was found that good chromosome pictures can be displayed on a 24-in. (61-cm.) television screen using a microscope with a built-in light source (Baker "patholette") and a $\times 80$ apochromatic objective. This procedure can be useful for teaching purposes.

Projecting an enlarged image from a chromosome negative on to a Thorn image-retaining panel² produced a well-defined orange-coloured luminescent image which increased in intensity the longer the plate was left under the enlarger. The image was retained for half an hour after the enlarger was switched off provided the voltage across the plate was maintained. This procedure may be of assistance in examining and classifying individual chromosomes.

I am indebted to Mr. J. Andrews and Mr. K. Cecil for preparing the chromosomes and buccal smears for examination at this hospital. Also to Miss J. Ishmael and Dr. R. M. Lawrence of Llandough Hospital for carrying out five chromosome analyses and for their help and encouragement in the establishment of a chromosome unit at this hospital.

—I am, etc.,

Llanfrehfa Grange Hospital,
Cwmbran, Mon.

T. S. DAVIES.

REFERENCES

- ¹ Sanderson, A. R., and Stewart, J. S. S., *Brit. med. J.*, 1961, 2, 1065.
- ² Henderson, A. S., *New Scientist*, 1962, 16, 686.

Oral Phosphates for Hyperparathyroidism

SIR,—In the Humphry Davy Rolleston lecture before the Royal College of Physicians, June, 1962 (December 1, 1962, p. 1419, and December 8, p. 1495), Dr. C. E. Dent discussed the feeding of phosphate as a form of medical treatment for primary hyperparathyroidism. Whereas we agree that the feeding of phosphorus can reverse many of the biochemical changes of hyperparathyroidism, we would like to re-emphasize the danger of such treatment. As Dr. Dent points out, metastatic calcification may occur. A more important consequence may be a decrease in renal function or a rapid worsening of renal function if it is already impaired. In two patients, who received 8 g. of neutral phosphate daily for two to three weeks, the glomerular filtration rate decreased from control levels of 86 and 55 ml. per min. to 68 and 35 ml. per min., respectively.¹ In a group of normal dogs, each of which received 9 g. of neutral phosphate daily for four to six weeks, glomerular filtration rate decreased 30 to 50% from control levels. Two months later the glomerular filtration rate was still depressed. Relatively large changes in glomerular filtration rate may occur without obvious changes in blood urea nitrogen or in serum creatinine. These findings raise the question as to whether it is ever safe to use oral phosphate in the treatment of conditions characterized by hypercalcaemia. Certainly, if phosphate is used at all, measurements of renal function should be performed at frequent intervals.—We are, etc.,

JOHN R. GILL.

Clinical Endocrinology Branch,
National Heart Institute,
Bethesda 14, Md., U.S.A.

DOROTHEA E. HELLMAN.
FREDERIC C. BARTTER.

REFERENCE

- ¹ Gill, J. R., and Bartter, F. C., *J. clin. Invest.*, 1961, 40, 716.

Treatment of Umbilical Hernia

SIR,—Your interesting annotation on the treatment of umbilical hernia (May 11, p. 1249) does not stress the importance of invagination of the umbilical hernia

before the application of strapping, which is so necessary for this procedure to be successful. Also there was no mention that the application of any padding or coin underneath the plaster defeats the object of the application and might indeed result in enlargement of the hernial orifice. It might also have been mentioned that transparent "sellotape" is very useful for fixing the invaginated umbilical hernia and causes less reaction in the skin than strapping, but needs more frequent application. The state of the skin and the hernia can easily be seen through the sellotape.

Personally, I think that strapping with invagination speeds the recovery of this condition in most cases and certainly stops the further distension of the hernial orifice that occurs when the baby cries and the hernia protrudes more obviously.

With regard to avoiding the surgical treatment of these herniae before the age of 4 to 6 years, may we truly hope that yet another unnecessary operation, so often involving traumatic separation of baby and mother in the most vulnerable years, be given up?—I am, etc.,

Kettering, Northants.

ROBERT WIGGLESWORTH.

