non-pregnant cervix in vitro have a tendency to contract in response to $PGF_{2\alpha}$ and to relax with PGE_2.[9] It is therefore possible that PGE_2 is less likely to cause or allow such damage to occur, though it must be said that these experiments have not been performed using strips of pregnancy cervix in vitro, nor have such observations been made in vivo. That cervicovaginal fistulae have also been reported following abortion induced with intra-amniotic hypertonic saline[10 11] and intra-amniotic hypertonic urea[12] suggests that the primary abortifacient agent is not a contributory factor. The relationship of route of administration and type of prostaglandin used is therefore equivocal. Similarly the incidence of fistulae does not appear to be related to short abortion times[1-4] and by inference excessive uterine activity per se. Before this can be conclusively stated, however, individual intrauterine pressure records will require critical analysis.

The problem probably lies in developing levels of uterine contractility in the presence of an inadequately dilating cervix. The existing hormone balance may be of importance. It has been shown in sheep at term that induced uterine contractility does not produce cervical dilatation if progesterone levels are maintained.[13] In prostaglandin and saline terminations in the human the progesterone level declines during the contractile phase, though in some cases the significant decline does not occur until shortly before abortion.[14] It is possible therefore that if hormonal balance does not favour cervical effacement and dilatation in some individuals cervical fistulae may result.—We are, etc.,

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1 Shearman, R., Smith, I., and Korda, A., *Journal of Reproductive Medicine*, 1972, **9**, 448.
2 Wentz, A. C., Thompson, B. H., and King, T. M. *American Journal of Obstetrics and Gynecology*, 1973, **115**, 1107.
3 Kajanoja, P., *et al.*, *Journal of Obstetrics and Gynaecology of the British Commonwealth*, 1974, **81**, 242.
4 Lowensohn, R., and Ballard, C. A., *American Journal of Obstetrics and Gynecology*, 1974, **119**, 1057.
5 Brenner, W. E., *et al.*, *American Journal of Obstetrics and Gynecology*, 1973, **116**, 923.
6 Corson, S. L., Bolognese, R. J., and Merola, J., *American Journal of Obstetrics and Gynecology*, 1973, **117**, 27.
7 Craft, I., Walker, E., and Youssefnejadian, E., *Prostaglandins*, 1974, **5**, 397.
8 MacKenzie, I. Z., Hillier, K., and Embrey, M. P., *British Medical Journal*. In press.
9 Najak, Z., Hillier, K., and Karim, S. M. M., *Journal of Obstetrics and Gynaecology of the British Commonwealth*, 1970, **77**, 701.
10 Skajaa, T., *Acta Obstetrica et Gynaecologica Scandinavica*, 1961, **40**, 68.
11 Goodlin, R., *Obstetrics and Gynecology*, 1972, **40**, 82.
12 Bradley-Watson, P. J., Beard, R. J., and Craft, I. L., *Journal of Obstetrics and Gynaecology of the British Commonwealth*, 1973, **80**, 284.
13 Liggins, G. C., *Memoirs of the Society of Endocrinology*, 1973, **20**, 119.
14 Souka, A. R., Hillier, K., unpublished data.

Renal Involvement in Henoch-Schönlein Purpura

SIR,—It emerged from Dr. J. S. Cameron's interesting article on glomerulonephritis[1] that the systemic condition associated most commonly with acute nephritis was Henoch-Schönlein purpura. We have since studied the records of all the children suffering from Henoch-Schönlein purpura admitted to paediatric departments in hospitals in Copenhagen during the period 1960-9 inclusive. Out of a total of 203 children 60 (30%) had primary renal involvement with haematuria of at least three erythrocytes per microscopic field in one portion of centrifuged urine. Thirty-three of the 60 children were boys and 27 girls. The youngest was 12 months and the oldest 14 years of age (average age 6 years).

We followed up, for periods of from six months to 11 years after first admission, the children with Henoch-Schönlein purpura and got reliable information about 50 out of the 60 with primary renal involvement. One child died two months after the onset of the disease because of rapidly progressive glomerulonephritis. Eight children developed chronic glomerulonephritis, which in six was verified by biopsy and the two remaining patients had proteinuria and haematuria for more than four years. However, 30 children without primary renal involvement were all healthy at the time of follow-up. Chronic glomerulonephritis developed in 4% of the children with Henoch-Schönlein purpura and in 18% of those with primary renal involvement.

