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THE ACTION POTENTIAL OF THE SUPERIOR CERVICAL GANGLION.

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LANGLEY strongly upheld the view that sympathetic ganglia act solely as relay stations in the efferent sympathetic pathway. Thus he showed [1900 *a*, *b*] that all the reactions of ganglia which had been regarded as due to true reflex activity were pseudo-reflexes dependent on axon reflexes in preganglionic fibres each of which supplied branches to the ganglion cells situated in more than one ganglion. Further, he showed [1904] that there was no evidence that commissural fibres formed functional connections between ganglion cells of similar function within a ganglion, and the absence of such commissural or internuncial neurones was demonstrated histologically by Johnson [1918] in the frog and by Ranson and Billingsley [1918] in the cat, and has recently been confirmed by Heusner [1935]. However, Langley [1904, p. 259] was careful not to infer that ganglion cells receive branches from only a single preganglionic fibre.

More recently the physiology of the sympathetic ganglion has been studied mainly by the three following techniques:

(1) *Comparison between the effects on the end organ (nictitating membrane) of preganglionic and postganglionic stimulation.* Experiments of this type by Querido [1924] seemed to provide a confirmation of Cannon's suggestion [1914] that the neurones of the sympathetic ganglia transformed the frequency of the impulses reaching them. However, Veach and Pereira [1925] and Knoefel and Davis [1933] have pointed out that Querido's results were vitiated by experimental errors, so Cannon's suggestion is at present without experimental support. Further evidence against this suggestion is provided by Brown's [1934] more refined experiments, which show that a single maximal preganglionic volley sets up after a short synaptic delay a single discharge from

all the ganglion cells. The relatively refractory period following this discharge does not appear to be longer than 15 msec. (a value indicated by the effect of an antidromic volley), so the ganglion should be capable of transmitting relatively high frequencies.

(2) *Comparison of the pre- and postganglionic electrical records.* Bishop and Heinbecker [1932] have also concluded that a single maximal preganglionic volley gives rise to the discharge from the ganglion of a single maximal postganglionic volley, but their values for synaptic delay and refractory period are much longer than those of Brown. In addition they described four groups of preganglionic fibres each of which appeared to be in sole functional relationship with a corresponding group of ganglion cells in the superior cervical ganglion. Recently Bronk, Pumphrey and Hervey [1935] have found that with the stellate ganglion a single preganglionic volley sets up a single postganglionic volley without any evidence of after discharge.

(3) *Perfusion of sympathetic ganglia.* Kibjakow [1933] found that the (non-eserinized) perfusate of a ganglion subjected to prolonged stimulation would on reinjection stimulate the same or another ganglion, and so suggested that a chemical mediator was concerned in the transmission of impulses. Using a similar technique Feldberg and Gaddum [1934] have been unable to confirm Kibjakow, but on occasion have obtained stimulation on reinjection when using an eserinized perfusing fluid. This they have shown to be due to acetylcholine, which appears to be liberated in the ganglion by preganglionic impulses [cf. Feldberg and Vartiainen, 1934]. It is suggested by them that under normal conditions the acetylcholine liberated from the preganglionic fibres is a chemical mediator exciting the ganglion cells to discharge impulses.

All this work is in general agreement with Langley's conception of the sympathetic ganglia as mere relay stations, and both physiological [Bishop and Heinbecker, 1932; Brown, 1934] and histological [Billingsley and Ranson, 1918; de Castro, 1932] investigators have gone further than Langley and considered that each preganglionic fibre ends in relation with its own specific group of ganglion cells, an impulse in the preganglionic fibre setting up a discharge from each of the ganglion cells. Such a preganglionic-postganglionic connection is under normal conditions functionally equivalent to a mere branching of the preganglionic fibre. A more complex behaviour of ganglion cells is, however, suggested by the experiments of Boshamer [1925] [cf. Schilf, 1926], which seem to indicate that an autonomous activity of ganglion cells develops some hours after section of the preganglionic fibres.

The present series of papers describe an attempt to investigate further the physiology of the sympathetic ganglion by analysing the ganglionic action potentials produced under various conditions, and by comparing them with the preganglionic and postganglionic action potentials. Three short preliminary accounts of some of this work have already been published [Eccles, 1933; 1934 *a*, *b*]. Apparently the only previous attempt to record electrically from sympathetic ganglia was made by Fischer and Löwenbach [1933], who describe the irregular series of spike potentials associated with the normal activity of the stellate ganglion, and state that there appear to be at times slower potential changes as well.

Method.

Almost all experiments have been done on cats decerebrated under deep ether anæsthesia, sufficient time being allowed (at least 2 hours) for the effect of the short anæsthesia to pass off. The remaining experiments were performed on Belgian hares under Nembutal anæsthesia (40 mg. per kg. body weight intraperitoneally), control experiments on cats showing that such anæsthesia did not produce appreciable changes in the behaviour of the ganglion. Some decerebrate cats in which nicotine or eserine was injected were also under Nembutal anæsthesia.

The superior cervical ganglion is prepared for recording by careful isolation of the postganglionic trunk up to its entry into the base of the skull, where it is cut. The ganglion is then isolated from the ganglion nodosum and the surrounding tissues, care being taken not to endanger its blood supply through the several small arteries which pass to it from the region of the carotid sinus. Two fine loops of thread soaked in the tissue fluid of the animal are used as leads, one being tied to the postganglionic trunk, and the other forming a loose ring around the ganglion. Fine chlorided silver hooks connect these loops to the input leads of the amplifier and in addition lift the ganglion and postganglionic trunk off the underlying tissues. The cervical sympathetic is separated from the vagus, special care being taken to preserve its blood supply. Two fine thread loops moistened in saline are tied loosely around the nerve and serve to lift it up and connect it to the stimulating electrodes.

In order to minimize the distortion of the slow potential waves a resistance capacity coupled amplifier has been constructed with large grid leaks and condensers. The deflection produced by a rectangular input potential reaches its maximum in about 0·05 msec. (milliseconds) and falls off to half in about 1·9 sec. Provided that the output potential does not swing more than 40 V. on either side of zero, it bears a practically

linear relationship to the input potential, the deviation being less than 5 p.c.

A Cossor cathode-ray oscillograph has been used for recording in all but the earliest experiments, when a Matthews' oscillograph was employed. By an optical system the spot is focused without magnification on to the slit of a plate camera which in recent experiments has been a specially designed rising plate type having a maximal speed of 2 metres a second. With 850 V. on the gun of the oscillograph sufficient light is obtained to photograph at this speed with Golden Isozenith plates (speed H and D 1400). At that gun voltage 40 V. between the deflecting plates gives a spot deflection of about 15 mm., hence spot deviations up to this value are practically proportional to the amplifier input potentials producing them.

Break induction shocks from coreless coils are used for stimulation, the primary circuits being broken by a Lucas pendulum synchronized with the camera. The strengths of the shocks are varied by altering the resistances in the primary circuits, the secondaries completely overlapping the primaries. The primary circuits are earthed through 20,000 ohms, and in some experiments an earthed bridge balancing device [cf. Blair and Erlanger, 1933] has been used between the stimulating electrodes in order to reduce stimulus artefacts. In most experiments this was unnecessary.

Screening presented difficulties owing to the proximity of many kinds of electrical disturbance. A completely screened room has therefore been constructed from soldered galvanized iron sheets, the only opening being a door designed to give a complete pressure contact of metal against metal when shut. Electric mains are excluded, batteries being used throughout. In this way the effect of all outside electrical disturbances is very greatly reduced, *e.g.* nothing is picked up by a portable wireless apparatus inside. It has not been found necessary to screen any apparatus. Even when the relatively high resistance of the nerve is across the input the fluctuations of the base line are seldom greater than $1\,\mu$V.

