Another piece of evidence for heating is that human bone samples from the biological supply house, although exhibiting the same per cent increase in current per degree as fresh bone, had dark currents two orders of magnitude lower (lower water content), and a current increase on illumination of 50–100 per cent rather than the 2–3 per cent of fresh bone. According to the curves of Becker *et al.*[2] for the conductivity of bone versus temperature, fresh bone exhibits currents of 10^{-8} or 10^{-9} amp, and a current change of a few per cent over 20° C, while the current in dried bone changes by more than an order of magnitude, from 10^{-9} to $0{\cdot}5 \times 10^{-11}$ amp, in the same interval. In the present investigation, where the temperature change on illumination is a fraction of a degree, one would expect a current change of a few per cent for fresh bone and ~100 per cent for dried bone, solely from heat. This is exactly what was observed.

Several additional checks were made. The intensity from the mercury lamp at some distance from the thermopile was measured to be 2×10^4 μW/cm². The predicted increase in temperature for a sample 2 mm thick with a specific heat of 0·5 at that position should be 0·00125 degrees/sec, which was approximately the rate observed.

In another check, various filters which passed different regions of the spectrum were inserted between sample and lamp. The ratio of the current produced with the filter to that without was, for each filter, the same as the ratio of the intensity with the filter to that without.

We conclude that if there are photocurrents in bone they must be less than 5 per cent of the currents measured. This would imply photocurrents less than 0·1 per cent of the dark current for fresh human bone. The question then arises whether it is meaningful to regard such small currents as semiconducting in character, as most insulators exhibit photoconductivity to some extent.

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Influence of Ethylenediamine Tetraacetate and Calcium Ions on Vascular Tension

IT is well known that the contractility of muscles is suppressed after removal of the calcium ions from the extracellular liquid and that injection of a small quantity of calcium ions into a fibre of skeletal muscle induces contraction. A definite quantity of intracellular calcium ions is necessary to establish the association of actin and myosin in the presence of ATP, and, therefore, the activity of actomyosinatepase to provoke contraction[1–6]. During stimulation of the skeletal muscle fibre, calcium ions are taken from the extracellular liquid and are restored later[7–10] No uptake of calcium has been detected during contraction in smooth muscle[11,12].

The contractile proteins of vascular muscle exhibit physical and chemical behaviour which is different from that of skeletal muscle[13–17]. We assume, therefore, that some mechanism, other than that of skeletal muscle, is responsible for tonic contraction in smooth muscle[18]. For this reason, we investigated further the influence of ethylenediamine tetraacetate (EDTA) and calcium ions on vascular tension and contraction. In order to remove any stress, vascular sections from cow carotids 15–20 mm long were kept for about 45 min in a bath of 100 ml. of Tyrode solution at 37° C. The Tyrode solution was free from calcium and magnesium. When the equilibrium of tension was reached, different quantities of EDTA (*p*H 7·4) were added. Fig. 1 shows that the addition of 5 mmolar EDTA provokes a progressive increase of tonus which sets in after a latent period of several seconds up to 2 min and may last for 10–20 min before it slowly dies down. It seems paradoxical that an equivalent amount or more of calcium added to EDTA shortly after the onset of contraction, during the rise of tension, will stop the contraction within a few seconds (curve 2). If calcium is applied before EDTA, no contraction occurs. The influence of calcium ions is not specific. It does not matter which one of the bivalent cations (calcium, magnesium, iron, cobalt, copper, manganese) is added in order to form a chelate. Addition of greater amounts of EDTA may sometimes, but not always, provoke a contraction, even if the vessel is bathed in normal Tyrode solution. The muscle of rat diaphragm and of rectus abdominalis does not contract in similar conditions under the influence of EDTA. The active transport of potassium and sodium is cancelled by EDTA. Our results make it unlikely that EDTA provokes contraction of the vascular muscle by depolarization of the muscle membrane. We believe rather that it acts within the cell where it favours the syneresis of tono-actomyosin through a binding of free calcium and magnesium ions which leads to shrinkage of the muscular cell.

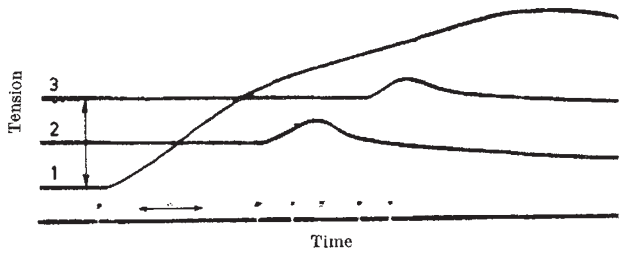

Fig. 1. Tension of carotid samples; (1) given 5 mmolar EDTA at the first signal (marked by faint dots above time scale); (2) given 5 mmolar EDTA at the second signal and 5 mmolar calcium chloride at the third signal; (3) given 5 mmolar EDTA at the fourth signal and 5 mmolar magnesium chloride at the fifth signal. ⟷1 time interval of 1 min; ↕ tension interval of 176×10^3 dyne/cm²; initial tension, $345–365 \times 10^3$ dyne/cm².

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