Portal Hypertension following Cholecystectomy

SIR,—The frequency of gall-stones has been estimated at 15 to 20% of all adult subjects in this country. Of the operations performed on the gall-bladder and the common bile-duct 90% are for gall-stones or their complications, or for complications following cholecystectomy. It is not surprising that cholecystectomy is the second most commonly performed intra-abdominal operation, being exceeded only by appendicectomy. Maingot¹ has listed a formidable number of complications, one of which was portal hypertension secondary to biliary cirrhosis resulting from long-standing biliary obstruction by gall-stones.

I wish to draw attention to the fact that portal hypertension can develop as a result of portal-vein damage or constriction from fibrosis following cholecystectomy. This was stated by Milnes Walker² to be a rare occurrence, but in view of the increasing number of cholecystectomies and common bile-duct explorations it might become a not uncommon complication. The following case illustrates this point.

A woman, aged 63 years, was admitted to a hospital elsewhere with cholecystitis in June, 1955. Cholecystectomy was carried out. The common bile-duct at operation appeared normal and contained no palpable gall-stones. Therefore it was not explored. The patient made a satisfactory recovery.

Two months later she complained of episodes of colicky epigastric pains associated with vomiting, but no jaundice or fever. In May, 1956, she sought medical advice. An intravenous cholangiogram was carried out. This showed a gross dilatation of the common bile-duct due to stone. She was admitted to hospital for exploration of the common bile-duct. The liver-function test showed a mild degree of obstructive jaundice. At laparotomy a stone was removed from the common bile-duct and an operative cholangiogram was done. This revealed a free passage of the "dye" in the duodenum and there were no residual stones. On the fourth post-operative day she was noticed to have ascites. Abdominal paracentesis was carried out and 1½ litres of clear fluid was drained. After this she became completely symptom-free and there was no evidence of ascites.

In December, 1960, she was readmitted to hospital with haematemesis. This was treated by blood transfusion and she made a good recovery. Barium-meal examination at

that time revealed oesophageal varices. The liver and spleen were not palpable. She was subsequently readmitted twice in the following two years with recurrence of the haematemesis. On the last occasion she was noted to have an enlarged spleen, but the liver was impalpable. At all times the liver-function tests were normal. Surgical treatment was ruled out because of the prohibitive risk to the patient, since her cardio-respiratory reserve was extremely poor. She succumbed to the last attack of haematemesis.

Post-mortem examination revealed massive oesophageal varices, a normal liver, and a moderately enlarged spleen. The portal vein and the common bile-duct were constricted by fibrous adhesions. There was ante-mortem thrombus in the portal vein.

It seems reasonable that portal hypertension should be considered in all patients presenting with haematemesis having a previous history of such operations. The portal hypertension in these cases, if not caused by biliary cirrhosis, is likely to be caused by extra-hepatic portal-vein obstruction. Such obstruction may be a result of damage to the portal vein at the time of operation, or else peri-portal vein fibrosis resulting from infection and bile leakage. Since the liver is both functionally and histologically normal the prognosis in such cases after treatment should be better than in those with biliary cirrhosis or portal cirrhosis. Obviously porta-caval shunt is not feasible in such cases. Therefore a lienorenal anastomosis or porta-azygos disconnection is the operation of choice.

I wish to thank Mr. R. W. Doyle and Mr. C. R. Helsby for allowing me to publish this case.

—I am, etc.,

Royal Infirmary,
Liverpool.

K. EL RIFI.

REFERENCES

- ¹ Maingot, R., *Ann. roy. Coll. Surg. Engl.*, 1963, **32**, 42.
- ² Milnes Walker, R., *Pathology and Management of Portal Hypertension*, 1959. Arnold, London.

Menopausal Flushes

SIR,—I was interested by Dr. D. E. R. Kelsey's letter (May 11, p. 1290). Naturally, in dealing with neurotic patients, one encounters a very considerable number of women who, in the menopause, complain with considerable fervour of hot flushes. This symptom is very often no more intense than many others. There are, however, cases in which the intensity of the flushes appears to be more catastrophic and the patient's complaints about them persist after they are better as a whole. It would be interesting if one knew more about what autonomic and glandular factors are involved in these more fulminating cases. The hormone extracts as used at present are, for the most part, useless and may be dangerous.—I am, etc.,

Bath.