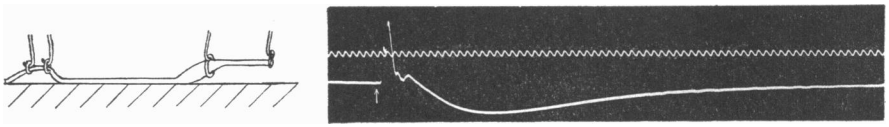
The animal has been enclosed in a heated box the temperature of which in the more recent experiments has been as high as 35° C. moist. Previously the blood supply and the close contact of the ganglion with the warm tissues of the neck were relied on to prevent its temperature falling significantly during the short periods of its exposure on the recording electrodes. A thermometer placed against the ganglion at the end of such periods usually registered a temperature about 33–34° C., the rectal temperature of the cat being 37–38° C. There has been in some

experiments much trouble from slow electrical drifts sometimes with a potential as high as 1 mV. These appear to be caused by localized changes in temperature or evaporation, as they have been much less troublesome when using the moist warm box.

Results.

A. *Introduction.*

In the standard leads which have been used in this research (see Text-fig. 1) the earthed lead to the amplifier has been placed on the ganglion, while the grid lead has been on the isolated postganglionic trunk often beyond a crushed region. Text-fig. 1 is a typical example of the complex action potential recorded with such leads when a maximum volley of impulses is set up by a single induction shock applied to the



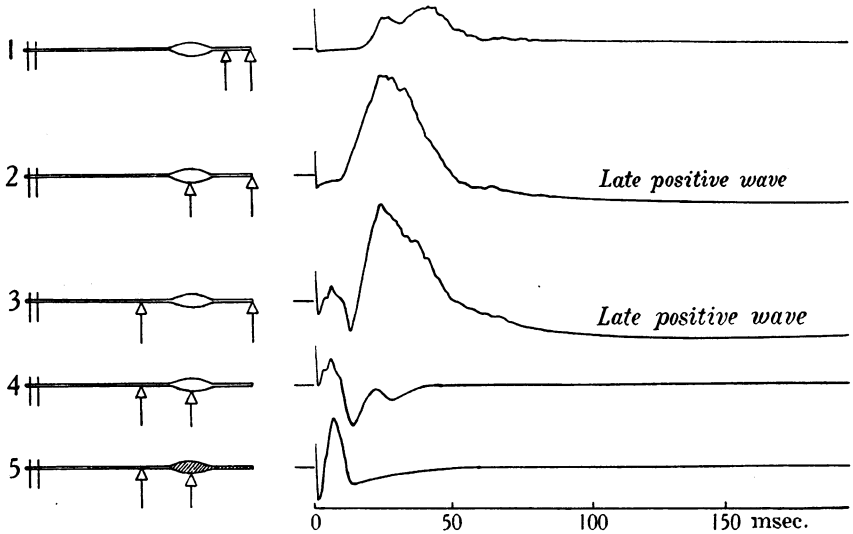
Text-fig. 1. A single induction shock is applied through electrodes on the preganglionic trunk (to the left of the diagram) and the action potential so set up in the ganglion is recorded with the earthed electrode on the ganglion and the grid on the isolated postganglionic trunk, both stimulating and recording leads being raised from the cat (shown in the diagram by the shaded area). An upward deflection in this and all subsequent records indicates a negativity of the earthed lead relative to the grid lead. The arrow indicates the stimulus artefact, which can barely be detected about 6 msec. before the action potential. 1 d.v. = 10 msec.

cervical sympathetic. Shortly after the stimulus artefact which signals the time of the induction shock, the action potential begins with a complicated potential wave during which the earthed lead from the ganglion is negative to the grid lead from the postganglionic trunk, but later the ganglion becomes relatively positive. This positivity reaches a maximum at about 0·15 sec. and has completely passed off at about 0·5 sec. after the stimulus. A similar action potential is recorded by a string galvanometer either directly or after amplification by a battery coupled amplifier, so it must be concluded that no part of the action potential, *e.g.* the phase of relative positivity of the ganglion, is an artefact produced by the resistance capacity coupled amplifier. Moreover, a similar action potential is recorded if the grid lead is on the intact postganglionic trunk, the preganglionic trunk being also intact, so it may be concluded that Text-fig. 1 is a normal action potential of the ganglion, being essentially

unaffected by the division of the pre- and postganglionic trunks. It is therefore of importance to attempt an analysis of this complex action potential in order to determine if possible the mode of its production.

B. *Analysis by altering the position of the leading-off electrodes.*

In order to accomplish this analysis with minimum disturbance from possible electrical artefacts, the ganglion with its pre- and postganglionic trunks has been investigated immediately after complete excision. It will be seen later that this removal of blood supply causes a lengthening

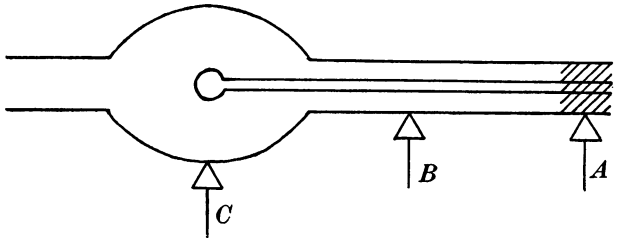


Text-fig. 2. Enlarged reproductions of action potentials of an excised ganglion set up by single maximal stimuli to the preganglionic trunk, the stimulating and recording leads being applied as shown in the accompanying diagrams. The stimulus artefact beginning at zero on the time scale is an upward deflection which overshot on its return with records 2, 3 and 5, the initial base line being shown by the short horizontal line preceding it. Further description in text.

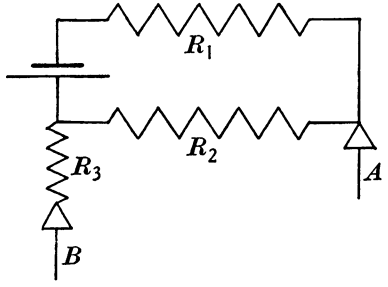
of the transmission time through the ganglion and a slowing of the action potentials. For some time, however, the action potentials remain in other respects normal; excision therefore does not invalidate the results of the analysis.

Text-fig. 2 showing a series of action potentials obtained with the leads shown in the accompanying diagrams is typical of many similar experiments. The complex negative potential wave is later with lead 1 than with lead 2 and more clearly separated into two waves, and with lead 1 there is no trace of the late positive wave which is clearly seen

with lead 2. The main potential with lead 3 is very similar to that with lead 2, but in addition there is an initial wave during the latent period of the response recorded with lead 2. This initial wave is also present in the responses recorded with leads 4 and 5, so it must be produced by the propagation of the preganglionic volley. The absence of this initial wave with lead 2 therefore indicates that the whole of the action potential recorded with this lead arises beyond the preganglionic terminals. A comparison of the action potentials recorded with leads 3 and 4 confirms this



Text-fig. 3. Diagram of ganglionic and postganglionic trunk showing relation of leads to a schematic ganglion cell and its axon.



Text-fig. 4. Diagram showing electrical conditions obtaining for leads *B* and *A* of Text-fig. 3.

inference, for the main action potential with leads 2 and 3 (including the late positive potential) is practically absent with lead 4. In fact the action potential with lead 4 differs from that with lead 5 mainly in the change of the preganglionic action potential from the diphasic towards the monophasic type consequent on the crushing of the ganglion (indicated in the diagram by the shading). A detailed consideration of these results will be attempted after a preliminary treatment of the principles involved in the electrical recording.

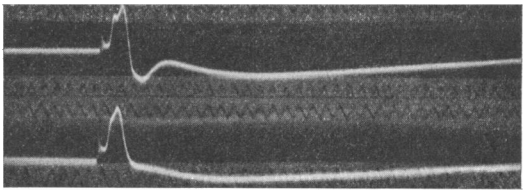
In Text-fig. 3 electrodes *B* and *A* are placed as with normal monophasic leads. In recording the potential difference as an impulse in the

nerve fibre passes B, B and A respectively act as virtual leads from the outside and inside of the fibre. As well as being a specific lead up the centre of the nerve fibre in question, A of course also acts as a diffuse lead from the outside of the fibre at B through all the other conducting tissue of the nerve trunk. The conditions are shown roughly in Text-fig. 4, where R_1, the resistance of the specific lead up the centre of the nerve fibre, is very high compared with R_2, the diffuse lead from the outside of the fibre. The resistance across the input of the amplifier is also very high compared with resistances R_2 or R_3, so the recording instrument acts as a potentiometer measuring the changes of potential between B and A which are produced by changes in the E.M.F. of the battery. The resistance R_2 shunts the potentiometer, hence the potential recorded is only a very small fraction (approximately R_2/R_1) of the actual change in E.M.F. The diffuse lead of electrode A from the outside of the nerve fibre (Text-fig. 3) similarly acts as a low-resistance shunting the potentiometer (amplifier *plus* oscillograph) recording the potential difference between the outside and inside of the nerve fibre opposite the electrode B. Provided that this shunting resistance remains constant and low relative to the resistance of the recording instrument, the action potential recorded at any instant from the whole nerve trunk will approximate to the sum of the individual action potentials which would be recorded from each of the individual nerve fibres.

