weed the non-reactors out it is quite unnecessary to make them collect all their urine for three weeks. The induction of uterine bleeding 10 days to a fortnight after the treatment shows that there has been some response. Basal temperature recording will show in the majority of cases whether ovulation has taken place and may indeed be the earliest indication that conception has taken place. Serial vaginal cytology (vaginal smears taken daily or on alternate days) will give an accurate indication of the oestrogenic response, the induction of ovulation, and the post-ovulatory progesterogenic response. Women of even moderate mental capacity can be taught to take their own smears, and they can bring them up to the clinic once a week, once a fortnight, or even once a month. All that is needed is a vaginal cytology unit to interpret them. At present there are very few. There should be more. If there were a great deal of unnecessary routine steroid hormone estimations on 24-hour urine specimens would be avoided and steroid chemists would be able to get on with their research work.—I am, etc.,

PETER BISHOP.

Chelsea Hospital for Women,
London S.W.3.

Neutropenia Caused by Methyldopa

SIR,—So far as we are aware only two cases of neutropenia caused by methyldopa (Aldomet) have been recorded,^{1,2} and therefore we think it worth while to report a third.

A Persian doctor's wife aged 43 consulted us because of pain in the back. This was found to be due to osteoarthritis. In the course of routine examination she was found to have a blood pressure of 200/120 mm. Hg. The urine was normal in all respects. An intravenous pyelogram and a renal arteriogram showed nothing abnormal. Methyldopa and a low-calorie diet were prescribed. Her blood pressure fell satisfactorily. After 10 days her temperature rose to 102° F. (38.9° C.) and a white cell count showed a total of 2,000 with 800 neutrophils. Methyldopa was discontinued and ampicillin prescribed. Three days later the leucocytes were still 2,000, and the neutrophils had fallen to 460 per cu. mm. A marrow examination by Dr. M. J. L. Patterson showed the following results—Cellularity: reduced. Erythropoiesis: normoblastic and somewhat depressed. Granulopoiesis: markedly depressed. Megakaryocytes present and appear normal. Reticulum cells infrequent. Plasma cells normal. Mitoses reduced. Iron present in normal amounts.

Thereafter the white cell count improved. After a further five days the total leucocytes numbered 3,000 per cu. mm. with 1,290 neutrophils. By the end of three weeks the leucocyte count had risen to 4,000 and the neutrophil count to 2,160.

One difference between this case and Dr. K. G. A. Clark's¹ is that, whereas his showed active granulopoiesis to the metamyelocyte stage, in ours granulopoiesis was much depressed. There was no evidence of autoimmune haemolysis which methyldopa occasionally causes: the red cell osmotic fragility was normal, the serum bilirubin 0.4 mg./100 ml., and the direct antiglobulin test negative.—We are, etc.,

RAYMOND GREENE.

A. W. SPENCE.

London W.1.

REFERENCES

- Clark, K. G. A., *Brit. med. J.*, 1967, 4, 94.
- Hallwright, G. P., *N.Z. med. J.*, 1961, 60, 567.

Low Back Pain

SIR,—Although it is now commonly believed that defects in the neural arch (spondylolysis) are stress fractures rather than congenital in origin,^{1,2} it is quite misleading to suggest that such stress fractures of the pars interarticularis are common findings in patients with low back pain ("Medicine Today: Low Back Pain," 11 November, p. 341). Furthermore, it is rare to find a defect in the pars interarticularis which can be shown on serial x-rays to go on to unite. Perhaps this is because the forces acting on the lower lumbar vertebrae tend to separate the fracture surfaces.

Defects of the neural arch, with or without an associated forward slip (spondylolisthesis), are more commonly found in association with chronic low back pain, and 7.9% of a total of 4,411 patients from four series quoted by Rowe and Roche had defects demonstrable on x-ray. When symptoms occur they are the result of the mechanical weakness or lumbar instability³ resulting from the defect, and are the same as those seen in lumbar instability from any other cause, such as degenerative disease of the lumbar spine.⁴ When conservative treatment fails the logical treatment is spinal fusion⁴ to stabilize the unstable segment, a form of treatment not mentioned in your article.—I am, etc.,

A. W. F. LETTIN.

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

REFERENCES

- Rowe, G. G., and Roche, M. B., *J. Bone Jt Surg.*, 1953, 35A, 102.
- Stewart, T. D., *ibid.*, 1953, 35A, 937.
- Newman, P. H., in *Modern Trends in Diseases of the Vertebral Column*, 1959, p. 271, ed. H. J. Burrows and R. Nassim. London.
- Lettin, A. W. F., *J. Bone Jt Surg.*, 1967, 49B, 520.

