It seems to me that the very factor which makes the risk of post-operative brachial plexus paralysis greater—that is, the greater relaxation obtained in modern anaesthesia—also makes the steep Trendelenburg position unnecessary. The same principle applies to the other extreme positions. Surely a compromise is the answer, the steep Trendelenburg only being used for a very short period while the surgeon packs off the operation field with abdominal towels, and then the horizontal or very moderate slope is assumed.

Dr. Wood-Smith mentions bending the legs in order to reduce the weight taken by the shoulder-rests. If one agrees with the theory that pressure on the calf veins is the most likely predisposing cause of embolism (Mr. McNeill Love, April 26, p. 920 ; Professor Lambert Rogers, *Lancet*, 1946, **1**, 715), this would appear to substitute one bad risk for another. If the steep Trendelenburg position must be used, then the pelvic rest is probably the best solution.

But perhaps the correct conclusion is that these extreme positions are traditional and hazardous, rather than really necessary or really safe, no matter how careful the theatre team may be.—I am, etc.,

Zurich. R. A. C. HERRON.

Pulmonary Embolism from Calf Veins

SIR,—The letters of Dr. H. M. Hanschell and Mr. A. J. Partridge (June 28, p. 1405) are very interesting. It is curious that an argument can develop as to the precise mechanism whereby a prophylactic manœuvre produces its beneficial effects when so far it has not been shown that it produces any effects at all.

Many preventive measures have been suggested from time to time, such as raising the foot of the bed to encourage venous return, raising the head of the bed so that the patient is goaded into activity in his efforts to prevent himself slipping to the bottom, the abolition of Fowler's position, the encouragement of early ambulation, and the administration of drugs such as digitalis and thyroid extract. Unfortunately the incidence of thrombo-embolic disease remains about the same as ever, although its treatment and control have been revolutionized by the anticoagulants. I do not employ any of the above measures except Fowler's position, yet I find that in my last 1,000 abdominal operations there have been only three deaths from pulmonary embolus. This figure is slightly lower than one would expect from the published statistics of large series of cases. Your leader on this subject (July 5, p. 29) is most timely, and I hope that it will provoke a more critical examination of the current theories of the pathogenesis of phlebothrombosis.

Venous thrombosis has been identified with venous stasis for so long, and the insidious power of repetition is so strong, that the two conditions have become inseparable in our minds. One might as well try to convince a schoolboy that Columbus did not discover America (which he did not !) as convince a medical man that vascular stasis is a normal physiological state, in itself of no pathological significance. There seems to be a conception, almost Harveian in its simplicity, that the blood is constantly whizzing round the body, and that should it falter in its onward rush it is likely to solidify. Nothing could be farther from the case. In most textbooks of physiology there is a chapter on haemodynamics which points out that the cardiovascular system may be regarded as a closed circuit, and that at any given point and time in the circuit the velocity of the blood is inversely proportional to the *total* sectional area at that point. The break-up into the capillary bed is associated with an increase of several hundred per cent. in total cross section, and the velocity of the stream in the capillaries is thus very much reduced, a circumstance which facilitates the interchange of gases, food materials, and waste products.

Further, it is known that blood (or any other liquid) flowing along a tube does not move *en masse*, but that its central elements flow much faster than those at the periphery. The outermost layer—i.e., that next to the intima—is actually stationary. The circulation may thus be likened to a river. When the river bed narrows the stream is fast, and may even become a torrent ; when it broadens out the current is gentle, and yet is much faster in the middle of the stream than at the banks, where it is almost motionless. It will be seen, therefore, that vascular stasis is a perfectly normal, and indeed inevitable, state in the human body. Some of the blood is stagnant all the time, the rest is stagnant some of the time.

Nevertheless, it is a truism to say that pulmonary embolism is a disease of patients. The victim does not become a patient *because* he has a pulmonary embolus: he is a patient *before* the catastrophe. The pregnant woman, with a tumour in her belly obstructing the veins of her legs, does not die of pulmonary embolism, though she may do so in the puerperium, when the obstruction has been relieved. Nor is it primarily a disease of the bedridden ; it is a disease of the acutely ill. It is a result of the sudden translation from activity to the sick-bed.

Finally, we should not forget that intravascular clotting is overwhelmingly more common in the swift arterial current than in the leisurely venous stream. It is for these reasons that I venture to suggest that venous thrombosis has little to do with venous stasis, and that the cause must be sought elsewhere. My own hypothesis (*Lancet*, 1951, **2**, 1180) may well be wrong, but I feel that it is a stumble in the right direction.—I am, etc.,

Barnet, Herts. V. J. DOWNIE.

CDE Notation for Blood Groups

SIR,—One of the numerous weaknesses of the CDE notations has been the difficulty of expressing oneself orally when using these symbols. As a result Fisher and Race have resorted to so-called shorthand symbols which are merely minor modifications of the International Rh–Hr Nomenclature. Drs. S. Haberman and J. M. Hill (April 19, p. 851) have suggested an additional method whereby the CDE symbols can be used orally. Their solution merely serves to expose further the weaknesses of the CDE system. In Table I of their paper is given their suggested verbal usage for the CDE symbols, but these constitute nothing more than a literal translation into CDE of the Rh–Hr nomenclature used by me. Race, Levine, and others have criticized the International Rh–Hr Nomenclature because the phenotype symbols "do not include the Hr factors." But, as I have pointed out, since there is a reciprocal relationship between the Rh and Hr factors, the latter are actually included by implication, just as the designation group O implies the presence of anti-A and anti-B agglutinins. Moreover, if the criticism were valid, it would apply equally to the shorthand symbols used by Race and the newly suggested verbal usage. Thus, the new proposal again exposes the inconsistencies and lack of logic of the CDE notations, especially the very first line of the table, where it is suggested that, instead of stating orally, "Little c, little d, little e over little c, little d, little e," the expression "Rh negative" be used.

In Table II, on the other hand, some of the symbols which these authors ascribe to me are not part of the International Rh–Hr Nomenclature at all, and have never appeared in any of my papers. What Haberman and Hill have done is to translate Race's ideas concerning the rh^w (C^w) factor and Rh_o variants (D^u) into the International Nomenclature. These ideas have been shown by me to be incorrect, and of course they are just as fallacious no matter what nomenclature is used.

It seems hardly necessary again to refer to my past publications on the subject, but it may be of interest to call attention to some recent ones—namely, in the *Lancet*, February 2, p. 256, and April 26, p. 876. A comprehensive review of the entire question, entitled "Mosaic Structure of Red Cell Agglutinogens," is due to appear in the June issue of *Bacteriological Reviews*.—I am, etc.,

Brooklyn, New York. A. S. WIENER.