We agree with Dr. Cameron that the histological picture seen on renal biopsy varies from the mildest proliferation to very severe, rapidly progressive nephritis with crescent formation, but usually the histological changes affect the kidney patchily. —We are, etc.,

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1 Cameron, J. S., *British Medical Journal*, 1970, **4**, 285.

Screening for Sickle-cell Disease

SIR,—Dr. D. I. K. Evans (19 October, p. 162) has some reservations about the random screening of British populations for sickle-cell haemoglobin. While I do not dispute that there are many more cases of sickle-cell trait than sickle-cell disease I still believe that sickle-cell disease is not so very uncommon. Over a period of one year (1968-9) in Sheffield 73 patients considered at risk from a haemoglobinopathy were screened. Eight had positive sickle-cell tests, one of whom had the homozygous condition, sickle-cell disease. In subsequent years we have detected more cases of sickle-cell disease. There is also evidence that sickle-cell trait is not always the benign condition it is generally thought to be.[1] The percentage of sickle-cell haemoglobin in the case of sickle-cell trait varies, and may have a direct bearing on the degree of hypoxia which will be tolerated by these patients before sickling occurs.[2]

Dr. Evans places a faith in the "quick gas" in the dental chair for cases of sickle-cell trait which is not shared by others who are engaged in dental anaesthesia. The very nature of outpatient dental anaesthesia, which involves the sharing of an airway, makes it impossible to guarantee full oxygenation in every anaesthetic.[2 3] Severe hypoxia can occur even in the best hands.[4] K. A. Oduro[5] believes that "fatalities in patients with sickle-cell trait tend to occur in the short and minor surgery, where the usual 'quick whiff' is given or anaesthesia is by an intravenous induction agent alone."

I believe that identification of patients with both sickle-cell trait and sickle-cell disease is important and that the quick gas is not without risk in patients with sickle-cell trait.—I am, etc.,

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1 Dalal, F. Y., *et al.*, *British Journal of Anaesthesia*, 1974, **46**, 387.
2 Howells, T. H., and Huntsman, R. G., *Anaesthesia*, 1973, **28**, 339.
3 Young, T. M., *Anaesthesia*, 1973, **28**, 707.
4 Young, T. M., *Anaesthesia*, 1974, **29**, 614.
5 Oduro, K. A., *British Journal of Anaesthesia*, 1973, **45**, 123.

SIR,—Dr. D. I. K. Evans (19 October, p. 162) raises what appears to be a valid reservation over the need for routine sickle screening of susceptible patients before general anaesthesia for dentistry. He states: "The dangers of anaesthesia for major surgery in sickle-cell trait are well recognized, but is there any danger in the customary dental anaesthetic?" I am not clear what a "customary dental anaesthetic" is but, in common with many anaesthetists, I am wary of the "quick gas" Dr. Evans refers to. Certainly the traditional gaseous anaesthetic which was rapidly induced depended on an element of hypoxia for its effectiveness. Indeed, the method is still practised widely. A most "potent" variant of the method was that using secondary saturation by nitrous oxide, which was colloquially termed "black gas anaesthesia." Despite the ethnic connotation, these techniques are unsuitable for both the pigmented and Caucasian subject.

The management of anaesthesia for the negro dental outpatient, even when elective hypoxia techniques would not be used, includes very careful airway control, because the airways are under constant challenge by the operating dentist and appreciation of inadvertent hypoxia is difficult. To proceed with general anaesthesia in this case without knowing the sickling status of the patients seems to me quite cavalier.

I cannot accept the hypothesis that screening may do more harm than good. It could be argued that the caution taken by the anaesthetist when sickle-cell haemoglobin has been identified in his patient may be unjustified (though I doubt it), but this criticism is one of a clinical judgement and not one of an investigation. (A doctor who measures his patient's blood pressure and, finding hypertension, causes more trouble and inconvenience by treating it rather than leaving the hypertension does not create an argument for abandoning the practice of taking the blood pressure.)

In another letter (19 October, p. 163) Dr. F. F. Casale asks "should the absence of a sickle-cell test in fit and non-anaemic adults and older children of possible Negro descent be a contraindication to general anaesthesia?" The logical answer, I concede, is no. However, while assessing patient fitness and taking a blood sample for haemoglobin estimation (apart from other investigations) —and I assume these assessments are not being made just by looking at the patient—then I would answer that the absence, or omission, of a simple sickle screen test is a contraindication to proceeding with general anaesthesia. That this may not be the