This is further illustrated by the experiment illustrated in Fig. 2. Here current pulses of constant intensity are passed with an electrode fixed in one cell and the resulting changes in membrane voltage recorded with another electrode from inside cells located at varying distances along the cylindrical portions of the gland. The voltage becomes progressively smaller as one records from more distant cells, but the decrement is only of an order one would expect if the gland with its 200 cells were to behave like a cable conductor with a continuous core bound solely by an external membrane. In fact, in those cases in which the gland diameter was small in relation to the length constant, the agreement with cable theory[14] was fairly good. For example, in a representative case of a gland of 200μ diameter, the length constant, as obtained from a hyperbolic cosine expression for a short linear cable[15], was 1·2 mm, which is practically the same as that of squid giant axon at equivalent diameter in which, indeed, there are no core septa; and the outer surface membrane resistance was 10,000 ohm cm². (The capacitance of the outer membrane calculated from the time constant of the rising and falling phases of the membrane voltage was of the order of 1 μF/cm².)

Another way the close electrical coupling between cells is brought into evidence is by puncturing the outer cell membrane. If the outer membrane of a single cell is ruptured and prevented from 'healing over', the cell will depolarize, and with delays depending on distance, all other cells of the gland follow suit.

Electronmicrographs of the contact regions between cells, kindly provided to us by Drs. D. Spiro and J. Wiener, show the following relevant features: (*a*) The surface membranes of neighbouring cells are closely apposed; the width of the intercellular space is 100–150 Å. (*b*) Over about one-third of their area, the contact surfaces are aligned parallel in and interconnected at regular intervals of 1000 Å. The remainder of the contact surfaces are infolded and intertwined. From measurements of the areas of the contact and outer surfaces[16], and of their respective resistances, it is calculated that the specific membrane resistance at the contact surface is, at least, 4 orders of magnitude lower than that of the outer surface. The permeability of the contact surface is so high that ions move through it almost as freely as through cytoplasm. The resistance through a chain of cells (150 ohm cm), as measured directly with intracellular electrodes, is only slightly greater than that in extruded cytoplasm (100 ohm cm). The resistance along the intercellular space to the exterior, on the other hand, is very high. Here there exists an ion barrier of, at least, 10^4 ohm cm² (ref. 17).

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Histamine and Hyperæmia of Muscular Exercise

A NEW concept of *in vivo* mobilization of histamine[1,2] may be capable of explaining the microcirculatory aspects of post-exercise hyperæmia[3].

A large amount of evidence suggests that an inducible form of histidine decarboxylase is located in or near cells of the small blood vessels and that it continually catalyses synthesis of minute quantities of free histamine[4]. This 'induced' histamine may be intrinsic; its dilator actions seem to be blocked by topical antihistaminos but not by systemic antihistamines in the usual doses[1,5]. Although the inducible form of histidine decarboxylase undergoes changes in activity, detectable by *in vitro* assay, these changes occur too slowly to be pertinent to the present discussion. However, increased production of induced histamine might also occur rapidly. In exercising muscle, the local rise in temperature may cause an almost immediate increase in induced histamine synthesis simply because histidine decarboxylase, like other enzymes, is a more efficient catalyst at increased temperatures within the physiological range. Microcirculatory dilatation should follow rapidly. Presumably moderate warming of a tissue by any means might lead to the same result, while moderate cooling, by reducing induced histamine synthesis, may permit the vasoconstriction which is observed[3].

Because of experimental difficulties satisfactory means of testing the validity of this hypothesis have not been found. One must detect a small increase in local *in vivo* synthesis of labelled induced histamine in the presence of labelled histamine formed locally in mast cells, or transported from other parts of the body, under conditions which may increase the rate of histamine destruction and dispersal. Publication of these views without direct supporting evidence seems justified since virtually every plausible candidate as mediator has been eliminated[3,6], and since histamine, an early candidate[7], was dropped on the basis of tests[8] which are unsuitable for evaluating induced histamine[1,5].

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Correlations between Blood Sugar-levels after Insulin Injection and Ordinary Meals

IT has been shown that correlations between successive blood sugar-levels throw some light on the regulatory process[1]. They suggest, *inter alia*, that after ingestion of glucose, regulation tends to introduce a limited statistical orderliness, not necessarily to restore the previous one.

Now, what happens if, instead of increasing the blood sugar-level, we try to decrease it by an insulin injection (20 I.U./kg body-wt.) ? Table 1 shows that during the first 3 h there is no correlation between the fasting and the following levels: in the upper row coefficients are very low, partly negative and none is significant until the fourth hour when a positive correlation appears, the numerical value of which (0·72) approaches the short-term glucose reliability (r = 0·79). (In my previous communication, the long-term reliability of blood sugar-level was given as