SIR,—As I presume your "refresher" column on back troubles (11 November, p. 341) is intended to help general practitioners, I think it should be remarked that the clinical picture as described differs somewhat from that seen in general or industrial practice, where loss of the ankle jerk in sciatica occurs in a comparatively small minority of cases, and muscular weakness or wasting are excessively rare except following surgical intervention.

Further, the observed natural history of the syndrome leaves one in no doubt of the common origin of lumbago and sciatica, from intervertebral disc lesions, without the need for aetiological differentiation.

Lumbago is usually a mild condition, and, while one cannot doubt the wisdom of strict bed rest for a first attack in the slender hope that full healing may take place, when the condition becomes recurrent, with an admitted frequency of about two attacks a year, this advice is not only overcautious but quite impractical. In fact the majority of cases of lumbago improve most quickly with the earliest possible mobilization, and in what must be thousands of cases in industrial practice I have never once seen the threatened sequelae of urinary or other sinister complication.—I am, etc.,

W. I. D. SCOTT.

Chester.

Congenital Dislocation of the Hip

SIR,—Your leading article (18 November, p. 371) on the admirable study of hip-dislocation by Dr. H. V. L. Finlay and his colleagues (same issue, p. 377) raises a point which perhaps needs a brief comment. You state that congenital dislocation of the hip is congenital but is "not a genetic defect in the same class as the developmental errors like harelip, cleft palate, and absent or supernumerary fingers." In fact none of these conditions are entirely genetically determined, but there is an important degree of genetic predisposition for all of them. The evidence for this in twin and family studies is as good for congenital dislocation of the hip as for cleft lip and palate, and rather better than for most instances of absent or supernumerary fingers.¹ On the other hand, if you are using "genetic" not in the sense of determined by genes but in the older sense of developmental (timing and the class of morphological abnormality involved) your statement is of course valid.

The familial aggregation of congenital dislocation of the hip is of some practical importance. Even where all babies are screened for the condition it is worth, for example, examining a baby girl whose brother or father had congenital dislocation of the hip with special care; just as it is worth examining a little girl who is first-born and breech-born with special care.—I am, etc.,

M.R.C. Clinical Genetics
Research Unit,
Institute of Child Health,
London W.C.1.

C. O. CARTER.

REFERENCE

- Carter, C. O., in *Progress in Medical Genetics*, 1965, Vol. IV, ed. A. G. Steinberg and A. G. Bearn. New York.

SIR,—Dr. H. V. L. Finlay and others are to be congratulated on their study of congenital dislocation of the hip in the newborn infant (18 November, p. 377). They give a valuable description of Barlow's tests and rightly emphasize the importance of direct palpation of the head of the femur. Unfortunately your leading article tends to blur the distinction between the Barlow and Ortolani tests. The distinction between these tests is of vital importance.

In Ortolani's test¹ the knee is held with the hip flexed and the hip is then slowly abducted. In the dislocated hip reduction of the joint will occur with a palpable "jerk" or "jolt," which is usually visible. ("Click" is not a good translation of the Italian "sbalzo" and "scatto"). Ortolani emphasized that his test was practically identical with the Lorenz manoeuvre for reduction of congenital dislocation of the hip. It is therefore effective only if the hip is actually dislocated. For this reason it will not always detect congenital dislocation of the hip in the early stages when the hip is merely unstable or where the hip slides in and out of the acetabulum without a jerk or jolt. Ortolani did not in fact recommend the test in the first three months of life.

Barlow's test,² on the other hand, is designed specifically for use in the newborn and the first three months of life. The hip is held in a pincer grip between the thumb in front and the middle finger behind. In this way when the flexed hip is abducted pressure