Letters to the Editor

(This new section is being started on a purely experimental basis, and may be discontinued after a year's trial if it proves difficult or unproductive. It is intended to serve for short notes, corrections unsuitable as corrigenda, and conceptual material, all related to papers that have been published in the American Journal of Physiology or the Journal of Applied Physiology. No polemic material will be considered. The section may appear in only the last issue of a volume (June or December).

A 'letter' must comply with the following: *a*) it must deal with substantive material of fact or concept; *b*) it should not exceed one column in length (about 600 words); *c*) assurance must be given that the content of the letter has been seen, without objection, by the author(s) of the paper to which it refers.)

Note on the sigma phenomenon

I feel that I should correct a misinterpretation of history in a recent article by Haynes (*Am. J. Physiol.* 198: 1193, 1960). Dr. Haynes restricts the term 'sigma phenomenon' to the explanation proposed by Dix and Scott Blair (*J. Appl. Physiol.* 11: 574, 1940) which involves summations, giving the ingenious explanation that 'sigma' refers to the Σ sign.

This is historically quite incorrect. The use of the symbol σ was proposed by Schofield and Scott Blair (*J. Phys. Chem.* 34: 248, 1930) to describe anomalies in the fourth-power-of-radius law without reference to any particular theory, even our own. To determine these anomalies, the late Dr. Schofield and I used to plot the mean shear stress at the capillary wall against the average velocity of flow. For a Newtonian liquid, these plots give straight lines whose *slopes* (which we called σ, the letter S then being used for stress) are proportional to the radius (R). In the anomalous systems which we were studying, the curves plotting $\sigma . R$ were reasonably linear, but did not pass through the origin. The intercepts, which we called σ_0, gave a convenient measure of the magnitude of the anomaly. Dr. Schofield and I always referred to the anomalies as 'sigma phenomena,' therefore, though this term may not have appeared specifically in the literature until 1935 (Schofield and Scott Blair, *J. Phys. Chem.* 39: 973).

It would be unfortunate if the term 'sigma phenomena' came to be associated with the special treatment much later proposed by the late Mr. Dix and myself, since it has been used in the literature over many years, in its original wider connotation. I have often expressed the view (*Rheol. Acta*, 1: 123, 1958), which I still hold, that some sigma phenomena depend mainly on finite shearing layers, while in other systems, impoverished wall layers primarily account for the anomalies.

GEORGE W. SCOTT BLAIR
The National Institute for Research in Dairying
Shinfield, Reading, England

Asphyxiated heat and ventricular fibrillation

An article by Coffman and Gregg in this Journal (198: 955, 1960), reported that 33 dogs were asphyxiated by clamping the intratracheal tube and the electrical responses were determined. The electrical response terminated in ventricular fibrillation in one third of these experiments after 10.8–39.5 min. of asphyxia. The electrical response terminated in standstill in the others after 6.5–78.0 min. of asphyxia. The following quotation is from this article: "the finding that uniformly anoxic hearts can develop ventricular fibrillation casts some doubt on the current theory that oxygenated muscle adjacent to anoxic muscle creates an irritable zone giving rise to ventricular premature beats and ventricular fibrillation."

From my laboratory Wolfe reported on 77 dogs that were asphyxiated by clamping the intratracheal tube (*Am. J. Cardiology 4: 229, 1959*). The period of anoxia was 2 min. after the heart stopped beating and after the aortic pulse disappeared. The total period of asphyxia varied from 4.5 to 9.5 min. Fibrillation occurred in none. The clamp was removed from the intratracheal tube; the lungs were inflated with O_2 and the heart was pumped by hand. Of these dogs, 17% fibrillated. They were defibrillated. The coordinated beat was restored in each of the 77 dogs. Observations in a series of 50 of these dogs 24 hours later showed death in 28%, severe brain damage in 36% and slight or moderate damage in 36%. Recovery might have occurred in some, but the experiment was terminated in 24 hours. The conclusions were: *a*) the anoxic heart, blue all over, stopped beating in standstill, maintained electrical equilibrium and did not fibrillate; and *b*) well oxygenated blood delivered abruptly to anoxic muscle produced fibrillation by creating O_2 differentials in some of these hearts.

Lest the observations by Coffman and Gregg (*group 1*) and those by Wolfe (*group 2*) appear to be at variance the following comment is in order. *Group 1* showed that some of the asphyxiated hearts fibrillated. *Group 2* showed that none of the asphyxiated hearts fibrillated, all stopped in standstill. Each statement is correct. Coffman and Gregg were measuring electrical responses only in a dog long since dead (10.9–78 min. of asphyxia) and well beyond the time limit for recovery of the dog. Wolfe measured asphyxia in terms of cessation of the heart beat (2 min. after the heart stopped beating) and within the range of getting the heart to start beating again but not within the range of cerebral recovery. The observations in *group 1* pertain to the electrical response after death and beyond recovery while the observations in *group 2* pertain to the electrical response in an anoxic heart that can be made to beat again. *Group 1* has physiological significance primarily. *Group 2* has practical significance in terms of reversing death. This distinction is important.

The electrical response in *group 1* is interpreted to throw doubt on the current theory of oxygen differentials in muscle giving rise to 'ventricular premature beats and ventricular fibrillation.' This statement needs clarification. A heart that makes ventricular premature beats is, of course, a beating heart. It is at the point of fibrillating but it may have good color and this is in contrast to the heart with prolonged anoxia. There is little or no relationship between the experiments of *group 1* and the heart that makes premature beats and suddenly fibrillates. The anoxic heart might be made to beat again after 10.9+ min. of anoxia but the brain is damaged and complete recovery is not possible. The latter type of heart often appears to be well oxygenated, and it can be made to beat again without brain damage.

This discussion has clinical significance. The human heart that fibrillates is often the heart of the young energetic person who falls over dead from a fatal heart attack. Often, 63%, there is no new disease in the heart. The heart on one day is the same as on the previous day except that an electrical charge developed in the interval which was strong enough to fibrillate the heart. This fibrillating current is dissociated from injury and injury current because it occurs without injury. There is another kind of heart that behaves differently. The heart with severe disease in arteries and muscle is anoxic, weak and often in failure. At the time of operation these hearts have been observed to stop in standstill or they may fibrillate or they may go from standstill to fibrillation and back again spontaneously. As a rule the heart beat cannot be restored in these hearts and from the clinical standpoint it does not matter what they are doing. The response in a good heart is quite different.

CLAUDE S. BECK
Western Reserve University and
University Hospitals
Cleveland, Ohio