Exactly the same conditions hold when electrodes C and A (Text-fig. 3) are the leads to the potentiometer. C acts as a lead from the outside of the ganglion cell, its dendrites and axon, and A acts as a specific lead up the inside of the axon. In the present state of our knowledge it seems justifiable to extrapolate this interior lead as far as the axon hillock, for presumably the axon behaves as an ordinary nerve fibre peripheral to this point and possesses no transverse membranes either normally polarized or polarizable by activity. From the axon hillock centrally, however, the possibility of transverse polarizable membranes makes extrapolation a doubtful procedure. The action potential led off by electrodes C and A is therefore a record of the changes in potential between the exterior of the ganglion cell (including its dendrites) and the interior of its axon as far as the axon hillock. Histologically the ganglion cells show an absence of orientation in so far as the spatial distribution in the ganglion of the dendrites and axons is concerned. However, the specific lead from A up the centre of the axon of each individual ganglion cell ensures that all the ganglion cells have a similar electrical orientation with regard to the leads CA. It may

therefore be assumed that the action potential so recorded approximates to the sum of the action potentials which would be recorded from the individual ganglion cells, the same reservations being made as in the case of the similar assumption for nerve fibres.

In Text-fig. 2 the action potential with electrodes *B* and *A* (lead 1) is distinguished from that with electrodes *C* and *A* (lead 2) mainly by the absence of the positive wave. Other experiments show that the latter part of the negative wave (marked by *N* in observations 1 and 2, Pl. I, fig. 4, and Pl. II, fig. 6) is also absent with lead 1, though this is not evident in Test-fig. 2, for it is there submerged by the positive wave. It will be seen in a later paper [Eccles, 1935 *b*, section A] that the small remaining fractions of these waves which sometimes appear with post-ganglionic leads are probably produced by electrotonic spread from the ganglion. It may therefore be provisionally concluded that these two



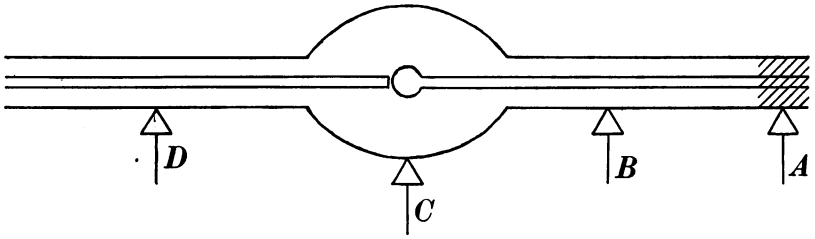
Text-fig. 5. Records of ganglionic action potentials, the second showing the removal of the diphasic artefact after crushing the postganglionic trunk.

late potential waves are mainly records of the ganglionic action potential, *i.e.* they are potential differences between the exterior of the ganglion cell and its dendrites on the one hand and the interior of the axon as far as the axon hillock on the other. The initial waves of the action potential with lead 1 are later than with lead 2, and this temporal discrepancy is directly proportional to the distance *BC* (Text-fig. 3, cf. Text-fig. 7 and section G), so these parts of the action potential are undoubtedly the spike potentials produced by impulses arising in the ganglion cells and without appreciable delay propagated at a uniform velocity along their axons, the postganglionic fibres. This is confirmed by the marked diphasic character which these potential waves possess when the grid lead is on the uncrushed postganglionic trunk (Text-fig. 5). In fact a monophasic record is seldom obtained even when the postganglionic trunk is freshly crushed, and there is always a gradual reversion to the original diphasic response.

The large positive after potentials which Bishop [1934] describes in the non-medullated fibres of the vagus must not be confused with the

slow positive potentials such as those of Text-figs. 1 and 2, for such positive after potentials did not persist for longer than 40 msec. and, presumably, if present in our records, would be fused with the diphasic artefact. Thus, when the postganglionic trunk under the grid lead is painted with 2 p.c. cocaine, a procedure specially recommended by Bishop for recording only the potential changes under the proximal electrode, a small part of the "diphasic artefact" usually persists, which possibly is the positive after potential of the propagated postganglionic impulses, the later slow positive potential being confined to the ganglion.

It therefore seems justifiable to divide the action potential with leads *C* and *A* (lead 2) into those components which are confined to the ganglion cells (with probably some electrotonic spread along the axons), and those which are propagated



Text-fig. 6. Diagram of preganglionic trunk, ganglion and postganglionic trunk showing relations of leads of Text-fig. 2 to a schematic preganglionic fibre, ganglion cell and its axon.

along the postganglionic fibres, and subsequent evidence, *e.g.* the action of nicotine (section C) supports this subdivision.

Text-fig. 6 shows diagrammatically a single preganglionic fibre ending in contact with a single ganglion cell. Electrodes *D* and *C* (lead 4, Text-fig. 2) will give a diphasic record of an impulse in the preganglionic fibre. But there is then only a trace of the action potentials recorded with leads *C* and *A*, *i.e.* of the potential recorded between the outside of the ganglion cell and the interior of its axon. A similar small potential may be recorded with non-specific leads from the ganglion, *e.g.* with both electrodes on the ganglion or with one electrode on the ganglion and one either on its external carotid branch or on its branch to the upper cervical nerves (the grey rami), the preganglionic stimulus being too weak to set up a discharge along these branches, but still setting up a discharge along the main postganglionic trunk (see observations 1, 3 and 7, Pl. II, fig. 6). It may therefore be concluded that, although the preganglionic fibre ends in synaptic relationship with the

ganglion cell, it cannot act as a specific lead from the interior of the ganglion cell—or at least through the ganglion cell from the interior of the axon at the axon hillock.

The converse holds, for with *C* and *A* as electrodes (lead 2) no trace of the preganglionic action potential was recorded. Moreover, an antidromic impulse backfired down the postganglionic fibre gives a large diphasic spike with electrodes *B* and *C*, but with electrodes *D* and *C* no more of the action potential is recorded than with the non-specific leads mentioned above. Presumably the barrier which prevents electrical continuity between the pre- and postganglionic neurones is related to the absence of continuity which is revealed by histological investigations and degeneration experiments.

With electrodes *D* and *A* (lead 3, Text-fig. 2) it was seen that both the preganglionic and postganglionic action potentials were recorded. It might at first seem that this would necessitate the postulation of some electrical continuity between pre- and postganglionic neurones in contradiction to the above conclusion. Reference to Text-fig. 6 shows that this is not so, for electrode *A* together with the postganglionic trunk would be similar to electrode *C* for recording the preganglionic potentials, and then electrode *D* together with the preganglionic trunk would likewise be similar to electrode *C* for recording the potentials from the ganglion cells and their postganglionic fibres . It would therefore be expected that the action potentials with lead 3 would be a combination of the action potentials with leads 2 and 4, as is actually observed (Text-fig. 2).

C. *Analysis by the action of nicotine.*

When the ganglion is painted with a strong solution of nicotine (0·2 p.c. or more) a preganglionic volley fails to set up any action potential either with ganglionic (lead 2, Text-fig. 2) or postganglionic (lead 1) leads (Pl. I, fig. 1, observations 4 and 5). This corresponds with a total block in the transmission of impulses through the ganglion [cf. Langley], and it shows that block occurs before the stage at which any part of the ganglionic action potential is produced. However, the transmission of impulses in the preganglionic fibres right into the ganglion is not abolished by this strength of nicotine, for with lead 4 (Text-fig. 2) a typical diphasic preganglionic response is still obtained. This evidence confirms the above argument in indicating that the action potential of the preganglionic volley does not contribute appreciably to the action potentials recorded with ganglionic and postganglionic leads.

