to the benefit of the patient for whose treatment the consultant has final responsibility.

Today, with more money spent to improve the facilities of hospitals for mental handicap, more staff, a slowly falling inpatient population, and a growing community commitment, there are more exciting prospects than ever before for the doctor who is prepared to specialize in mental handicap to enjoy a varied, satisfying, and rewarding career.—I am, etc.,

D. A. SPENCER

Meanwood Park Hospital,
Leeds

¹ Royal Medico-Psychological Association, *British Journal of Psychiatry*, 1971, 119, 95.

Troubles with I.U.C.D.s

SIR,—Your leading article (7 April, p. 2) is timely in the light of the experience of the wife of one of us (D.M.H.) in respect of her intrauterine contraceptive device. Whether the complication is called extrusion or translocation, the postulated mechanism in this case and the history are interesting.

The patient was fitted with a Lippe's loop in February 1969 at her postnatal examination after the birth of her second child. At the examination the uterus had involuted normally but was retroverted. The I.U.C.D. was inserted by an experienced operator with the patient in the left lateral position. As she sat up she was aware of severe left shoulder-tip pain which lasted 24 hours. This was commented on, but no action was taken. The patient did not breast-feed her baby, and menstruation occurred normally in April. Further periods occurred in May and June but these were abnormal, there being only altered blood, and this was followed by amenorrhoea. Pregnancy tests were negative and a hysterosalpingogram confirmed the presence of the loop outside the uterus. At laparotomy (N.G.) in September 1970 the loop was removed from the pouch of Douglas. A further pregnancy then occurred resulting in the birth of a normal female child on 26 August 1971. Despite the previous episode, a second Lippe's loop was inserted at her postnatal examination on 14 October, when the uterus was again retroverted. The patient breast-fed her baby and menstruation did not occur. The loop was apparently normally placed in January and May of 1972, but symptoms of pregnancy recurred at the beginning of July. A male child was born normally on 13 February 1973 and the placenta was complete. The loop was not delivered and plain radiography suggested that it was again outside the uterus. On 26 March the I.U.C.D. was retrieved from the pouch of Douglas laparoscopically (N.G.). Inspection of the uterus revealed a small scarred area on the fundus in which the nylon strings were still embedded.

It seems reasonable to suppose that the first loop was embedded in the uterine wall at insertion and that the device went on to "extrude" into the peritoneal cavity. The shoulder-tip pain occurring at insertion suggests that perforation of the uterine wall occurred at that time. The second loop was known to be normally placed for six months after insertion and was then seen laparoscopically to have sought out the weak point in the uterine wall and made its way into the peritoneal cavity via that site in the fundus. The conclusion can be drawn that it may be unrealistic to insert a second Lippe's loop where a first has already perforated the uterus.—We are, etc.,

D. MICHAEL HUMPHREYS
NIGEL GARDNER

Exeter

SIR,—In your leading article (7 April, p. 2) you state that in order to minimize the risk of perforation or translocation "the cautious clinician will usually wait six weeks or longer after abortion or delivery before putting in an I.U.C.D." However, a W.H.O. scientific group reported that the risks of perforation or translocation "appear to be smallest if the I.U.D. is inserted immediately after delivery or three or more months post-partum."¹ Furthermore, studies suggest that the risk is highest between four and eight weeks after delivery.^{2,3} Thus the injunction to wait "six weeks or longer" before insertion may be counterproductive as it could result in insertions being performed during the period of maximum risk, and owing to the delay some women who might accept an early postpartum insertion will fail to return for an appointment.

Later in the same article you state that "it is difficult to be sure if ectopic pregnancy has a truly raised incidence when an I.U.C.D. is in place." The evidence suggests that the incidence of ectopic pregnancies per woman-month of exposure is in fact reduced.^{1,4} Studies which have indicated an increased incidence of ectopics usually estimate ectopic pregnancies per 1,000 normal or total pregnancies, but the I.U.C.D. protects a patient from uterine pregnancy to a greater extent than from tubal pregnancy. Thus the use of total pregnancies as the numerator of the incidence rate leads to a statistical artefact and a spurious increase in the incidence of ectopics per 1,000 pregnancies among I.U.C.D. users when compared with non-users. However, calculations of the incidence of ectopic pregnancies per woman-month of exposure avoid this pitfall and have shown that the number of ectopic pregnancies is possibly reduced by the I.U.C.D. but to a lesser extent than uterine pregnancies.—I am, etc.,

R. H. GRAY

London S.W.1

- ¹ World Health Organisation, *Intra-Uterine Device*, Technical Report Series, no. 397, Geneva, W.H.O., 1968.
- ² Ratnam, S. S., and Tow, S. H., in *Post-partum Intra-uterine Contraception in Singapore*, ed. D. Wolfers. Amsterdam, Excerpta Medica, 1970.
- ³ Tietze, C., *Co-operative Statistical Programme for the Evaluation of Intra-uterine Devices: Sixth Progress Report, Part I*. New York, National Committee on Maternal Health, Inc., 1965.
- ⁴ Kleinman, R. L., ed. *Intra-uterine Contraception*, London, International Planned Parenthood Federation, 1972.

