It is interesting to note that the three patterns of responses observed in the olfactory centre of the cockroach are quite similar to those in the optic lobe of insects[5] and of the ganglion cells of the mammalian retina[6].

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Conductance or Resistance?

IN circulatory physiology, there may be difficulty in expressing quantitatively the response of a vascular bed in which blood flow has altered. If change of flow is used to express the response, then a large change in arterial pressure may be overlooked. For this reason it has been customary to express responses as changes of resistance, vascular resistance being the pressure difference across the vascular bed divided by the flow through it.

One disadvantage of this method can be appreciated when two simple examples are considered. First, if flow changes from 50 ml./min to 2 ml./min, while the pressure difference remains constant at 100 mm of mercury, then the resistance changes from 2 to 50 units. Second, if flow changes from 50 ml./min to 1 ml./min, while the pressure again remains constant at 100 mm of mercury, then resistance has changed from 2 to 100 units. In these examples the flow changes—48 ml./min and 49 ml./min—are similar and many flow meters would fail to register the difference between the two responses, but the resistance changes are 48 and 98 units, the difference between the two being large.

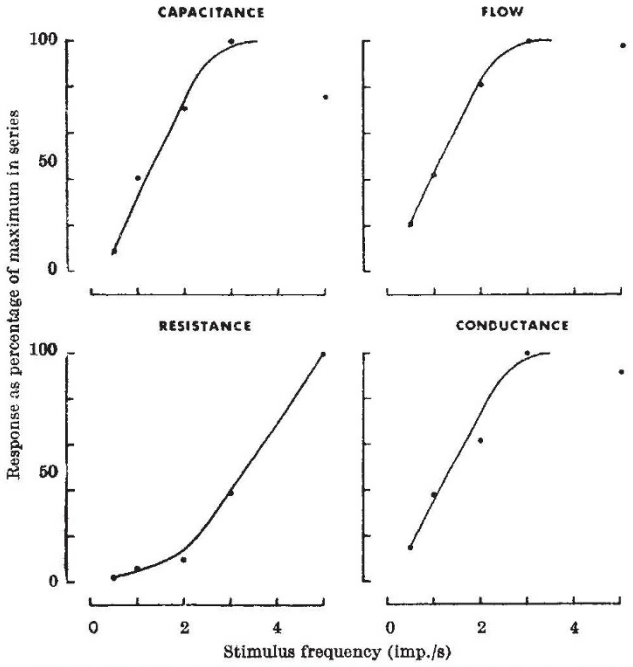

Fig. 1. Capacitance, flow, resistance and conductance responses during splenic nerve stimulation. These responses are measured as the differences between the pre-stimulation values of splenic weight, splenic artery flow, resistance and conductance, and the respective values at the end of the second minute of nerve stimulation. The differences are expressed as percentages of the maximum changes in each parameter (ordinates) and are plotted against the frequency of nerve stimulation (abscissae). Splenic weight change is a measure of capacitance change. For details of the methods see ref. 1.

Vascular resistance is a derived term and is inversely related to blood flow. When vascular smooth muscle contracts, flow decreases and capacitance decreases but resistance increases. Is it not then misleading to compare capacitance with resistance in conditions of nerve stimulation or drug infusion?

Conductance, which is obtained by dividing flow by pressure difference, incorporates pressure but has a direct relationship to smooth muscle length. In electrical terms it is more significant to compare conductance (A/V) with capacitance (A × s/V), rather than resistance (V/A) with capacitance. In circulatory research, it is usual to disregard the pressure denominator when evaluating capacitance.

Fig. 1 shows the responses obtained in one cat when the splenic nerves were stimulated at frequencies between 0·5 and 5 impulses/s. The changes in splenic weight (capacitance), splenic artery flow, resistance (pressure/flow) and conductance (flow/pressure) are expressed as percentages of the maximum responses in the series and plotted against the frequency of stimulation. Smooth curves have been drawn. Capacitance, flow and conductance each have a similar relation to frequency, but the resistance curve shows a marked separation from that of capacitance.

In conclusion, I feel that conductance is the most satisfactory method of expressing responses of a vascular bed in which blood flow or perfusing pressure changes.

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Heparin and the Stabilization of Fibrin

IN experiments to compare the activity of factor XIII (fibrin stabilizing factor) in serum and plasma, it was necessary to activate the factor XIII in plasma by addition of calcium, but to prevent the plasma from clotting. Heparin was therefore used as an anti-coagulant. Addition of heparin to the test system resulted in the factor XIII deficient substrate clot being rendered insoluble in 5 M urea or 1 per cent monochloracetic acid (MCA) without addition of a source of factor XIII. An investigation of the effects of heparin on the solubility of fibrin in these reagents was therefore undertaken.

During the final stages of blood coagulation, fibrinogen is converted, by the enzymatic action of thrombin, to fibrin monomer which will then spontaneously polymerize to form a fibrin clot. This fibrin is soluble in reagents which will split hydrogen bonds such as 5 M urea or 1 per cent MCA and is referred to as soluble or unstable fibrin[1].

A further enzyme, factor XIII, which is normally present in plasma reacts with this unstable fibrin in the presence of calcium ions and converts it into a form which is insoluble in 5 M urea or 1 per cent MCA but which is soluble in 0·1 M 2-mercaptoethanol. The enzyme is believed to be a transamidase[2,3] which is activated from a precursor form by thrombin[4]. The molecular weight of factor XIII has been estimated as 350,000. A tendency to dissociate into inactive sub-units of molecular weight 110,000 has been observed[5] and this dissociation was inhibited by cysteine.

Purified human fibrinogen (Kabi Grade *L*) was clotted with thrombin (Maws bovine 5 U/ml.) in the presence of 0·5 mM calcium chloride and 5 mM cysteine. After incubation for 60 min at 37° C, the clots, when mixed with