Correspondence

Letters to the Editor should not exceed 500 words.

Reference to the Coroner

SIR,—Dr. Gavin Thurston's forensic article (6 May, p. 361) contains welcome advice on how to reduce unnecessary references of deaths to this official. However, most of the deaths in hospitals that are reported to the coroner are at his desire and not the doctors? Some coroners require to be notified of patients dying from any of a long list of disorders, and no doubt this is one reason why the number of notifications to coroners is increasing.

The inference that reporting a case to the coroner increases the accuracy of death certification is not always justified. Coroners' pathologists are naturally interested mainly in medico-legal causes of death. Too many of their necropsy examinations are performed in ill-equipped mortuaries far from hospitals, where facilities for full pathological and histological investigations, such as are required to elucidate purely medical causes of death, are lacking.[1] Hospital doctors who should be seeing these necropsy examinations cannot do so, and valuable pathological material is lost.

The importance of postgraduate study for every member of the hospital staff is now generally recognized. Coroners could play a useful role by allowing more necropsy examinations to be performed in the hospitals where deaths take place, either by hospital pathologists (as is done in many areas) or, in particular cases, by pathologists selected by coroners. It can be said with confidence that medicine would benefit from hospital doctors watching the work of medico-legally experienced pathologists; and that the latter would gain from working in hospitals.—I am, etc.,

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REFERENCE

1 Coghill, N. F., *Lancet*, 1962, 2, 1053.

Selection of Women at Risk

SIR,—Recent publication of the findings of the Medical Research Council Committee on Oral Contraceptives (6 May, p. 355) has drawn attention to the risk of venous thrombosis and pulmonary embolism during their use. Though a precise measure of the risk is not yet feasible, a useful estimate can be made from the medical history and certain laboratory tests now found to correlate with the presence of venous thrombosis. It is proposed that estimation of risk as proposed herein permits the best judgement now possible in this matter. Furthermore, records of such data will be useful subsequently when morbidity and mortality statistics are reviewed. The incidence of venous thrombosis has been reported to be increased in the presence of cancer, heart disease, obesity, varices, gout, arthritis, collagen disorders, anaemia, and advanced age. Relative contraindications to the use of oral contraceptives may exist under these conditions, though this has not been clearly established. A positive contraindication exists for women who have previously had venous thrombosis or pulmonary embolism, and in the presence of stasis, such as elective surgery or serious injury. The general examination should devote special attention to the vasculature. Valuable baseline information can be obtained by recording the circumference of the lower leg at the level of the calf and ankle.

An abnormality in the blood characteristic of thromboembolism has been sought in patients with venous thrombosis and in patients having conditions associated with a risk of thrombosis. Numerous reports during the last three decades have raised hopes that one or more factors will be found to provide solid evidence to offset the uncertainty of clinical examination. Recently, multivariate analysis of a large number of different blood tests has indicated that information of significance for discriminating between patients with venous thrombosis and comparable control individuals is contained in the partial thromboplastin time of dilute plasma, the haematocrit, and the entrapment of platelets in a column of glass spheres. Ten other blood tests did not significantly improve discrimination.[1 2] No one of the three significant tests provided a better separation of individuals than was possible by calculating a discriminant function based on all of them.[3]

These laboratory methods have been used by me in the study of women using anovulatory hormones having symptoms referred to the lower extremities. Equivocal findings such as mild subjective tenderness of the calf or pain on dorsiflexion of the foot often left unreconciled doubt about the diagnosis. The use of anticoagulants "just to be safe" was in fact rarely necessary after considering the result of these blood tests. By comparison with the venous thrombosis series, these women were found to fall within the normal range of blood-test values. With this reassurance it has been possible to follow them expectantly, a decision justified by subsequent events. Prospective study of a small group of women before, during, and after use of oral contraceptives revealed shortening of the partial thromboplastin time during therapy in all cases to various degrees. In six the net change was less impressive during the first treatment cycle than when the drugs were stopped after several cycles. In only one of six was a significant change in platelet adhesiveness observed. A larger study was subsequently begun.

It now appears that positive contraindication to oral contraceptives exists if there has previously been venous thrombosis, or if stasis occurs, particularly with operation or accidental injury. I would also advise against their use by women who before treatment is begun are found to have blood-test results outside the normal range, and corresponding to the range characteristic of venous thrombosis. Further experience is needed to interpret the risk associated with a major change in the blood-test results during therapy, or by the combination of lesser changes in association with conditions listed above as relative contraindications.