Painting the ganglion with a more dilute solution of nicotine (0·02 p.c.)

or injecting intravenously from 0·2 to 1·0 mg. per kg. changes the ganglionic action potential to the postganglionic type (Pl. I, fig. 1, observations 2 and 3), *i.e.* the latter part of the negative and almost the whole of the positive potential wave are abolished. Transmission of impulses through the ganglion still occurs, for the diphasic spike potential waves are still present both with ganglionic and postganglionic leads, though they may be reduced in size (observation 3) indicating that the discharge from some ganglion cells is blocked. Thus the latter part of the negative and the positive potential wave are distinguished from the initial negative waves (spikes) both by being confined to the ganglion and by a greater sensitivity to the action of nicotine. Moreover, impulses can be transmitted through the ganglion and travel along the postganglionic trunk in the absence of the late negative and the positive waves. Further consideration of these waves will be postponed to a later paper.

D. *Conduction in the preganglionic trunk.*

In observations 1 and 3, Pl. I, fig. 2, where the stimulus is applied to the preganglionic trunk 10·7 cm. from the ganglion, the complex action potential formed by the spike potential waves (henceforth called *S* waves) is more spread out than it is in observations 2 and 4, where the preganglionic conduction distance is 3·8 cm. When the preganglionic pathway is very short, the *S* waves are still closer together, and are often indistinguishable from one another. However, there is no significant alteration of the total area of the composite spike action potential if due allowance is made for the diphasic artefact and the shift of base line produced by the slow negative and positive potential waves. It may therefore be concluded that with all lengths of preganglionic pathway approximately the same number of impulses are discharged along the postganglionic fibres and that the later *S* waves are not due to the repetitive discharge of impulses from the ganglion cells. This is in agreement with Bishop and Heinbecker [1932] and Brown [1934], and the experimental evidence presented in subsequent papers confirms this conclusion. Further, it may be concluded that the group of ganglion cells responsible for each *S* wave has its own group of preganglionic fibres with a characteristic conduction velocity, the increasing separation of the waves with increasing preganglionic conduction being ascribable to differences between the respective preganglionic conduction velocities.

Usually four S waves are distinguishable, and they have been called S_1, S_2, S_3 and S_4 in order of decreasing speed of preganglionic conduction, but any one of these groups may be composite, S_1 in particular separating out into subsidiary waves with long preganglionic conduction (cf. observations 1 and 3, Pl. I, fig. 2). S_2 is always the largest wave, and S_3 is variable, being sometimes as large as S_1 (observations 5 and 6, Pl. I, fig. 4) and sometimes absent. There is no clear division between the S_1, S_2 and S_3 groups, *e.g.* the slowest S_1 preganglionic impulses have rates comparable with the fastest S_2 impulses [cf. Blair and Erlanger, 1933]. However, it will be seen that though overlapping in some properties, the S_1 and S_2 groups are clearly differentiated in other respects. With a long conduction distance the S_4 wave usually appears as a low elevation, often complex, completely separated from the other S waves (observation 5, Pl. I, fig. 4), but in some experiments it overlaps with S_3. Thus in observations 1 and 3, Pl. I, fig. 2, it is not possible to subdivide the three waves following S_2 into S_3 and S_4 groups.

Measurements of the intervals between the stimulus and the S waves with short and long preganglionic conduction paths allow approximate values to be calculated for the conduction velocities of the fastest impulses in each preganglionic group. Columns 2 and 3 of Table I show

TABLE I.

Experiment	Preganglionic velocity in metres per sec.				Approximate preganglionic threshold in arbitrary units		Synaptic delay in msec.		Postganglionic velocity in metres per sec.		Refractory period of ganglion cells in msec.	
	Indirect		Direct									
1	2	3	4	5	6	7	8	9	10	11	12	13
	S_1	S_2	S_1	S_2	S_1	S_2	S_1	S_2	S_1	S_2	S_1	S_2
5. xii. 33	21	13	—	—	10 –	10	3·5	5·5	—	—	4·5	6
12. xii. 33	18	14	—	—	7·1	10	2·7	6	—	—	—	—
30. x. 34	17·5	14·5	20	10	10 –	10	3·6	—	8	3·4	—	—
13. xi. 34	19	—	18	12	—	—	2·2	4·5	—	—	—	—
20. xi. 34	22·6	13·2	22	11	3·3 – 2 +	5 – 3·3 +	2·8	6·5	5	5	—	—
27. xi. 34	17·7	13·8	20·6	11	—	—	3·8	8·4	—	—	3·7	—
3. xii. 34	20·8	7·7	26	12	—	—	2·9	8	—	—	—	—
4. xii. 34	17	11	23	11·5	5	10	2·4	5·8	—	—	4	6
14. xii. 34	14·8	8·4	—	—	3·3	5	3	6·4	5·5	1·7	4·7	5·5
28. iii. 35	16·5	10·6	20·7	12·2	5	10	3	8	5·6	3·5	—	—
1. vi. 35	—	—	—	—	—	—	—	—	—	—	2·9	6
	S_3	S_4	S_3	S_4	S_3	S_4	S_3	S_4	S_3	S_4	S_3	S_4
Approximate values for S_3 and S_4	7–10	1·2–4	8	1·3–5	15	40	?12–18	?10–32	2	1	9	12

that the values thus calculated indirectly agree approximately with the values in columns 4 and 5 determined immediately afterwards by stimulating and leading directly from the preganglionic trunk (Pl. I, fig. 3). The occasional discrepancies with the S_2 wave may be accounted for by the difficulty in locating the beginning of this wave after long conduction

distances (*e.g.* observations 1 and 3, Pl. I, fig. 2), a difficulty which is so troublesome with S_3 and S_4 waves as to prevent any accurate measurements.

The usual four S waves correspond respectively to the M_1, M_2, M_3 and U waves described by Bishop and Heinbecker [1932], but the preganglionic conduction velocities are considerably higher than the values given by them, a discrepancy which may be due to the condition of their excised nerves. The M_1 wave of Bishop and Heinbecker, *i.e.* our S_1 wave, is partly due to the ganglion cells supplying the nictitating membrane, which have been particularly investigated by Brown [1934].

E. *Alterations in the strength of the stimulus setting up the preganglionic volley.*

In the series of ganglionic action potentials shown in Pl. I, fig. 4, a progressive weakening of the stimulus applied to the preganglionic trunk is seen to cause S_4, S_3 and S_2 in turn to become smaller and drop out, only S_1 being produced by the weakest stimulus. With the exception of S_4 the thresholds of the fibres of each group overlap with those of adjacent groups, *e.g.* a small S_2 wave is usually produced by a strength of stimulus still submaximal for S_1 (observation 2, Pl. I, fig. 4). The approximate range of thresholds for each group is shown in columns 6 and 7 of Table I, but a more complete account of the relative thresholds of S_1 and S_2 fibres is given by the curves of a later paper [Eccles, 1935 *b*].

If a stimulus which sets up only an S_1 spike be still further weakened, there is at first a decrease in both the potential and duration of the spike, but the duration soon reaches a limiting value, further weakening of the stimulus only causing a decrease in the potentials. The spike thus obtained must be due to an almost synchronous discharge of impulses from the S_1 ganglion cells, and its time course must be practically identical with that of the spike produced by the discharge of a single ganglion cell. The total duration of such a spike is about 5 msec., the rising phase being about 2 msec. and the falling phase 3 msec.

