concerning behaviour of capillary circulation in aspects of escape in the hind limb vascular bed have been found. Under the constant flow experimental procedure, increased tissue pressure may induce at least 2 shifts of flow in the hind limb: one of them is redistribution of the flow fractions. Since capillaries are most exposed to collapse due to higher extravascular pressure, the persisting flow could presumably open alternative channels at the time when capillaries are closed. However, the existence of shunts in the skeletal muscles of the hind leg remains a matter for dispute[8]. Then, it seems to be reasonable to consider another possibility, namely flow persistence through capillaries due to perfusion pressure elevation which balances higher tissue pressure. If this is a fact, then lack of escape could originate from detoriation of the fluid passage across capillary wall between the intra- and extravascular compartments.

In light of reports evidenced by Rodbard et al.[8] and Hinshaw et al.[10], the tissue pressure factor exerts an important influence on the pattern of flow regulation at the capillary level. Therefore disappearance or diminution of escape could result from 'stiffness' of the extravascular compartment which ceases to work in terms of the vasoregulatory function. Both hypotheses concerning the mechanism of escape changes under condition of higher tissue pressure indicate that the capillary level of circulation may be considered to be an important factor in the creation of this phenomenon.

Conclusion. During adrenaline, noradrenaline or acetylcholine infusion into the femoral artery at 5 µg/min for 10 min, a variable degree of escape from alocal typical response to tested compounds was observed either in intact or skinned and denervated dog's hind limb. Escape effect disappeared or markedly decreased under elevated tissue pressure in the hind limb. These results support the hypothesis that the tissue pressure factor and the component involved of peripheral blood flow regulation at the capillary leve could take part in the escape mechanism occuring in hind dog's leg vascular bed during the adrenaline, noradrenaline or acetylcholine prolonged infusion.

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Calcium transients in a molluscan smooth muscle

K. Kometani[1] and H. Sugi

Department of Physiology, School of Medicine, Teikyo University, Tokyo 173 (Japan), 6 March 1978

Summary. The changes in myoplasmic calcium concentration during contraction were recorded in the anterior byssal retractor muscle of *Mytilus edulis* using murexide as calcium indicator, and were found to be qualitatively similar to those in striated muscles except for their slow time course.

It is well established that the contraction-relaxation cycle in muscle is regulated by changes in myoplasmic calcium concentration[2]. Though such changes (calcium transients) have been demonstrated in intact striated muscle fibres[3-9], no attempts have hitherto been made to record the calcium transients in various types of smooth muscle fibres. The present experiments were undertaken to study the calcium transients in the anterior byssal retractor muscle (ABRM) of a bivalve mollusc, *Mytilus edulis*, using murexide as a calcium indicator. *Material and methods.* The ABRM was dissected from the animal with a piece of shell attached to one end and the byssal organ left at the other, and teased in artificial sea water (ASW, 497 mM NaCl, 10 mM KCl, 20 mM $CaCl_2$, 52 mM $MgCl_2$, pH 7.2 by $NaHCO_3$) to obtain a fibre bundle of 1-1.5 mm diameter. The preparation was equilibrated in Ca-free ASW containing 4 mM murexide for 2-4 h to allow murexide to enter into the interior of the fibres. Then, the preparation was washed well with ASW, and mounted vertically in an acrylite chamber (4 × 3 × 1 cm thick) filled with ASW. The shell end was clamped, while the byssal end was connected to a strain gauge to record isometric tension at in situ fibre length. The preparation was stimulated with transversely applied sinusoidal a.c. current (100 Hz) through a pair of Ag plate electrodes. To obtain reproducible results, possible damage to the preparation during stimulation was avoided by the use of a.c. current which does not cause electrolysis[10], and by applying currents of submaximal strenght. No appreciable difference in the mechanical response was observed between the preparations treated with murexide and the untreated preparations. The calcium transients were recorded by means of a double-beam spectrophotometer

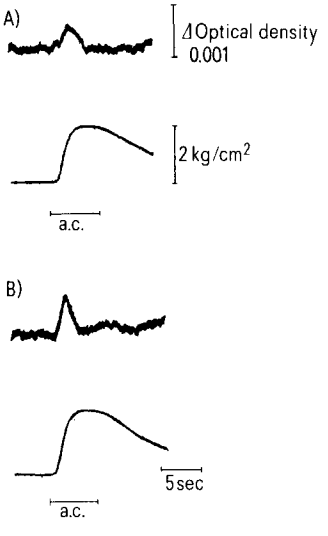

Fig. 1. Simultaneous recordings of the calcium transients (upper traces) and the isometric tension (lower traces). The preparation was stimulated with a.c. currents of varying intensity.

