due to viral myocarditis was given 10 mg verapamil intravenously over 30 seconds. Although his heart rate fell immediately to around 100 per minute from over 200 per minute, his systolic blood pressure diminished from 80 mm Hg to 50 mm Hg. This was accompanied by sweating and restlessness. Improvement took place over the next 30 minutes, his heart rate remaining at 100 per minute although still in atrial fibrillation. He was not digitalized at the time of receiving verapamil.

We should like to advise caution in the use of this drug in hypotensive patients.—We are, etc.,

E. F. VAUGHAN-NEIL
N. J. C. SNELL
G. BEVAN

Edgware General Hospital,
Edgware, Middx

Post-gastrectomy Acidity

SIR,—In the surgery of duodenal ulcer, there seems to be a good deal of confusion over what preoperative or postoperative characteristics of gastric secretion are grounds for expecting a good clinical result. In particular, it is often understandably assumed that a low maximal acid output after operation is a good guide. But in our experience, using the augmented histamine test, this is of no prognostic value.

In a few (30) patients in Oxford undergoing both the augmented histamine test, with and without vagal block, and an insulin test, before and after operation, the most valuable prognostic guide was the postoperative basal acid output.

It is perhaps worth noting also that the postoperative repetition of the augmented histamine test with and without vagal block (by hexamethonium) seemed as good as the insulin test in predicting the clinical result—the absence of reduction in maximal acid output by vagal block, presumably indicating adequate vagotomy. Both were, however, prognostically inferior to the basal acid output.

In all tests, any collection period from maximum half-hour to first two hours, and titration to either pH 3·5 or pH 7, seemed equally good. The main pitfalls, as usual, were in failing to recover all secretions, and we found a sump tube far superior to other varieties.—We are, etc.,

PAUL SNELL

Hôpital Protestant,
Dabou,
Republic of Ivory Coast, West Africa

G. J. DRAPER

Department of Social Medicine,
Oxford

Need for Continued Oral Therapy in Diabetes

SIR,—Dr. J. W. Todd raises the problem of the obese diabetic who does not sustain a restricted diet (29 April, p. 295). Since I do not subscribe to the doctrine of original sin and since I believe prolonged hyperglycaemia to be harmful, I prefer not to allow such patients to stew in their own syrup. I agree that either insulin or the sulphonylureas may lead to further obesity. Phenformin or metformin should be prescribed. The biguanides have the double virtue of reducing the blood sugar and of reducing the weight.—I am, etc.,

ARNOLD BLOOM

London W.1

Skin Sensitivity in Au-antigen Carriers

SIR,—Australia antigen (Au) was detected in 32 out of 413 children age 1-16 years investigated by the Ouchterlony double-diffusion technique (Table).[1] Only one Au-positive patient had symptoms of hepatitis; 17 had been transfused previously. The study was repeated two months later, and Au was present in samples from 25 children.

TABLE—*Australia Antigen in Children*

Disease	No. of Patients	Au-antigen positive
Acute lymphoblastic leukaemia	39	8 (8*)
Down's syndrome	24	3
Hodgkin's disease	10	6 (4*)
Lymphosarcoma	7	3 (3*)
Others (pneumonia, nephritis, asthma, rheumatic fever, diabetes mellitus)	333	12 (2*)
Total	413	32

* Transfused

In these 25 Au carriers the skin sensitivity to a chemical agent—dinitrochlorobenzene (D.N.C.B.)—was studied. The D.N.C.B.-sensitization procedure consisted of the application of 0·1 ml of a 1% acetone solution to a circular area 2 cm in diameter on the ventral surface of the forearm. After evaporation of the solvent the area was occluded by Band-aid for six days. Twenty-one days later the patients were retested with the same solution of D.N.C.B. Out of 25 tested only two Au-carriers developed delayed hypersensitivity to D.N.C.B. Control group consisting of 13 Au-negative children was similarly tested, and the D.N.C.B.-test was positive in all of them.

It seems from this that the delayed hypersensitivity type of reaction to a chemical contact sensitizing agent may be impaired in Au-carriers.—We are, etc.,

BOGUSLAW HALIKOWSKI
RYSZARD KORCZOWSKI
JANUSZ ZAJACZKOWSKI

Paediatric Institute,
Medical Academy,
Kraków
and
Rzeszów District Hospital,
Poland

1 Aisenberg A. C., *Journal of Clinical Investigation*, 1962, **41**, 1964.

Liver Injury

SIR,—Mr. L. H. Blumgart and Dr. T. Vajrabukka (15 January, p. 158) described liver injury in 20 cases, 17 of which were caused by traffic accidents. We present the case of an 11-year-old boy who was kicked in the abdomen by a donkey and brought to our hospital in a state of shock 14 hours after the injury. A plain upright film of the chest showed a bubble of gas under the right diaphragm.

At laparotomy, there was a foul odour as soon as the abdomen was opened. The liver was crushed and lacerated on the superolateral aspect (an area of about 10 × 5 × 5 cm). The peritoneal cavity was full of foul smelling dark blood. The gall bladder was distended but no bowel perforation was found. The liver was repaired by primary closure.

The material received for pathological examination consisted of about 5 g of necrotic brownish tissue fixed in formalin. Microscopic examination of haemotoxylin and eosin and reticulum-stained sections showed necrotic liver tissue with many cystic spaces. Gram stain (Fig.) demonstrated numerous Gram-positive bacilli diffusely scattered throughout the section. The approximate