The potentials of the S waves gradually decrease as the earthed lead is moved from the ganglion further and further postganglionically [cf. Text-fig. 1, Eccles, 1935 *b*], a decrease which seems adequately to be accounted for by the increasing temporal dispersion of the individual impulses, by the relatively forward shift of the diphasic artefact, and by the increasing proximity of the killed end of the postganglionic trunk. No evidence is at present forthcoming which proves that impulses in the ganglion cells themselves are partly responsible for the S waves of the

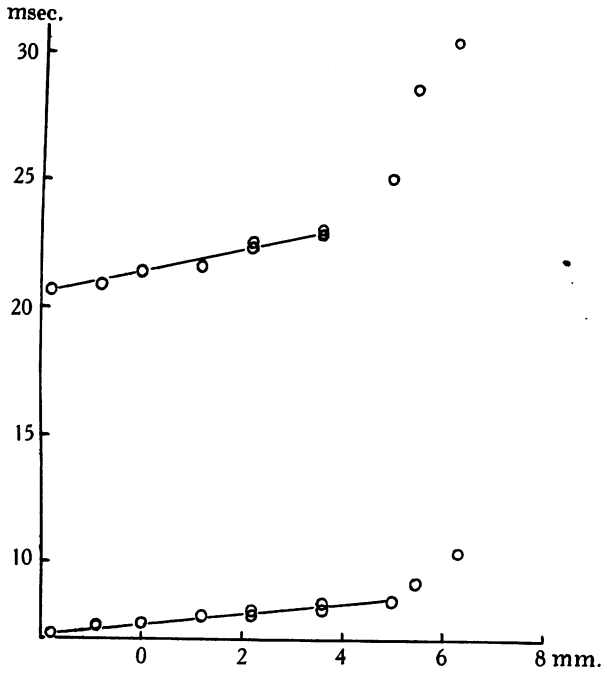
ganglionic action potential, though it does not seem likely that the impulses in the postganglionic axons during their short course within the ganglion give rise to the whole of such S waves. Thus it is probable that an impulse arises as an explosive act at some local point in a ganglion cell, spreading thence over it, and, as with the impulse in a nerve fibre, a maximum at any one point is reached rapidly but not instantaneously. The rising phase of the ganglionic spike potential would be caused both by the stage of invasion, and by this rapid increase to a maximum. In any case it is certain that the beginning of each S wave in the ganglionic action potential may be taken to represent the instant when the earliest nerve impulses arise in the group of ganglion cells producing that wave.

F. *The synaptic delay in the ganglion.*

From the preganglionic conduction velocity determined as above, the total preganglionic conduction time can be calculated for each S wave, the length of the preganglionic pathway being measured from the point of stimulation to the electrode on the middle of the ganglion. The conduction time so calculated is found to be less than the interval between the stimulus and the beginning of the corresponding S wave of the ganglionic action potential by an amount which varies from about 3 msec. for S_1 to as much as 8 msec. for S_2 (Table I, columns 8 and 9) and to still longer durations for S_3 and S_4. This interval is conventionally known as the synaptic delay, but in deriving it the assumption has been made that the propagation of impulses along the intraganglionic part of the preganglionic pathway occurs in a straight line and at the same rate as in the preganglionic nerve trunk.

Now microscopical examination [Ranson and Billingsley, 1918; de Castro, 1932; Heusner, 1935] shows that in the ganglion the preganglionic nerve fibres branch a great many times to form a complicated interlacing system of very fine fibres about $0{\cdot}5\mu$ in diameter. The conduction of impulses in these fine non-medullated fibres is probably much slower than 1 m. a second, so it is likely that a considerable part of the so-called synaptic delay is really occupied in these fine preganglionic branches. The remainder of the calculated time may be called a true synaptic delay. It measures the time between the arrival of the forefront of the foremost impulse at the synapses and the beginning of the impulse set up in the ganglion cell. The various processes which occur during this true synaptic delay will be considered later. The synaptic delay of the S_1 wave is in good agreement with the value, not more than 2 msec., determined by Brown [1934], for in calculating this latter value the

preganglionic conduction velocity was assumed to be only 10 m. per sec. The much longer delay (15–20 msec.) obtained by Bishop and Heinbecker [1932] is probably due to the fact that they worked on excised ganglia (section K). The ill-defined character of the S_3 and S_4 spikes introduces such errors into the determination of the preganglionic conduction velocity that little significance can be attached to the synaptic delays shown at the bottom of Table I.



Text-fig. 7. Curves showing postganglionic conduction, the abscissæ being distance measured from the postganglionic pole of the ganglion, and the ordinates the corresponding intervals after the stimulus at which the action potential occurs. The lower curve is for the beginning of the S_1 wave, the upper curve for the crest of the S_2 wave. The grid lead from the killed end was 8 mm. distal from the ganglion.

G. *Conduction in the postganglionic trunk.*

It has already been noticed that with postganglionic leads the S waves are later than with the ganglionic lead by intervals representing the conduction times from the ganglion. In Text-fig. 7 the plotted points show that for the first 4–5 mm. these conduction times increase linearly with the conduction distance from the ganglion, *i.e.* the conduction velocity is constant. Thereafter the conduction slows, an effect presumably

due to the proximity of the killed end. Columns 10 and 11 of Table I give the postganglionic conduction velocities for those experiments in which the postganglionic trunk was long enough to allow approximate values to be determined. The relatively fast S_1 values indicate medullated postganglionic fibres [cf. Bishop and Heinbecker, 1932], though some of the values for the S_2 group, which they state to be non-medullated, are also relatively fast.

A more accurate determination of postganglionic conduction rate has been attempted by stimulating the cervical sympathetic and recording the action potentials so set up in the long and short ciliary nerves (Pl. I, fig. 5), allowance being made for the preganglionic conduction time and for the synaptic delay. As would be expected, the value so obtained for the S_1 fibres, 7–8 m. a second, is rather higher than that obtained from the short isolated postganglionic trunk.

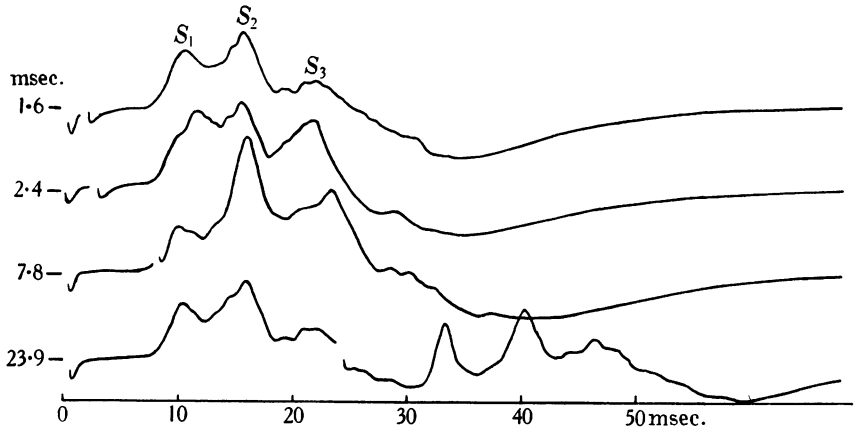
H. *Action potentials in other branches of the superior cervical ganglion.*

With one lead on the ganglion and one either on its branch to the external carotid, or on its branches to the upper cervical nerves (grey rami), three spikes are usually recognizable (Pl. II, fig. 6, observation 5). The thresholds and conduction velocities of the corresponding preganglionic fibres and the synaptic delays correspond with those for the S_2, S_3 and S_4 spikes of the main postganglionic trunk. Sometimes a small ripple appears corresponding to the S_1 spike, *e.g.* the small downward deflection occurring during the latent period of the S_2 spike in observations 6 and 7, the branches presumably acting as non-specific leads from the S_1 ganglion cells (cf. section B), all of which appear to send their axons along the main postganglionic trunk. This is in agreement with the view that the axons of S_1 ganglion cells are distributed solely to structures in the orbit [Bishop and Heinbecker, 1932]. The S_2, S_3 and S_4 ganglion cells of the grey rami and external carotid branches do not differ appreciably from those in the main trunk.