A. GUIRDHAM.

SIR,—Dr. Denys E. R. Kelsey (May 11, p. 1290) asks for information about research into the causation and mechanism of the very troublesome flushes associated with the menopause. My own investigations in this field started in 1950 when I also noticed what Dr. Kelsey describes so well—the immeasurable suffering caused by these symptoms and the apparent lack of concern by the profession.

The surprise came when I was forced to conclude the very opposite to what I had been taught as a student—and what now, according to Dr. Kelsey, is widely held—"that the basic pathology lies in the hypothalamus rather than in ovarian deficiency." Treatment based on this belief hardly ever failed to bring relief, usually full

though occasionally only partial, to the patient. These findings formed the basis of an essay subsequently published in the *Transactions of the Hunterian Society*.¹

Hormone-replacement therapy is rarely necessary in the treatment of the woman suffering from a difficult climacteric, nor indeed for that matter in gynaecological medicine at all. In another survey in my own practice I found that only 10 out of 178 women with gynaecological symptoms required hormone therapy.²

Those interested in this most important subject can do no better than refer to the works of Weiss and English³ and of Fritz Wengraf.⁴—I am, etc.,

London N.W.3.

PHILIP HOPKINS.

REFERENCES

- ¹ Hopkins, P., *Trans. Hunter. Soc.*, 1954/55, **13**, 90
- ² ———, *J. Coll. gen. Practit.*, 1959, **2**, 246.
- ³ Weiss, E., and English, O. S., *Psychosomatic Medicine*, 1957, 3rd ed. W. B. Saunders and Co., Philadelphia, London.
- ⁴ Wengraf, F., *Psychosomatic Approach to Gynaecology and Obstetrics*, 1953. C. C. Thomas, Springfield, Illinois.

Drugs for Parkinsonism

SIR,—I was interested to see Professor S. Alstead's article on the use of drugs in Parkinsonism¹ and also to see an article on the same subject in "To-day's Drugs" in the *Journal* (February 9, p. 385). For the past five years I have been working in a large Parkinson's Disease Research Clinic at Dudley Road Hospital, Birmingham. About 320 patients attended regularly.

All the drugs mentioned in the two articles, and many more, have been used or tried by us over the past five years. During 1962 we had 60 patients taking a preparation, biferidan ("akineton"). This drug is widely used in the U.S.A., Canada, and Europe, and we felt we should try it. The results of this trial are to be published in the next few weeks. In over half of the cases the findings were favourable, and indeed the patients continued to take akineton. We also used akineton in combination with other drugs mentioned in the articles.

I felt that doctors treating Parkinsonism would be interested to know that there was another useful preparation available.—I am, etc.,

Birmingham.

D. M. MUSTARD.

REFERENCE

- ¹ Alstead, S., *Prescribers' J.*, 1962, **2**, No. 6.

Wright Peak Flow Meter

SIR,—I was most interested to read of the work of Dr. Jean R. Nairn and Dr. R. S. McNeill (May 18, p. 1321) in adapting the Wright peak flow meter for recording inspiratory values. Work of a similar nature in a larger series has been in progress at the London Chest Hospital for about two years, and I look forward to seeing the final published results from that centre.

The simple test that so far seems to have escaped notice is the measurement of the forced inspiratory volume in one second (F.I.V.1). This can easily be performed at any clinic where there is a rotating drum spirometer. There is no need for any extra apparatus and the technique is easily learned and taught.

In my own series of cases (not yet published) the F.I.V.1 was normally a little less than the F.E.V.1. In emphysema it is considerably greater than the F.E.V.1 and in pulmonary fibrosis considerably less. This agrees closely with the findings of Nairn and McNeill.

It seems possible that there is a close correlation between the F.I.V.1 (or P.I.F.) and the lung compliance—provided that when doing the test there is no bony