As indicated in the report of the Medical Research Council, the physician prescribing oral contraceptives must weigh the risk in each case. It is proposed that an enlightened judgement is possible on the basis of the medical history, and that certain laboratory methods promise even greater assurance.—I am, etc.,

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REFERENCES

1 Hume, M., *Surgery*, 1966, **59**, 110.
2 —— *Ann. Surg.*, in press.
3 —— and Chen, Y.-K., *J. Amer. med. Ass.*, 1967, **200**, 747.

Varicose Veins, Oral Contraceptives, and Thromboembolism

SIR,—We wish to report the death from thromboembolism of a woman who had injections for varicose veins while she was also receiving norethynodrel and mestranol (Conovid E).

A married woman of 46 with two children, who had no history of thromboembolic disorders, presented with moderate varicose veins of typical long saphenous distribution. The right leg was treated first, using the injection/compression method of Fegan,[1 2] and a good clinical result was obtained. She later attended for treatment of her left leg, one week after she had been placed on a combination of norethynodrel and mestranol for a menstrual disorder. Treatment was by the usual Fegan technique, and a total of 10 ml. of sodium tetradecyl was injected. She collapsed and died 21 hours later.

At post mortem there were thromboemboli in both pulmonary arteries extending far out to the periphery. The left saphenous vein showed thrombus throughout its length extending right up to the sapheno-femoral opening. On histological section of the left saphenous vein it showed antemortem thrombus with no organization. Stains for fibrin (P.T.A.H. and acid-picro Mallory) showed little fibrin present, leading to the assumption that platelets were responsible for most of this thrombosis. Section of thrombi in the lungs showed a similar appearance with a lack of organization and a similar paucity of fibrin. Section of the veins in the right leg showed satisfactory sclerosis of the superficial and perforating veins down to the deep fascia. There was no evidence of deep thrombosis. The uterus was non-gravid.

Thromboembolic complications of the Fegan method have not previously been

reported, although about 20,000 patients have now been treated, many of them during pregnancy.[3] In the past we have treated patients who have been taking oral contraceptives, but we do not now feel that it is prudent to do so.—We are, etc.,

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REFERENCES

1 Fegan, W. G., *Lancet*, 1963, **2**, 109.
2 —— in *Modern Trends in Surgery (2)*, ed. Irvine, W. T., London, 1966.
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Chloasma and the Contraceptive Pill

SIR,—With increasing frequency in the use of contraceptive pills there is, as would be expected, an ever-increasing list of side-effects reported, which includes breakthrough bleeding, gastrointestinal symptoms such as nausea and vomiting, change in menstrual flow, change in libido, reduction in frequency and severity of dysmenorrhoea, weight gain, breast tenderness, headaches and dizziness, depression, leg pains and cramps, fatigue, nervousness, hirsutism, amenorrhoea, jaundice, moniliasis, chorea, and thromboembolic disorders.

To this list has recently been added adenoma of the breast and chloasma.[1] Very little reference is made to chloasma alone as a frequent complication. One survey involving 203 patients studied through 2,664 cycles of taking contraceptive pills reported only one patient—that is, 0.5%—with this condition.[2]

I have, however, seen six such cases in my practice in the three-month period August to October 1966, and suggest that this complication of contraceptive pills occurs more frequently than is commonly appreciated. All six patients were Caucasians permanently resident in the Tropics. Three of the six cases had recurrent chloasma uterinum, which cleared after delivery, except on the last occasion, when the pill method of contraception was commenced, and the pigmentation in each case then deepened. In the other three, pigmentation appeared for the first time three to six months after the pill was taken.

In three cases where the pill was abandoned, lightening of the chloasma became significant and progressive over this three-month period. No different effect was noticed in these six cases between one contraceptive pill and the other. With a larger selection of patients using different products one might be able to determine which products cause chloasma more readily, and form some opinion as to whether the progestogen or the oestrogen is the culpable hormone.

Armed with this knolwedge, the doctor is then in a position to prescribe to his susceptible patients the combination least likely to promote this particular side-effect.

I am grateful to the Ministry of Health, Trinidad and Tobago, for permission to publish this letter.

—I am, etc.,

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REFERENCES

1 Gregg, W. I., *New Engl. J. Med.*, 1966, **274**, 1432.
2 Behrman, S. J., *Obstet. and Gynec.*, 1964, **24**, 101.

Contraceptive Failure Rates

SIR,—In your leading article (6 May, p. 327) you comment (apropos the M.R.C. report on oral contraception): "It is generally accepted that the overall failure rate of other methods of contraception is about 10% per woman year, and it might be estimated therefore that about 80,000 of these women would have become pregnant."