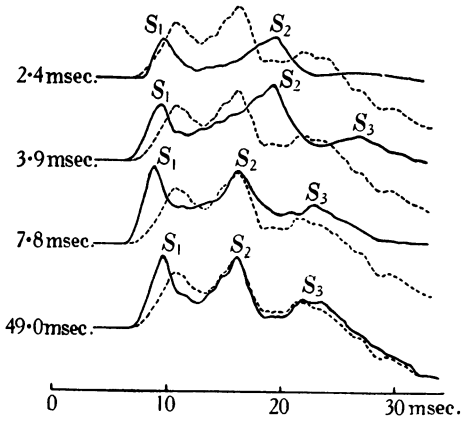
I. *The interaction between two successive maximal volleys.*

Text-fig. 8 shows a typical series of records of ganglionic action potentials in which two maximum preganglionic stimuli have been applied at various intervals apart. The second stimulus is effective in giving rise to an action potential when the interval is 2·4 msec. or longer, but it is only at much longer intervals that the second action potential approaches its full size. The ineffectiveness of the second stimulus at an interval of

1·6 msec. presumably is due to an absolutely refractory period of some part of the preganglionic-ganglionic pathway. The shape of the second action potential can be drawn by subtracting the action potential for



Text-fig. 8. Enlarged drawings of postganglionic action potentials set up by two maximal preganglionic volleys at various intervals apart. The first stimulus occurs at zero of the time scale, the second at the beginning of the break of the curve.



Text-fig. 9. The continuous lines show the action potentials by the second stimuli at the indicated intervals as determined by subtracting the first action potential from the combined action potentials (cf. Text-fig. 8). The broken lines show the action potential set up by the second stimulus alone, all stimuli being synchronized at the zero of the time scale.

the first stimulus alone from the combined action potential, and a series of curves constructed in this way is shown in Text-fig. 9 together with the action potential set up by the second stimulus alone. With a very

short interval, 2·4 msec., only S_1 and S_2 waves can be recognized and the latent period of both is increased. With a longer interval, 3·9 msec., S_3 can also be recognized and now the latent period of S_1 is shorter than with the second stimulus alone, and its rise is steeper, though it is still probably submaximal in size (cf. 7·8 msec.). This steeper rise of S_1 can be detected at intervals as long as 100 msec., and is invariably present after a previous volley. There is often a similar shortening of latent period and a steepening of the rise of S_2, but these effects are much less obvious and only occur at relatively short intervals. The latent period of the S_3 wave is also increased at short intervals, *e.g.* 3·9 and 7·8 msec. (Text-fig. 9).

In Text-fig. 10 the intervals between the stimuli are plotted as abscissæ and the intervals between the beginnings of the S_1 waves as ordinates. The shortening of the S_1 latent period for all except the very short intervals is shown by the points lying below the line at 45° from the zero origin. For these very short stimulus intervals, the intervals between the S_1 waves tend to reach a limiting value of 2·9 msec., which presumably is determined by the following three factors:

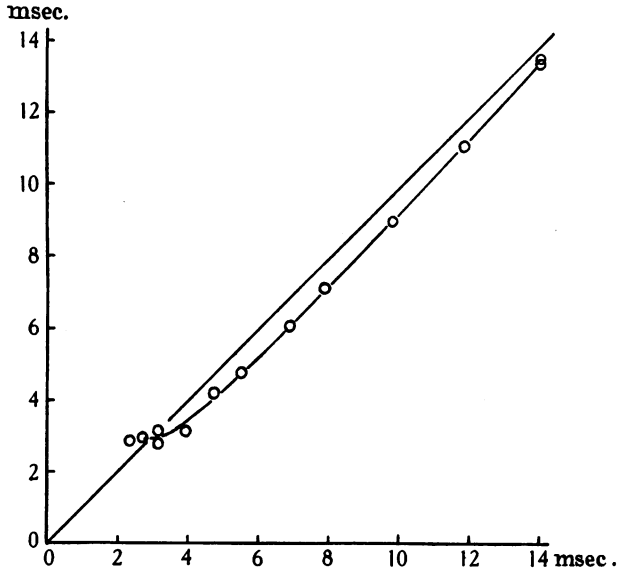
(1) The second preganglionic volley is travelling in the relatively refractory period of the first, the delay so caused being greater, the shorter the stimulus interval.

(2) For the same reason the second preganglionic volley is smaller, the shorter the stimulus interval.

(3) The ganglion cells are refractory immediately after their previous discharge, and only when their excitability has sufficiently recovered are the incident excitatory impulses of the second preganglionic volley able to set up a discharge.

The first factor would only be effective with a long preganglionic conducting distance. With shorter distances factors 2 and 3 cut across one another in determining the limiting value, *i.e.* as the stimulus interval lengthens both the size of each preganglionic impulse and the excitability of the ganglion cells increase. It is therefore clear that the limiting value of Text-fig. 10 is slightly longer than the true absolutely refractory period of the ganglion cells. Limiting values of the response interval, determined as above, are given in columns 12 and 13 of Table I. The length of the least interval at which the second stimulus sets up a full-sized S wave is also not a measure of the relatively refractory period of the ganglion cells, for, as will be seen in a later paper, this interval is also dependent on other factors. The S_1 wave is usually full-sized at an interval of 10 msec.

Brown [1934] found that the shortest stimulus interval at which a second discharge can be evoked from ganglion cells supplying the nictitating membrane (S_1) was about 1·7 msec. This value is in agreement with the value 2·9 msec. here determined, for with the long preganglionic path, 5 cm., used by Brown, the second volley would actually reach the ganglion at considerably more than 1·7 msec. after the first. The much longer least interval, 20–30 msec., determined by Bishop and Heinbecker [1932], is probably due to their ganglia being excised (section K).



Text-fig. 10. Intervals between stimuli (cf. Text-fig. 8) are plotted as abscissæ against as ordinates the corresponding intervals between the beginnings of the S_1 responses set up by the two stimuli. The straight line is drawn at 45°, and shows the position the curve would occupy if the stimulus interval equalled the response interval.

The steeper rise of the S_1 wave at all intervals in Text-fig. 9 must be due to a less asynchronous discharge of the S_1 ganglion cells. This decreased asynchronism shows that the shortening of latent period is greater for those ganglion cells which normally have the longer latent period. This shortening of latent period cannot be ascribed to a diminution in the preganglionic conduction time. In fact that would be longer than normal with stimulus intervals less than about 10 msec., for the second volley would be travelling in the relatively refractory period of the first. The synaptic delay must be shortened by some facilitating effect produced by the preceding volley either on the

preganglionic terminals or the ganglion cells [Eccles and Sherrington, 1931; Eccles, 1935*a*]. This effect appears to be maximal at about 5–10 msec. after the first volley and gradually decreases until it cannot be detected at an interval of 100–150 msec. When the facilitating effect is well developed the S_1 wave of the second volley may be increased considerably in height (cf. Text-fig. 9, interval 7·8 msec.). However, this does not signify that more ganglion cells have discharged impulses, for the area of the S_1 wave is probably unaltered, *i.e.* a greatly decreased asynchronism seems to be an adequate explanation of the increased potential.

Actually the facilitating effect produced by the preceding volley might reach a maximum earlier than 5–10 msec., but at such short intervals the second volley would be at a disadvantage owing to the delay and diminution in size occasioned by the relatively refractory period of the preganglionic pathway (factors 1 and 2 above). Moreover, the refractory period of the ganglion cells themselves might also delay the appearance of this facilitating effect.

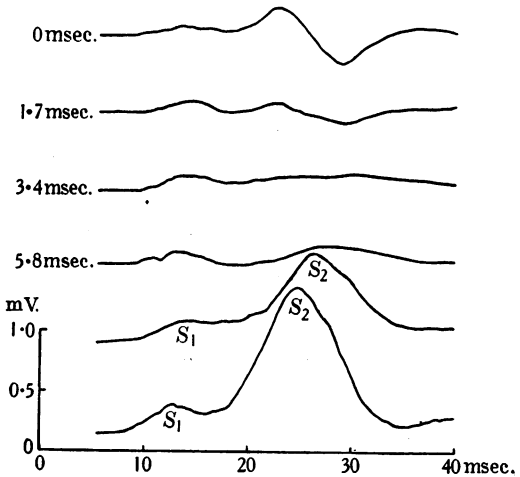
J. *The interaction between two volleys in different preganglionic fibres.*

Complications arising from the refractory period of the preganglionic pathway can be avoided by applying one stimulus to one branch of the annulus Vieussens and one to the other branch, the assumption being made that there are no anastomoses between the preganglionic fibres of the two branches on their course to the ganglion. In some preparations the absence of such anastomoses has been proved by the absence of action potential in one branch when the other branch has been stimulated (Pl. I, fig. 3, observation 5). Each branch usually gives a ganglionic action potential comprising all four waves, but in some preparations either branch is very small and only the S_2 wave may be identifiable. Rarely one or the other branch may be absent.

