POTENTIALS FROM STELLATE GANGLION OF *Loligo pealii*. PRE-GANGLIONIC NERVE IS STIMULATED AT 30 PER SECOND. LOWER TRACE OF DOUBLE BEAM OSCILLOGRAPH PICKED UP AT JUNCTION OF PRE-GANGLIONIC NERVE WITH GANGLION, UPPER TRACE FROM ORIGIN OF POST-GANGLIONIC NERVE. MULTIPLE EXPOSURE OF SUPERIMPOSED STIMULUS-TRIGGERED SWEEPS. ONSET OF FATIGUE SHOWING ALL OR NONE SPIKES AND FALLING LOCAL RESPONSE. TIME = 0.5 MSEC.

distributed, one in each postganglionic stellar nerve, to the mantle musculature. At the region of contact the presynaptic fibre and postsynaptic fibre, each 25–100 μ in diameter, lie side by side for 800 μ , making connexions through small holes in their sheaths, by many short collaterals. Cell bodies or dendrites are not involved.

In *Loligo pealii* a preparation is easily isolated consisting of pre-ganglionic nerve (mantle connective with fin nerve removed), stellate ganglion and the last stellar nerve. Single shocks at low intensity delivered to the pre-ganglionic nerve excite a single giant fibre therein and, after a delay in the ganglion of about 1.2 msec. (23° C.), the giant fibres in the stellar nerves. This delay does not include any significant conduction time in fine tapering terminals. Transmission is all or none and one to one; the synapse can follow upwards of four hundred impulses per second for short periods. Increasing shock intensity brings in many smaller pre-ganglionic fibres (including presumably Young's accessory second order giant), some lagging scarcely at all behind the presynaptic giant, but does not alter transmission noticeably. Antidromic impulses are not transmitted from postsynaptic to presynaptic fibre. Transmission is easily blocked by fatigue and for long periods can be kept at any desired level by maintained stimulation of the pre-fibre at controlled frequency. Thus a certain preparation, once fatigued, could be kept in a non-transmitting state by stimulation at 30 per sec. while dropping the frequency to 25 per sec. permitted transmission. Untransmitted impulses continue to exert an effect, maintaining fatigue of the junction, apparently by acting on the postsynaptic unit.

The preparation permits recording not only of single presynaptic and postsynaptic fibres but also of unit synaptic potentials (local activity of the unit synapse, the non-propagated, graded response corresponding to end-plate potential in muscle). A large electrode (0.2 mm. platinum wire) on the ganglion may pick up several of the ten or so junctions, but they are activated by the same presynaptic fibre, are well synchronized and rarely behave independently. Micro-electrode recording gives the same picture as large electrodes in situations studied so far. The synaptic potential detectable with macro-electrodes may be more than 300 μ V., rise to its peak in about 0.6 msec. and fall to a third in less than 1 msec. In fatigue the local response may suffer no change in latency but falls greatly in height, the propagated spike arising later and later, often apparently from the falling phase of local response (cf. Hodgkin⁵). By graded stimulation directly on the ganglion the synaptic potential may be graded. Its absolute refractory period is about 1 msec. and is followed by a relatively refractory period in which at first very small potentials are elicited, later increasingly large ones. Facilitation has been recorded in the fatigued preparation when the first stimulus of a pair elicits only local response whereas the second, if it follows within a critical interval during the relatively refractory period of local response, may result in a propagated impulse.

The general properties of the preparation are strikingly similar to those of the ephapse of Arvanitaki¹ and, with respect to the local response, those of the peripheral nerve fibre².

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Adrenal Cortical Hormone and Pigmentation

THE association between the adrenal glands and pathological pigmentation is well established, and it has been suggested that these glands may also control the physiological process^{1,2,3}. The influence of sodium chloride on melanin formation *in vitro* has been demonstrated⁴, and it follows that if physiological pigmentation is controlled by this mechanism, then deeply pigmented animals should have a low chloride concentration. To confirm this the concentration of chloride in the blood of agouti and black mice has been investigated, using a cross between the CBA (Strong) and C57 (Little) strains which segregates for these colours.

