of the sternum, in my experience, is a less important cause of paradoxical movement than the associated rib or costal cartilage fractures, and I believe that it is usually better to obtain fixation of the ribs rather than of the sternum. I would think that plating of the sternum is rather likely to cause trouble in the future because of the thinness of the overlying skin. The chief advantages of an open surgical approach, as Grant and I tried to show, are (1) it is relatively simple; (2) it gives an almost perfect anatomical and functional result, so that early mobilization is possible if other injuries permit; (3) it is possible to deal effectively with the pneumothorax or haemo-pneumothorax which so commonly accompanies the rib injury.

The question of timing of operation is important; it should be done as early as possible so as to forestall the "wet lung" state. We think that shock is less of a contraindication to operation than it appears at first. It can usually be corrected by blood transfusion and by dealing with the anoxia which is caused by the paradoxical movement and which seems to aggravate shock very markedly. The early commencement of positive-pressure anaesthesia—if need be for some hours before operation—may be life-saving in these difficult circumstances.

In conclusion, I agree very much with Mr. A. W. Fowler and Mr. J. E. Jacques (Journal, September 7, p. 92) when they say that stabilization of the loosened portion of chest wall is the prime object of treatment. Tracheotomy may be, as Mr. J. A. Rhind remarks (Journal, August 24, p. 470), dramatically beneficial, but to suggest that it is the only treatment necessary seems to me to overstate the indications for its use. I think that Mr. Henry is to be congratulated on his success in an otherwise hopeless case.—I am, etc.,

Liverpool, 1.

B. J. BICKFORD.

RECENTS

Malignant Change Following Herpes Simplex

Sin.—In his very interesting article on malignant change following herpes simplex (Journal, September 14, p. 615), Dr. R. Wyburn-Mason writes: "The pathological changes of herpes simplex or zoster and herpeticform lesions are those of an inflammation with oedema between the epidermal cells, and there is nothing specific about the changes."

I would like to point out that the histological changes in herpes simplex vesicles are distinctive and diagnostic. Acanthosis is followed by a disintegration of the structure of the spinal layer due to loss of prickles of the individual cell, and fluid from the underlying inflamed corium passes into the "dilapidated" epidermis to produce an intraepidermal vesicle. Many epidermal cells also undergo "ballooning degeneration" leading to the formation of mono- and multinucleated "balloon cells." The nuclei of many cells show centrally placed eosinophilic bodies (Lipshütz) and margination of chromatine. The vesicles are due to the action of herpes simplex virus. Similar changes take place in zoster and varicella.—I am, etc.,

HENRY HABER.

Sensitization to Tulle Gras Dressing

Sin.—I entirely agree with the opinions expressed by Dr. C. M. Ridley (Journal, May 25, p. 1224) and Dr. W. M. Jordan (Journal, September 7, p. 591) regarding sensitization to tulle gras. Looking through my records I found eight similar cases, all patients with hypostatic eczema whose legs had become inflamed after the use of tulle gras dressings. In every case patch tests were positive to balsam of Peru in paraffin molle flav. and negative to the base alone. Apart from its pleasant aromatic smell, balsam of Peru has little value. Its limited antiseptic properties do not make tulle gras a suitable dressing for infected cases, whereas petroleum jelly gauze alone is a safe and suitable application for clean ulcers.—I am, etc.,

HENRY HABER.

Blood Group Chimerae: Transplacental Bleeding

Sin.—Dr. C. C. Bowley and Mr. I. Dunsford (Journal, August 17, p. 408), referring to transplacental bleeding, state that, as far as they are aware, haemorrhage from mother to foetus has not been recognized. It should be pointed out that, although spontaneous recognition of this has not to my knowledge been reported, there are already several experimental studies of the problem. Naeslund and his colleagues,1,2 in three separate studies, transfused ellipotyctes and erythrocytes tagged with 59Fe and 59Fe into women near term, and Mengert et al., similarly transfused blood and cells tagged with 59Fe, and examined the infants' blood at birth for the presence of the particular cells transfused to the mother. The radioactive studies did not give a definite answer, but the morphological studies, particularly those using blood of the sickle-cell trait, supported the idea that erythrocytes in small numbers had crossed the placental barrier from mother to foetus. With these experiments in mind I examined the cord blood of a child born of a mother with asymptomatic hereditary elliptocytosis, but found no elliptocytes among the foetal cells. Of course, had elliptocytes been present, one would have had to wait and see whether the few elliptocytes at birth eventually disappeared, thus confirming bleeding from the mother, or whether they increased in number, thus indicating inheritance of the trait. Similar data might be sought in pregnant women with the sickle-cell trait.

As far as the controlling factor through any placental defect, the pressure gradient between foetal and maternal circulations must obviously be of prime importance. It should therefore be remembered that in the resting uterus the pressure within the foetal villous vessels is some 30 to 40 mm. Hg, while in the maternal intervillous lake it is less than 10 mm. Hg. Thus the speculation that the