Restoring Blood Volume

SIR,—I am honoured that my work about chlorpromazine, blood volume expansion, and skin temperature measurement in treatment of shock has been quoted in your leading article (2 May, p. 251).¹ However, I regret that you do not seem to have grasped the main advantage of the regimen. You suggest that if a patient has received chlorpromazine his skin temperature is an uncertain guide to hypovolaemia. This appears as the opposite of a recommendation.

The point, however, is simple. If a patient in shock has got cold feet in a warm room and does not respond to transfusion by getting warm feet, overloading may occur when the external jugular vein is observed to distend and infusion is continued. Small doses of chlorpromazine are given, followed by further plasma expansion with Rheomacrodex and the distension of the jugular vein observed. If the blood pressure drops during vasodilatation further infusion will restore the blood pressure, and when the skin temperature of the toes rises the infusion is stopped. The patient is now in a haemodynamic situation where he can carry his body heat to the surface. During vasodilatation there is a more direct relation between changes in blood volume and changes in blood pressure.

The measurement of skin temperature is therefore a guide as to whether the blood volume is sufficient to achieve a reasonable blood pressure and a good peripheral circulation from vasodilatation.

The motto is: Open up—and fill up. Stop when the feet get warm.—I am, etc.,

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REFERENCE

- ¹ Ibsen, B., *Diseases of the Chest*, 1967, 52, 425.

Infantile Gastroenteritis

SIR,—One aspect of infantile gastroenteritis due to pathogenic strains of *E. coli* which escaped comment both in your leading article and in the paper by Dr. A. G. Ironside and others (4 July, pp. 2 and 20) is the problem which arises when a parent works as a food-handler. In this situation the discharge home of a baby still excreting the organism can create a serious public health hazard. *E. coli* enteritis is not notifiable. The older children and adults who become infected from eating contaminated food are unlikely to be ill. Consequently the means by which the infection has been disseminated and brought home to babies may well escape detection. Clearly in this situation it is desirable that either the child be retained in hospital until no longer excreting pathogens, or the parent be temporarily suspended from working as a food-handler. Hospital staff should routinely either ascertain themselves whether any member of the household works as a food-handler or inform the district medical officer of health before discharging an excreter. This does not always occur at present.

It seems probable that termination of the "carrier" state currently depends upon competitive growth of non-pathogenic strains in the gut. The measures taken to prevent

pathogens from the child reaching others may also prevent competitive strains from reaching the child. As standards of hygiene and barrier nursing improve the duration of the carrier state may tend to increase. If these speculations are well founded one might expect this to be an increasing problem whose ultimate solution may depend upon "seeding" the patient with suitable non-pathogens. It would therefore be of considerable interest to learn whether anyone has had the opportunity to observe the effect of yoghurt upon the duration of the carrier state, or to compare the duration of excretion of pathogens in children discharged home with its duration among those retained in hospital under barrier-nursing conditions.—I am, etc.,

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Dentistry, Herpes Zoster, and Varicella

SIR,—Herpes zoster is uncommon in children, and is usually mild unless associated with neoplasms or leukaemia. I would therefore like to report the following case.

The patient was an 8-year-old boy, who six days before had an extraction of a left upper milk molar. Two days after the extraction his left cheek became swollen and painful, and the following day small blisters appeared on his upper lip. Over the next two days the pain became very severe, and the blisters increased in size and spread to

involve the whole of the left cheek. On the day of admission to hospital he had also developed a generalized rash. There was no known contact with infectious disease, and in the past he had been healthy and had not had varicella, although prenatally his mother had varicella in mid-pregnancy.

On examination he was toxic with a fever of 102°F. (39°C.). He had typical herpes zoster involving the region of distribution of the left maxillary and ophthalmic divisions of the trigeminal nerve, with marked swelling of the face and oedema of the eyelids, but without ocular involvement. In addition there was a generalized rash over the trunk

and limbs typical of varicella (Figure). Both the zoster rash and the varicella became more marked over the next two days, and thereafter slowly improved. A rising titre to the varicella complement fixation test occurred during the illness. All vesicles on the face healed after three weeks, but now, nine months after the acute episode, there is still a very unsightly keloidal scar involving the left cheek and upper lip.

Herpes zoster following dental extraction has previously been described in adults,¹ and is thought to be precipitated by trauma to the dental nerves. It is generally accepted that herpes zoster usually occurs in individuals who have some immunity to varicella, but the onset of varicella in this boy shortly after the onset of zoster suggests any previous immunity to varicella must have been slight. The role of the maternal varicella during pregnancy is uncertain. Zoster has been described in infancy following maternal varicella or zoster, but zoster over the age of 2 is usually characterized by previous varicella in the child.²

It seems likely in this boy that the herpes zoster was related to dental extraction. Therefore, I feel that dental surgery is contraindicated in any child who may be incubating varicella.—I am, etc.,

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REFERENCES

- ¹ Winstock, D., *British Journal of Oral Surgery*, 1966, 4, 29.
² Hope-Simpson, R. E., *Proceedings of the Royal Society of Medicine*, 1965, 58, 9.

Case of Ergot Poisoning

SIR,—The dangers and complications of ergotamine tartrate overdose are rightly stressed by Dr. S. T. Yao, and his colleagues (11 July, p. 86). It might be of interest to point out that death can occur as a complication.¹ Furthermore, peritoneal dialysis is effective treatment.—I am, etc.,

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REFERENCE

- ¹ Jones, E. M., and Williams, B., *British Medical Journal*, 1966, 1, 466.

Disseminated Intravascular Coagulation

SIR,—I should like to report a case in which disseminated intravascular coagulation resulted in fatal haemorrhage, and was probably due to infection with *Clostridium welchii* causing enteritis necroticans.

A 45-year old man was admitted having suddenly collapsed while getting dressed. He had a long history of indigestion and alcoholism and had woken that morning with epigastric pain. On examination, he was conscious but shocked, deeply cyanosed and dyspnoeic, and complained of severe upper abdominal pain. Scattered petechiae were present over the arms and trunk and his conjunctivae were suffused. Bowel sounds were present and no masses were palpable, but rectal examination revealed the presence of fresh blood. Oxygen failed to relieve the cyanosis, and his blood was found to be incoagulable. The platelet count was less than 10,000/cu. mm.,