(Shimazu 165) with a sample monochromator set at 470 nm and a reference monochromator set at 542 nm[3,11]; the formation of Ca-murexide complex was recorded as the difference in absorption by the preparation of the 2 alternating light beams. Optical chopping (200 Hz) was made with a revolving slotted wheel placed between the outputs of the monochromators and a fixed half-silver mirror. The optical changes and the tension were simultaneously recorded on an ink writing oscillograph.

Results. Typical examples of experimental records are shown in figure 1. The calcium transient reached its maximum during the rising phase of isometric tension, and almost returned to the initial level when the tension reached its peak. With increasing stimulus strenght, the height of both the calcium transient and the tension increased (figure 1, A and B). As shown in figure 2, the relation between the peak height of isometric tension and the total area of calcium transient was approximately linear. A similar time course of the calcium transients was observed when the preparation was stimulated with d.c. currents, though the tension decayed very slowly after the termination of d.c. currents[12].

That the above optical changes were a valid measure of the kinetics of calcium-murexide complex was supported by the following results; 1. no appreciable optical changes were produced by stretching the resting preparation or during the contraction of the preparation not treated with murexide; 2. the amount of optical changes during the contraction was dependent on the sample wavelength, decreasing markedly when it was increased from 470 to 500 nm; and 3. the same dependence on the sample wavelength was seen in the absorption changes during in vitro formation of calcium-murexide.

The calcium transients in the ABRM fibres observed in the present study are qualitatively similar to those in striated muscle fibres, except that the time course of both the calcium transients and the mechanical responses is much slower in the ABRM fibres. The slow rates of increase and decay of myoplasmic calcium may result from the slow inward movement of extracellular calcium or the slow release of intracellularly stored calcium[13], and may contribute to the slow mechanical response of the ABRM fibres together with the slow shortening velocity of the contractile mechanism[14].

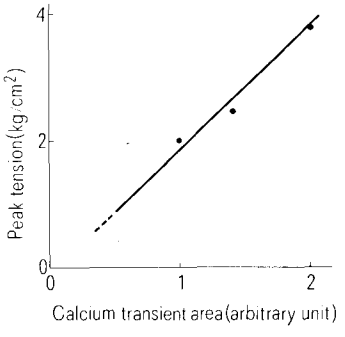

Fig. 2. Relation between the peak isometric tension and the total area of the calcium transient. The preparation was stimulated for 6 sec with a.c. currents of varying intensity.

1 Present address: Faculty of Pharmacoceutical Sciences, University of Tokyo, Tokyo 113 (Japan).
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Effect of low density lipoprotein and high density lipoprotein on sodium dodecyl sulphate precipitation of very low density lipoprotein from human serum

Ikpendu C. Ononogbu

Department of Biochemistry, University of Nigeria, Nsukka (Nigeria), 2 March 1978

Summary. In the presence of low density lipoprotein, the sodium sodecyl sulphate(SDS)-very low density lipoprotein(VLDL) complex sedimented, while in the presence of high density lipoprotein the complex floated. This SDS-VLDL aggregate floats at serum triglyceride to cholesterol ratio of 0.7–0.9 and sediments at a ratio of 0.2–0.5.

Sodium dodecyl sulphate (SDS) is a useful surfactant in the precipitation of proteins. It is also used in the precipitation of serum lipoproteins in the presence of divalent cations or protamine[1], and without divalent cations or protamine[2]. In precipitation of very low density lipoprotein (VLDL) and chylomicrons by SDS, a serum factor is reported to exist[2]. The effect of other lipoprotein classes, namely low density lipoprotein (LDL) and high density lipoprotein (HDL), on the precipitation of VLDL by SDS has not, however, been investigated previously.

The aim of the present investigation is therefore to find how these lipoprotein classes affect the precipitability of VLDL by SDS.

Materials and methods. Fasting venous blood sample was obtained from a healthy male colleague (J.E.) aged 38 years. Serum was separated at 2000 rpm and 4 °C for 20 min. 4 ml serum was subjected to preparative ultracentrifugation, and VLDL, LDL and HDL were obtained according to the method of Hatch and Lees[3]. SDS was purchased from Sigma chemical company (USA). The following solution preparations were made:

a) 2 ml serum, b) 1 ml serum + 1 ml LDL, c) 1 ml serum + 1 ml VLDL, d) 1 ml serum + 0.5 ml LDL + 0.5 ml VLDL, e) 2 ml VLDL, f) 1 ml LDL + 1 ml VLDL, g) 1 ml serum + 1 ml HDL.

The cholesterol and triglyceride concentrations of VLDL, LDL and HDL used in the assay were: 24 mg/100 ml and 63 mg/100 ml; 153 mg/100 ml and 20 mg/100 ml;