An I.U.C.D. Dating from 1890

SIR,—Readers of the leading article "Troubles with I.U.C.D.s" (7 April, p. 2) may be interested by the following note, which was found recently in the course of removing old pathological specimens. It was written in elegant, minute script on a label glued to the bottom of the remains of a small mahogany and bevelled glass case that contained a 22-carat gold collar stud set in a decaying blue velvet mount. The note is dated May 1890. It is reproduced verbatim, but for the name of the doctor:

"This intra-uterine device, a golden collar stud, was inserted by an atheistical practitioner of St. Marylebone, ——— M.D., since decd., as the means to procure non-procreation. After the passing of some months a state of pyometra was induced, a mischance, with toxicaemia that quickly subsided when the device was extracted from the canal of the neck of the womb (whence it had near exulcerated itself), a

free flow of puriform foul matter from the parts relieving the Lady of all symptoms of her distress. The practr. had several times before undertaken this same manoeuvre without suffering of his patients. It was the larger aspect of the device that he would place uppermost in the canal, not as might be thought on consideration of the object, the sphaerule, which indeed should not be so closely constrained as the other. Some minims of Chloroform on a mask of cotton calmed the pavor lusus [apprehension] s.o.s. [if necessary].

The local situation following a pyometra—a collar-stud abscess?—might well have precluded a uterine pregnancy thereafter. It is likely that a device so disproportionate in relation to the dimensions of the cervical canal, and with a narrow edge, would cause local ulceration and a substantial risk of ascending uterine infection. The stud measures a centimetre in length and the same in the diameter of its "larger aspect;" the "sphaerule" is 0.4 cm in diameter. It is in one solid piece, everywhere smooth and rounded, but the edge of the base no more than 0.1 cm thick.—I am etc.,

W. ST. C. SYMMERS

Northwood, Middlesex

Serum Alkaline Phosphatase and Rickets

SIR,—Professor A. S. Truswell (17 March, p. 677) is, of course, quite right in pointing out that among the children in our survey (10 February, p. 324) there were more Asian girls than girls of other races with serum alkaline phosphatase levels above 30 K. A. units, but whether it is valid to consider these apart from the rest is not clear. Certainly there was no significant difference between Asian, white, and West Indian girls in the mean alkaline phosphatase levels. While there may well have been some effect of winter on these findings, the first 100 schoolchildren were in fact studied in October, and thereafter in groups of 50-70 through to March.

We appreciate and indeed had not overlooked the many points raised by Dr. R. J. Prescott and his colleagues (7 April, p. 47) and by Dr. T. C. B. Stamp and Miss Joan M. Round (14 April, p. 113), as they will realize on carefully reading our paper. The level of alkaline phosphatase above which children were followed up was chosen by inspection of the histogram of the first 150. As it happens, it was similar to that chosen by Holmes *et al.*,¹ in their review of Asian children in Rochdale. The lowest level of any of our schoolchildren with radiological rickets was 31 K.A. units. The results of follow-up provide some support to our belief that these higher levels are abnormal since 60 children in the survey with these raised levels, treated for 12 months with ergocalciferol, increased, on average, 3.62 cm more in height and 3.87 kg more in weight than 90 children not so treated. The difference was significant in both parameters whether compared with the whole control group or only with those who had had comparable raised alkaline phosphatase levels ($P < 0.0005$) and was independent of race.

The question as to what constitutes normal levels of alkaline phosphatase is not easy to answer. What is clear to us is that a log-normal distribution cannot be used without further evidence as to the 25-hydroxycholecalciferol levels, particularly in those children with higher values of alkaline phosphatase found in the survey reported by

Miss Round.² We await the estimations of vitamin D levels to be completed on random samples from children in our survey. Maybe we shall be shown to be wrong, but in view of the marked difference between the heights and weights of our treated children and the control groups and the correlation of vitamin D levels with serum alkaline phosphatase found in Asians by Dr. O'Riordan and his colleagues³ we doubt it. A full report on the effects of treatment and on alkaline phosphatase levels will be submitted for publication shortly.—We are, etc.,

W. T. COOKE
P. ASQUITH

- Holmes A. M., Enoch, B. A., Taylor, J. L., and Jones M. E., *Quarterly Journal of Medicine*, 1973, 42, 125.
- Round, J. M., *British Medical Journal*, 1972, 3, 114.
- Preece, M. A., et al., *Lancet*, 1973, 1, 907.

Erysipelothrix Septicaemia

SIR,—In answer to the question put by Drs. W. D. Alexander and C. S. Goodwin (31 March, p. 804) our patient never received steroids for her psoriasis and was never on any form of immunosuppressive drugs. She remains well and there is no obvious predisposing factor that we can discover.

We agree that we would perhaps have been wiser to be less dogmatic and to report that there was "no clinical evidence of endocarditis," but in the cases previously reported the endocarditis has been gross, with severe valve damage in every case, and we felt that endocardial involvement was unlikely in our patients.—We are, etc.,

R. H. TOWNSHEND
M. HASAN YEKTA
A. E. JEPHCOTT

Sheffield

Thrombotic Thrombocytopenic Purpura after Influenza Vaccination

SIR,—We wish to report a case of thrombotic thrombocytopenic purpura following influenza vaccination.