A typical series of observations is shown in Pl. II, fig. 7, stimuli being applied at intervals from 32·0 msec. to simultaneity. It has already been argued that the recorded action potential approximates to the sum of the action potentials that would be recorded from each individual ganglion cell. Hence in a series of observations such as Pl. II, fig. 7, subtraction of the action potential of the larger first volley from the combined action potential leaves as a remainder the approximate action potential added by the second volley. In Text-fig. 11 these subtracted action potentials are shown for various intervals, and it will be seen that the

S_1 and S_2 waves behave very differently from one another. At all intervals the subtracted S_1 wave is smaller but otherwise does not greatly differ from the S_1 wave produced by the second volley alone, while the subtracted S_2 wave is greatly affected by the interval between the two volleys.

At an interval of 3·4 msec. the very small subtracted action potential shows that the second volley is setting up a discharge from very few S_2 ganglion cells. The other ganglion cells must fail to respond on account



Text-fig. 11. The first four curves show the action potentials set up by the second stimulus at the indicated intervals after a first stimulus applied to the other (larger) branch of the annulus, as determined by subtracting the first action potential from the combined action potential (cf. Pl. II, fig. 7). The last two curves show the action potentials (S_1 and S_2 waves marked) set up by the second and the first stimuli alone, the times of all stimuli being synchronized at the zero of the time scale.

of the refractory state set up by their response to the first volley; hence most of the S_2 ganglion cells which are excited to discharge impulses by the second volley alone, are also similarly excited by the first volley alone, *i.e.* on the S_2 ganglion cells the overlapping distribution of the preganglionic fibres of the two branches of the annulus must be so great that the response to the smaller second volley is almost totally occluded by the first. Now, when the stimuli are simultaneous, the subtracted curve seems to indicate the presence of an S_2 wave much larger than at an interval of 3·4 msec. (Text-fig. 11), a result which cannot of course be due to a diminished occlusion at simultaneity, for that, if anything, will be increased. The only probable

explanation is that with simultaneous stimuli the apparent increase in the S_2 wave of the subtracted curve is due to the earlier discharge of many ganglion cells which would in any case discharge impulses in response to the first volley alone. On such an explanation by the forward displacement of the S_2 action potentials, the increased S_2 wave of the subtracted curve should be followed, as it invariably is (cf. Text-fig. 11), by a trough of approximately equal area. Thus it seems that a shortening of the S_2 latent period has resulted from the combined bombardment of the two simultaneous volleys. This shortening of the latent period is not appreciable at the beginning of the S_2 wave, but the S_2 summit is 0·7 msec. earlier than with the first volley alone, and the descending part is still earlier, *i.e.* the greatest shortening occurs with those ganglion cells which normally have the longest latent periods.

The effect responsible for this shortening of latent period must occur at the point where the different preganglionic fibres converge, *i.e.* at the ganglion cell, and the shortening must occur at the expense of the true synaptic delay (as defined above). This is still more evident if the latent period of the response of the S_2 ganglion cells to the second volley be considered, for this S_2 summit is 1·3 msec. later than the S_2 summit for the first volley alone (Text-fig. 11), and therefore the latent period of these ganglion cells is shortened by about 2·0 msec. by the combined bombardment of the two volleys.

When the second volley is set up 1·7 msec. after the first (Text-fig. 11) the small wave followed by a trough indicates that it still produces a slight shortening of the latent period of the response to the first volley, but with a 3·4 msec. interval no such effect is detectable. Since the preganglionic pathways to the ganglion from the two branches of the annulus are practically identical, the above intervals will also obtain between the arrival at the preganglionic terminals of corresponding impulses of the two volleys. Now in this experiment the shortest calculated synaptic delay for S_2 ganglion cells is about 6·5 msec. and for the summit of the S_2 wave the value is about 7·5 msec., both times being calculated for combined stimulation of both branches of the annulus. If the whole of this calculated synaptic delay—averaging about 7·5 msec.—were due to the time of summation of the excitatory effects of the temporally dispersed impulses incident on the ganglion cells [cf. Eccles and Sherrington, 1931], the second volley should produce an appreciable shortening of the synaptic delay of the first volley, even when more than 4 msec. later—theoretically the shortening should occur with all intervals up to 7·5 msec. It seems therefore that all the calculated

synaptic delay is not a summation time on the ganglion cells. Presumably part may be accounted for as a preganglionic transmission time longer than that allowed for in the conventional calculation (section F), but a full treatment of synaptic delay must be deferred until further evidence is brought forward.

The subtracted curves of Text-fig. 11 are typical in showing that the S_1 preganglionic fibres of the two branches of the annulus overlap to a much smaller extent in their distribution to the ganglion cells. Moreover, when the stimuli are simultaneous, there is practically no shortening of synaptic delay.

K. *Removal of the blood supply to the ganglion.*

When the blood supply to the ganglion is cut off by the division of its vessels, the latent period of the ganglionic action potential lengthens usually by 1–2 msec., all its constituent waves become slower, and the refractory period shows a corresponding lengthening. The maximum changes are reached within a few minutes, and thereafter there is no significant temporal change, but the declining ganglionic potentials indicate that the synaptic transmission is gradually becoming blocked, a process which usually takes several hours for completion. When special precautions were not taken to minimize a fall of temperature of the excised ganglion, considerably larger temporal changes were observed [Eccles, 1933], and were thought to provide an explanation for the discrepancy between the long values determined for synaptic delay and refractory period by Bishop and Heinbecker [1932] on excised ganglia, and the short values of Brown [1934] and the present paper. But now this discrepancy still appears to be without a complete explanation, for, as Bishop and Heinbecker's excised ganglia were kept at 37° C., excision should have been associated only with the small changes noted above.

When the carotid is occluded above and below the origin of the ganglionic vessels from the region of the carotid sinus, the lengthening of all the ganglionic processes has usually been much less than after complete excision. Thus it seems that a very small collateral blood supply is sufficient to prevent most of the asphyxial change in the ganglion.

Conclusions.

All the preceding experimental evidence indicates that the four S waves are produced by four groups of ganglion cells which are excited to discharge by four corresponding groups

of preganglionic fibres. These groups may be distinguished from one another by the following criteria, none of which, however, serves for rigorous discrimination, as there is a considerable overlap in the properties of adjacent groups:

(1) Rates of preganglionic conduction (section D).
(2) Thresholds of preganglionic fibres (section E).
(3) Synaptic delay in the ganglion (section F).
(4) Rates of postganglionic conduction (section G).
(5) Distribution in the different branches from the ganglion (section H).
(6) Refractory periods (section I).
(7) Shortening of synaptic delays (sections I and J).
(8) Degree of occlusion (section J).

Presumably the four groups of preganglionic fibres differ only in regard to size and medullation, S_1, S_2 and S_3 being medullated and in descending order of size, and S_4 being still smaller and probably non-medullated [cf. Bishop and Heinbecker, 1932]. However, as seen above, there is no sharp differentiation between S_1, S_2 and S_3 by means of preganglionic properties alone. Histologically the sizes of the ganglion cells of the human superior cervical ganglion have been used as the criterion for their division into three groups [de Castro, 1932]. The largest cells comprise about 27 p.c. of the total and probably are the S_1 ganglion cells. The largest group are those of intermediate size (50 p.c.) and presumably are the S_2 group, the smallest belonging to the S_3 and S_4 groups (23 p.c.). There is, however, no structural basis for the physiological differences between the various groups of ganglion cells. Presumably the occlusion between the two branches of the annulus Vieussens is greater with S_2 than with S_1, because of the more extensive overlap in the distribution of the two respective groups of S_2 preganglionic fibres, but the structural basis for the variations in synaptic delay and its shortening are unknown.

