The discussions seem to revolve around the degree of harm caused by the practice, whether it is sufficiently serious to treat, and, if so, at what age.

My own statistical analysis of thumb-sucking amongst a group of 500 children[1] convinces me that the effect on the morphology and development of the basal bone of the jaws is nil, and that it is limited to the teeth and supporting alveolar bone. Its effect on the surrounding soft tissues is nil and on the digits themselves extremely rare.[2]

Protruding maxillary incisors may be associated with thumb-sucking, but for every one of these cases there are nine or ten others with protrusion and no thumb-sucking. The only malocclusion which can be attributed solely to this habit is the anterior open bite, and even here the tongue seems to play a significant role.

Any irregularity which can be attributed to thumb-sucking is temporary and improves either spontaneously when the habit ceases or can be corrected with simple orthodontic appliance treatment.

The ultimate harm to the child is therefore nil, but the greatest harm appears to be suffered by the parents (and over-solicitous medical or dental advisers), their pride being hurt by what is unfortunately regarded as an antisocial habit.

So inherently strong is the sucking impulse in a child that enforced attempts at correction accompanied by scolding and bribing nearly always create an undeserved sense of guilt from which the child will shrink and attempt to find solace (now secretively) in the very habit for which control is attempted.

I would urge, therefore, that orthodontic treatment be instituted only for the correction of the underlying malocclusion, and the optimum age for this is approximately 10–11 years, or occasionally a little earlier in the mixed dentition.

Appliance treatment earlier than this is of very doubtful value, and if any treatment is necessary at all it should be limited to encouragement of the child and removal of any suggestion of guilt.

The parents (and school-teachers) should be reassured and advised to ignore a relatively unimportant habit, as once they become obsessed with its correction the services of a child psychiatrist may be required in addition to those of an orthodontist.

Surely a wet thumb is preferable to a wet bed!—I am, etc.,

Watford, Herts. H. L. LEECH.

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Autotransfusion and Rupture of the Spleen

SIR,—It is said that autotransfusion of blood in cases of rupture of the spleen is dangerous, probably because a number of these cases occur in diseased spleens. We should like to report a case in which autotransfusion was successful.

A 31-year-old man, a known "bleeder," was admitted exsanguinated from a delayed traumatic rupture of the spleen. He had been admitted to hospital on a number of previous occasions with persistent bleeding from wounds, tooth sockets, etc., and there is a definite family history of bleeding. On his admission there was no compatible blood available, and with an unrecordable blood-pressure his condition warranted immediate surgery. At laparotomy a ruptured spleen was removed and six pints of blood autotransfused. His condition improved immediately after operation, but after six hours he again collapsed and did not improve after a transfusion with two pints of fresh blood. A further intraperitoneal haemorrhage was diagnosed and a second laparotomy performed. The only abnormality found was a persistent ooze from the splenic bed. This was dealt with by diathermy, and a further five pints of intraperitoneal blood autotransfused. All autotransfused blood was collected in citrated bottles, and 10 ml. of 10% calcium gluconate added to each bottle before administration.

Estimations of his fibrinogen level and prothrombin time were normal after operation, and his condition remained in the balance. Twelve hours after his second operation his blood-pressure began to fall off again, and in spite of another pint of blood transfused at that time his haemoglobin was estimated at 5.2 g./100 ml. A further three pints of fresh blood were now available, and after these were transfused he made an uneventful recovery. A subsequent thromboplastin regeneration test showed no abnormality.—We are, etc.,

E. R. WALROND,
J. E. HUTSON.
The Queen Elizabeth Hospital, Barbados.

Blindness of Abrupt Onset

SIR,—In a recent leading article (28 August, p. 496) on blindness of abrupt onset no mention was made of one cause of unilateral loss of vision which, although uncommon, is important because prompt treatment can usually save the sight of the eye. I refer to a metastatic malignant deposit in the choroid or retina. This condition usually presents as rapid impairment of vision in one eye, developing over a period of a few days as the disease encroaches upon the macula. Pain may or may not be a feature, but if present usually precedes the loss of vision. The diagnosis is made by ophthalmoscopic examination, and biopsy is not indicated in a patient with known malignant disease.

The commonest primary tumour to metastasize to the eye is carcinoma of breast; and breast has been the primary site in all the adult cases I have seen, except where eye involvement was part of the picture of gross generalized carcinomatosis. Many other primary sites are mentioned in the pathological literature based on necropsy material.

Radiotherapy to the involved eye will usually restore some of the visual loss, and if given early may completely restore normal vision for the remainder of the patient's life. It is for this reason that the early recognition of this cause of blindness is important. Metastatic disease of the eye should be the first consideration in any case of unilateral loss of vision in a patient with a previously treated cancer. Metastases elsewhere in the visual pathway can cause field defects in both eyes and other changes of visual perception; but such intracranial lesions are not so often misdiagnosed or left untreated.

Hurley Hospital, Flint, Michigan. A. F. PHILLIPS.

Eosinophilia in a Foetus

SIR,—The occurrence of an eosinophil leucocytosis in the blood of a 25-week human foetus seems worthy of record.

The mother is a 37-year-old schizophrenic and the father is unknown. The foetus, a male weighing 558 g., was delivered by abdominal hysterotomy and showed no anatomical abnormality. No leucocyte count was done, but a blood smear taken one hour after delivery gave the following differential analysis: neutrophil polymorphs 1.5%, lymphocytes 72.5%, monocytes 1%, eosinophils 23.5%, basophils 0.5%, and myelocytes 0.5%. Apart from the eosinophils this is in accordance with normal levels at this age,[1] and probably indicates an absolute eosinophil count of 1,500–2,000 per c.mm.

The mother gives no history of allergy or other cause of eosinophilia, but a subsequent blood count showed: total leucocytes 7,800 per c.mm., neutrophils 60%, lymphocytes 30%, monocytes 6%, and eosinophils 4%. The differential count was later repeated on 500 cells as follows: neutrophils 52.6%, lymphocytes 27.4%, monocytes 6%, eosinophils 11.8%, and basophils 2.2%.

Thus there may be a hereditary element to the foetal eosinophilia. Whether or not this is so, it raises the possibility that a stimulus to eosinophilia can perhaps cross the placenta. Furthermore, the case provides additional evidence that the mid-term human foetus is capable of at least some components of an immune response.

The Marsden Foetal Tissue Bank is supported by a grant from the M.R.C.

—We are, etc.,

HELEN BAKIRZIS.
H. E. M. KAY.
Foetal Tissue Bank, Royal Marsden Hospital, London S.W.3.

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Varicose Veins in Pregnancy

SIR,—The answer to Dr. F. I. D. Konotey-Ahulu's letter (30 October, p. 1065) presents no difficulty.

(1) My letter referred to Mr. Harold Dodd's visit to Ngutu in the Zululand Reserve of Natal. Mr. Dodd is the co-author of the detailed work on varicose veins, and went to Ngutu to verify for himself that varicose veins are for practical purposes absent in these eaters of unrefined maize, even in pregnancy.[1] An observer of this experience is not likely to remain long in doubt as to whether varicose veins are actually present or not.

(2) Mr. Anthony Barker has spent many years as Surgical Superintendent of the Charles Johnson Memorial Hospital, of 600 beds, at Ngutu, and obviously has enormous experience of surgical conditions in the African. In the *Lancet*[2] he reports five cases of varicose veins in over 14,000 admissions.

(3) Varicose veins tend to get worse and to cause serious symptoms. In the United States of America the operations on varicose veins are relatively as common in the negro