of the society more than a third suffered from nutritional anaemia. Can that be without effect on output? Those who hearken to vapourings about "all being well" and "there is no malnutrition in the country" live in a world of their own. What a chance for the iron-containing wheatmeal bread could be found among those munition workers! But Lord Woolton has decided it is not necessary. When will the Ministry of Health direct matters of nutrition?—I am, etc.,

Grimsby, Nov. 24. S. W. SWINDELLS.

Red Cell Suspensions in Treatment of Anaemia

SIR,—Dr. G. E. O. Williams and Prof. T. B. Davie (November 8, p. 641) emphasize the need in certain cases to produce the maximum rise of haemoglobin using the minimal volume of blood possible. When the haemoglobin has reached 60% they advise changing to fresh blood to obtain the advantages of antibodies and active leucocytes. But they do not mention a fact that I noticed in my much more limited experience—this is, that the amount of blood necessary to raise the haemoglobin varies directly with the age of the blood; this is more marked in the toxic patients.

In their series Dr. Williams and Prof. Davie used concentrated red cells, and the cases quoted in their Table I average 36.5 c.cm. for a 1% rise in the haemoglobin. But the individual figures vary very widely, and one wonders if the average would be much better if the cells were prepared from fresher blood. These all seem to be cases in which there was enough time to warn the blood bank of the transfusion and to send a sample of the recipient's blood to cross-group with suitable donors and to return their fresh blood.

• In the cases I have observed, which included those of haemorrhage, anaemias of pregnancy and puerperium, pernicious anaemia, and leukaemia, with the patients' weights varying from 7 to 11 stone, fresh defibrinated blood being used, the amount necessary to obtain a 1% rise in haemoglobin was always between 40 and 43 c.cm.: it was rarely as high as 43 c.cm. So constant was this figure—40 c.cm. for each 1% rise—that one could predict the amount of blood necessary to obtain any required rise in haemoglobin. The haemoglobin estimations were carried out eighteen to twenty-four hours after transfusion, and were always done by the same independent observer, who made the test before transfusion. If fresh citrated blood was employed and the volume of anticoagulant subtracted, then the actual amount of whole blood given was still the same for a 1% rise in the haemoglobin. With citrated blood that was a few days old, still allowing for the anticoagulant, the amount necessary to produce a rise in the haemoglobin rose steadily; seven- to ten-day-old blood being used, it rarely required less than 70 c.cm., and often as much as 90 to 100 c.cm., to obtain this 1% rise.

It seems obvious that the fresh defibrinated blood can compare very favourably with the concentrated red cells as these are prepared now: the results are more regular and the patient has the advantage of fresh antibodies, leucocytes, and red cells, with a difference in volume of only 3.5 c.cm. for each 1% rise in the haemoglobin. If concentrated cells were prepared from fresh blood it would probably require much less than 35.5 c.cm., maybe as little as 20 or 25 c.cm., for each 1% rise in haemoglobin.—I am, etc.,

Newcastle-upon-Tyne, Nov. 21. K. B. ROGERS.

Haemoglobinometry

SIR,—In his letter Dr. Robt. Campbell (November 22, p. 747) mentions his difficulty in deciding the correct method to use the Sahli instrument to estimate the haemoglobin percentage. I should like to suggest that it might be possible to overcome this difficulty by standardizing the Sahli instrument by comparison with a Haldane instrument. The exact technique used during this standardization should be carefully noted and thereafter strictly adhered to. The time which elapsed before diluting might be two minutes or forty minutes, but would be constant in every case.

I think this method would give sufficiently accurate results from "the viewpoint of the general practitioner," and would be simpler and cheaper than carrying sparklets of carbon monoxide.—I am, etc.,

Dundee, Nov. 22. A. J. E. MILLS.

Loss of Vision after Haemorrhage

SIR,—In view of Colonel H. L. Tidy's article (May 24, p. 774) and Sir James Barrett's letter (November 15, p. 711), a brief reference to the following case, which will be published in more detail later, may be of interest.

A woman aged 35 and thirty-two weeks pregnant was admitted to the Radcliffe Infirmary *in extremis* due to a most severe, concealed, accidental haemorrhage. She had suddenly lost her sight when she collapsed with pain and shock. In a brightly illuminated theatre she could recognize light from darkness, but could not distinguish the outline of anyone standing by her bed. She stated she could recognize a shadow there, but that was all. Ophthalmoscopy revealed no fundal lesion. Before a transfusion was administered oxygen was given with a B.L.B. mask and was continued for five hours. Within twelve hours vision was returning, and three days later the patient could read large type. Since then there has been a complete recovery of sight. Before this patient left hospital she was seen by Miss Ida Mann, who stated that it was probably the administration of oxygen that had saved the retina from destruction. It is possible, therefore, that the wider use of the B.L.B. mask in cases of severe haemorrhage may decrease the incidence of this rare but terrible complication.—I am, etc.,

Oxford, Nov. 19. JOHN STALLWORTHY.

Nicotinic Acid and Pellagra

SIR,—Your editorial article entitled "The Sprue Syndrome" (November 22, p. 731) contains the statement that pellagra "is curable by nicotinic acid." This statement surely requires some qualification.

It has become increasingly clear in recent years that pellagra is *not* due to the lack of a single dietary factor, as was formerly supposed, but is, in reality, a multiple deficiency syndrome. Besides the classic "three D's" of dementia, diarrhoea, and dermatitis, the "typical case" may present the following additional evidence of dietary insufficiency: (1) nutritional polyneuritis, usually (and probably erroneously) attributed to thiamin (vitamin B_1) deficiency; (2) anaemia, the cause of which is still uncertain in this condition; (3) cheilosis, angular stomatitis, and (4) vascularization of the cornea, which are currently attributed to deficiency of riboflavin; (5) folliculosis, the result either of vitamin A or vitamin C deficiency; and (6) a reduction in plasma protein, no doubt due to the aetiological factor you mention—"an unbalanced dietary deficient in protein." It is evident, therefore, that deficiencies of several dietary constituents ordinarily contribute to produce the clinical picture seen in pellagra. In fact, the only reason why it is possible to recognize pellagra as a clear-cut clinical syndrome is probably because of the uniform composition of deficient diets in those parts of the world where it occurs endemically. In other parts, including this country, where deficient diets are less uniform, certain features of pellagra are quite frequently encountered, but the full syndrome is rare.

The important question is, What part of this syndrome can be cured by nicotinic acid? Any critical clinician with experience of the condition will probably agree that this is a very difficult question to answer. The difficulty is that the average pellagrin improves remarkably on simple bed rest and good hospital care, even if the diet is so arranged that it provides no significant amount of the entire vitamin B complex. An efficient nurse, with the aid of soap and water, can often work wonders with a pellagrous dermatitis. This being the case, it is very hard to draw reliable conclusions from a therapeutic trial of nicotinic acid. However, there is now a considerable weight of evidence that it is at least effective in the relief of the gastro-intestinal manifestations of the syndrome and, perhaps, of the erythematous element of the dermatitis; but as to the psychologic changes—after four years' trial of nicotinic acid the mental hospitals of the Southern States of America are still providing for chronic pellagrins.

So far as I know there is no evidence whatsoever that nicotinic acid is in the least effective in the relief of nutritional polyneuritis, anaemia, cheilosis, angular stomatitis, corneal vascularization, folliculosis, or hypoproteinaemia, all of which are frequently encountered in the pellagra syndrome. Of course, it may be said that these changes are not a true part of the