Much difficulty has been experienced in obtaining reliable estimations from the small quantities of serum available. Even by draining the inferior vena cava it has not been found possible to guarantee more than 0.2 c.c. of serum, and all estimations have been made on this quantity. The method used has been that of Schaales and Schaales⁵, but with only 0.2 c.c. of serum the end-point is not certain. To eliminate errors from this source, a photometric apparatus has been constructed, the electrical circuit being taken directly from that described by Needham⁶. Illumination is by a 40-watt pearl bulb housed in a light-tight tin, in which are two $\frac{1}{2}$ -in. holes opposite to and at the height of the centres of the selenium cells. No lenses are used, but two movable screens with $\frac{1}{8}$ -in. holes are placed immediately in front of the selenium cells, to act as baffles. The whole is enclosed in a light-tight box, of which the ends can be opened. The apparatus is completed by a microburette mounted on a movable arm so arranged that the nozzle can either be brought to a position immediately in front of one of the selenium cells when the end of the box is open, or removed to such a position that the box may be closed. Galvanometer readings are made only with the box closed.

Estimations are made on protein-free filtrates, prepared by adding to 0.2 c.c. serum, 11 c.c. water and 0.5 c.c. of each of the Folin-Wu reagents. 10 c.c. of the filtrate are placed in an optical cell, with 0.06 c.c. indicator. The cell is placed immediately beneath the microburette between a movable screen and the selenium cell. The resistance is varied so that more current is fed to the galvanometer from this selenium cell than from the other, the difference being accurately adjusted to one degree on the galvanometer scale. Titration is carried out until sufficient colour is developed in the optical cell to balance the circuit and return the galvanometer needle to zero. This is regarded as the end-point. It is, of course, arbitrary, but gives constant results within the limits examined, that is, 100–150 milli-equivalents per litre of chloride. The calculation is made as in the original method, with the necessary adjustment for the quantity of serum used. In this way it has been found possible to estimate known solutions of sodium chloride, using only 0.2 c.c., with an error of 1 per cent.

The mean value for agouti mice is 121 milliequivalents per litre, and for black 124.5. Estimation of the significance of the difference between these means gives $P = 0.1$ for $n = 29$ ⁷. Therefore, instead of the anticipated low concentration of chloride in black as compared with agouti mice, there is a small rise in this concentration, the difference, however, not being significant. There is thus no evidence of any difference in adrenal cortical function between agouti and black mice, as judged by the concentration of chloride in serum. As these results are considered accurate to within 1 per cent, and a difference of very much more than 1 per cent in the chloride concentration is necessary to affect melanin formation⁴, it can definitely be stated that if physiological pigmentation in the mouse is controlled by the adrenal glands, the action is not through the chloride balance hormone of the cortex.

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Action of Pepsin on Serum Proteins as Measured by Electrophoresis

SINCE the beginning of this year, we have had the first apparatus for electrophoresis (Tiselius-Philpott-Svensson), which has been wholly built in Switzerland (Strübin and Co., Basle). Our main object is to study the physio-pathological alterations in the blood proteins during disease. In addition, we aimed at studying problems of general biological interest.

Thus we endeavoured to illustrate the action of pepsin (Fairchild) on serum proteins by taking electrophoretic patterns of the remaining proteins at different stages of the digestion. In order to measure the undigested remainder of the serum proteins we used the nephelometric method, as developed by Krijgsman¹. In this method the protein concentration is proportional to the turbidity which is produced with sulphosalicylic acid in a strongly acid solution. There is no accurate proportion between digested protein and the increase of the degradation products. Therefore a method measuring the last-named products would not be exact in our case. As buffer system we used veronal/sodium - sodium/acetate/hydrochloric acid (Michaelis), mostly used for electrophoresis. During the peptic digestion the pH was kept at 5.1, the temperature at 37° C., and the pepsin-protein ratio was 1 : 9. In order to stop the reaction, the pH was brought to 7.9 and the solution dialysed against buffer solution of an ionic strength of