The use-effectiveness of contraceptive methods is usually expressed in terms of pregnancies per 100 woman years of exposure, and on this basis the failure rates of traditional forms of contraception vary from 2.0 to 3.5 for the condom and I.U.D.[1] to 12.5 to 14.6[2] for the diaphragm and chemical spermicides. On this basis very few of the 800,000 women would have become pregnant during a single year.—I am, etc.,

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REFERENCES

1 Peel, J., *Med. Offr*, 1966, **116**, 357.
2 International Planned Parenthood Federation, *Medical Handbook*, 1965.

Preventing Pressure Sores

SIR,—Dr. Mary Bliss (18 March, p. 697) states that in her experience established infection does not occur in pressure sores. This is contrary to the findings at the Liverpool Regional Paraplegic Centre. Patients admitted with sores being either totally or partially anaesthetic over the whole body are unable to volunteer statements as to whether the sores are painful or throbbing. It is consequently necessary to pay attention to the systemic effects of infection. These are loss of appetite, apathy, and toxic confusional and psychotic states due to absorption of pus from the pressure sores. Laboratory investigations reveal a persistent anaemia, leucocytosis, and a depression of the serum albumin, sometimes with elevation of the serum globulin. When the sores have healed the general signs of infection disappear, the patient's morale, appetite, and haemoglobin improve, but it often takes a considerable time.

It would seem on reading the original article (18 February, p. 934) that Dr. Mary Bliss made similar observations, since she states, "that recovery of the sores with treatment was associated not only in the patient's sense of well being, but often with apparently real improvement in the patient's general condition also." Further confirmation of the severe systemic effects of pressure sores may be obtained by studying Dr. Tribe's observations[1] on 150 necropsies performed on paraplegic patients at Stoke Mandeville Hospital. Forty-eight of these patients had severe amyloidosis of their kidneys, and this was largely attributable to chronic pressure sores with underlying bony infection.

Unfortunately while no sore will heal while there is persistent pressure, removal of the pressure alone will not heal all bedsores. Some of the factors that prevent sores healing despite removal of pressure have been enumerated by Guttmann.[2] These are underlying osteomyelitis, shearing stresses due to spasms, and poor general condition of the patient.

A sore at the greater trochanter with severe underlying osteomyelitis is a particularly difficult problem, since the infection may track and involve the pelvis. Plastic surgery has little to offer when there is such infection, and such a sore is best treated by an orthopaedic surgeon, since an extensive dissection of the hip and the upper one-third of the femur is involved.

Antibiotics have been found to be of value in the treatment of pressure sores at two distinct stages of their treatment. Initially on admission when the patient is toxic and febrile he is placed upon a combination of cloxacillin and ampicillin. While the surgeon carries out the debridement of the sore and opens up all the closed pockets of infection, the use of systemic antibiotics localizes the infection to the sore, just as penicillin is used when a patient with valvular disease of the heart has a dental extraction. At a later stage when grafts are applied to a granulating area the sensitivity of the organisms in the sore is first determined prior to grafting and then the combination of systemic and local antibiotics appears to facilitate the take of the grafts, since some organisms, particularly the staphylococcus, are particularly liable to destroy skin grafts.

The best way of reducing cross-infection and preventing the emergence of resistant organisms is controversial, but it is undeniable that preventing pressure sores occurring and healing as expeditiously as possible those existing sores is a step in the right direction.—I am, etc.,

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REFERENCES

1 Tribe, C. R., *Paraplegia*, 1963, **1**, 19.
2 Guttmann, L., *Brit. J. plast. Surg.*, 1955, **8**, 196.

Iliac Vein Compression

SIR,—In their paper (1 April, p. 14) on 57 patients with ilio-femoral thrombosis, over half of whom also had varicose veins, Mr. F. B. Cockett and his colleagues state that "the main aetiological factor in these cases is shown to be a compression stenosis of the left common iliac vein by the overriding right common iliac artery."

I suggest the central weakness in this contention is that it implies the body to be incorrectly constructed at this point and that it does not account for the rarity of these venous conditions in peoples still living primitively. I have an alternative view, which is carefully developed elsewhere,[1] and have pointed out that these venous troubles, and their preponderance in the left leg, are related to a colonic cause. It is unfortunate that in 24 references the authors do not include a single one showing the rarity of these venous complaints in peoples still subsisting on an unrefined diet, as in the native reserves of South Africa, though such complaints readily occur in them directly they move on to sophisticated foods. It is indeed a tragedy that the remarkable figures available from these reserves[2 3] are so little known and appreciated in this country.

It is easy to see how the iliac artery might exert some influence on a distended thrombosed iliac vein, limiting the spread of a thrombosis from below, but this is very different from its exerting any influence on a healthy vein, and still less from its being the cause of an original thrombosis. The authors note that 18.5% of their post-thrombotic obstructions were localized in the region