A 23-year-old Sikh nurse was vaccinated with Flenzavax (Wellcome). No immediate reaction was noted, but 14 days later she developed the first of numerous spontaneous bruises, and after a further seven days increasing pallor prompted a blood count, after which she was admitted to hospital. Examination revealed anaemia, numerous bruises, a positive Hess's test, and a blood pressure of 180/95 mm Hg, but no other abnormalities.

Investigations showed: haemoglobin 9.2 g/100 ml, reticulocytes 9%, white blood count 8,200/mm³, platelet count 39,000/mm³, and peripheral blood film showing the typical features of a microangiopathic haemolytic anaemia with numerous schistocytes and microspherocytes. Direct Coombs test negative. Bone marrow showed hyperplastic erythropoiesis, normal leucopoiesis, and normal numbers of mature megakaryocytes but with little platelet budding visible. Fibrinogen titre normal. Fibrinogen degradation products not increased. L.E. cells absent. Blood urea 34 mg/100 ml. Midstream urine contained few red cells, no white cells or casts; sterile. Total serum bilirubin 4.7 mg/100 ml. Serum electrolytes, serum alanine aminotransferase, alkaline phosphatase normal. Immunoglobulins: IgA 133 mg/100 ml, IgG 2,800 mg/100 ml, IgM 55 mg/100 ml, anti-nuclear factor, thyroid, mitochondrial, and smooth muscle antibodies negative. Pregnoticon

test negative. A chest x-ray and barium meal showed no abnormalities.

The clinical course is shown in the figure.

Despite several days of intravenous heparin the haemoglobin and platelet levels continued to fall, the red cell abnormalities became more striking, and the blood urea level rose to 68 mg/100 ml. On the eighth day the patient became stuporose incontinent with marked neck stiffness, but without focal neurological signs. Heparin was discontinued and prednisolone 100 mg daily commenced. Over the next three weeks the platelet count rose and the evidence of red cell damage on the blood film lessened. The blood urea returned to normal and all neurological abnormalities disappeared within one week of starting prednisolone. At follow-up the blood film, platelet count, and blood urea are normal and prednisolone has been discontinued. She is normotensive, and there are no clinical findings to suggest a collagen disorder or disseminated malignant disease.

The aetiology of thrombotic thrombocytopenic purpura and the related condition of the haemolytic-uraemic syndrome of childhood are unknown. One current view^{1,2} is that in certain individuals circulating soluble antigen-antibody complexes might be formed, possibly due to impaired responsiveness, following antigenic stimuli such as virus infections or vaccinations. These complexes can trigger platelet aggregation, disseminated intravascular coagulation, and possibly vascular endothelial damage, with consequent thrombocytopenia and "microangiopathic" haemolysis. The development of thrombotic thrombocytopenic purpura after influenza vaccination in our case, and after triple-antigen and T.A.B. vaccination in another,³ might be relevant to this hypothesis. Similarly, cases of the haemolytic-uraemic syndrome have been reported after triple, polio, measles, and smallpox vaccinations and viral infections.^{1,4}—We are, etc.,

R. C. BROWN
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E. A. FRENCH
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General Hospital,
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Curarizing Substance in Myasthenia Gravis

SIR,—Dr. Mary B. Walker (7 April, p. 42) recalls experiments carried out in the 1930s which strongly suggested that a curarizing substance produced in forearm muscles during contraction to the point of fatigue will increase the weakness of proximal muscles when the blood from the forearm is allowed to re-enter the circulation by deflating the cuffs previously constricting the arms. She mentions that these experiments "have been confirmed, though not by all observers." I have demonstrated this elegant experiment of Dr. Walker's on many occasions. The accompanying photographs show the dramatic increase of weakness in facial muscles and upper lids one minute after deflation of the arm cuffs in one of my patients. My experience confirms Dr. Walker's opinion that failure might be due to attempting the experiment on too mild a myasthenic or on one under the influence of neostigmine.

An observation very much in line with Dr. Walker's, although less neat, was made in 1929 at the Salpêtrière by Pierre Bourgeois. In his Paris thesis on myasthenia¹ he reproduces two photographs to illustrate what he calls "*l'expérience du ptosis*"

FIG. 1—Patient looking up before inflation of cuffs.

FIG. 2—Patient looking up one minute after release of cuffs.

provoqué." His patient, a middle-aged man, was asked to flex the fingers of his right hand repeatedly to the point of exhaustion. As fatigue came on the eyelids drooped progressively, the right eye developed a slight external squint, and the face assumed the myasthenic mask.—I am, etc.,

L. P. E. LAURENT

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¹ Bourgeois, P., *La Myasthénie*, Paris, N. Maloine, 1929.

Increased Dosage of Disodium Cromoglycate

SIR,—Observations on the safety of disodium cromoglycate in the long-term use in children failed to show evidence of significant toxic or allergic damage.¹ In the course of the study it was, however, noted that 13.4%