It seems very likely that the four groups subserve different functions and this should be the basis of their ultimate differentiation, but in the present research this has been investigated no further than in confirmation of Bishop and Heinbecker's statement that the S_1 group is distributed to the nictitating membrane, Müller's muscle and the dilator of the pupil. Presumably this composite functional character is related to the frequently observed division of the S_1 wave into two or three partly separated subsidiary waves (Pl. I, fig. 2). Bishop and Heinbecker further showed that S_2 was largely vaso-constrictor and pilomotor, while S_3 and S_4 are still of unknown function.

Our results show no appreciable functional overlap between the various groups, *e.g.* S_1 preganglionic fibres appear to be in functional relationship only with S_1 ganglion cells, but the experiments do not prove the absence of all overlap.

SUMMARY.

This is the first paper of a series in which analysis of the action potentials of sympathetic ganglia is used in the study of synaptic transmission.

The main potential wave which a preganglionic volley produces in the superior cervical ganglion is a spike potential set up by impulses discharged from the ganglion cells along the postganglionic fibres. In addition a late negative potential wave and a still later positive wave are produced in the ganglion, but they differ from the above spike potential both by their relatively slight spread along the postganglionic trunk and by their much greater sensitivity to the action of nicotine.

The spike potential wave is composite, being usually separable into four component waves (confirming Bishop and Heinbecker), called S_1, S_2, S_3 and S_4, each of which corresponds to a discrete group of ganglion cells supplied apparently exclusively by its own group of preganglionic fibres.

Preganglionic conduction rate, preganglionic threshold, synaptic delay, postganglionic conduction rate, distribution of postganglionic fibres, refractory period of ganglion cells, shortening of synaptic delay by a previous volley, and the degree of occlusion between the two branches of the annulus Vieussens are the properties which have been used in discriminating between the S_1, S_2, S_3 and S_4 groups.

Thus the synaptic delay and refractory period are each about 3 msec. for the S_1 group and 6 msec. for the S_2 group, and the preganglionic conduction velocities are respectively about 20 and 12 m. a second. Further, the S_1 group, which is distributed to structures in the orbit, is distinguished from the other groups by the shortening of synaptic delay of the response to a volley which follows a previous volley by less than 150 msec.

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EXPLANATION OF PLATES I AND II.

PLATE I.

Fig. 1. Ganglionic action potentials set up by a single maximal preganglionic volley, the small stimulus artefacts being approximately signalled by the arrow. Observation 1 shows the positive wave and the latter part of the negative wave before their removal by nicotine. Observation 2 is taken after painting the ganglion with 0·01 p.c. nicotine, observation 3 after 0·1 p.c. nicotine, and observations 4 and 5 show the complete removal of the action potential by 0·2 p.c. nicotine. The amplification with observation 5 is 3·9 times that with the other observations. In this and all subsequent figures 1 d.v. equals 10 msec.

Fig. 2. Postganglionic action potentials set up by a single preganglionic stimulus applied through electrodes either 10·7 cm. (observations 1 and 3) or 3·8 cm. (observations 2 and 4) from the ganglion. The stimulus strength is 50 (arbitrary units) for observations 3 and 4, and 100 for observations 1 and 2. The arrows mark the summits of the component waves of the action potentials in observations 1 and 2.

Fig. 3. Preganglionic action potentials of monophasic type, the conduction distance being 9·0 cm. The strength of stimulus is 50 for observation 1, 20 for 2, 10 for 3, and 5 for 4, the amplification for this observation being 1·9 times that for the other observations. With observation 5 the stimulus is applied to one branch of the annulus Vieussens and the leading electrodes are on the other branch, the stimulus strength being 50. There is no trace of an action potential which could be due to impulses reflected from the superior cervical ganglion, but there is evidence of a small early potential which could come from the middle cervical ganglion.

Fig. 4. Ganglionic action potentials elicited by single preganglionic stimuli of various strengths: observation 1, 3·3; 2, 5·0; 3, 20; 4, 10; 5, 100; 6, 50. With observation 1 the amplification is 3·8 times that with the other observations. The numbers indicate the respective S waves, and in observations 1 and 2 N indicates the late negative wave.

Fig. 5. Action potentials in the short ciliary nerve set up by a single stimulus to the cervical sympathetic. It is almost entirely an S_1 spike, though there is a small later wave which is possibly an S_2 spike, followed by still smaller waves.

PLATE II.

Fig. 6. Ganglionic action potentials set up by preganglionic stimuli of varying strengths. With observations 1 and 2 the earthed lead is on the ganglion and the grid on the main postganglionic trunk, the stimulus strengths being 5 and 20 respectively. With observations 3, 4 and 5 the grid lead is on the isolated external carotid branch of the superior cervical ganglion and the earthed lead is on the ganglion, the stimulus strengths being 5·0, 6·7 and 50 respectively. With observations 6 and 7 the grid lead is on the isolated grey ramus from the superior cervical ganglion to the upper cervical nerves, and the earthed lead is on the ganglion, the stimulus strengths being 20 and 5 respectively. The amplifications of records 2, 5 and 6 are equal, those of 1, 4 and 7 are 4·1 times greater, and that of 3 is 7·6 times greater.

Fig. 7. Postganglionic action potentials set up by maximal stimuli applied to each of the two branches of the annulus Vieussens, that to the smaller branch being second, the intervals being for observation 1, 32 msec.; for 3, 15·8 msec.; for 4, 7·9 msec.; for 5, 3·9 msec.; and for 8, 0·0 msec. Observations 2 and 6 show the response to the second stimulus alone, and observation 7 that to the first alone.

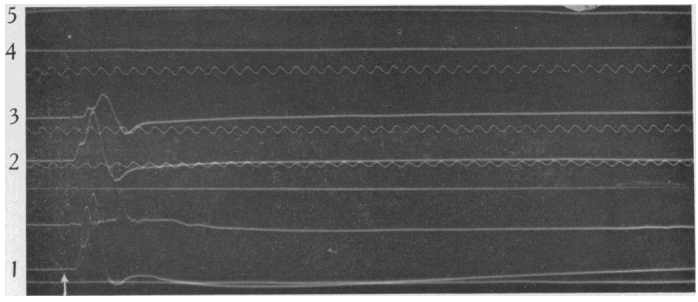

Fig. 1.

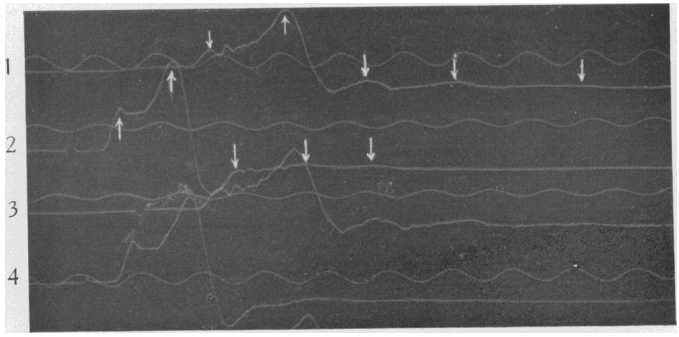

Fig. 2.

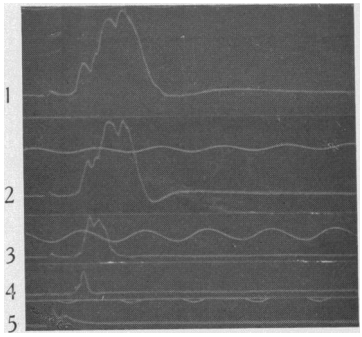

Fig. 3.

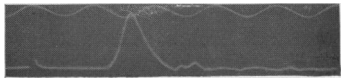

Fig. 5.

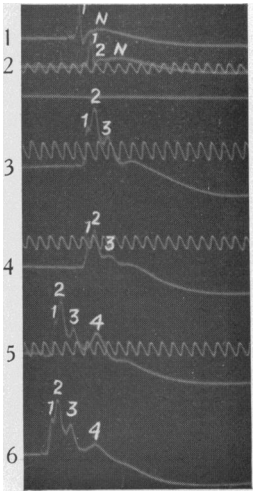

Fig. 4.

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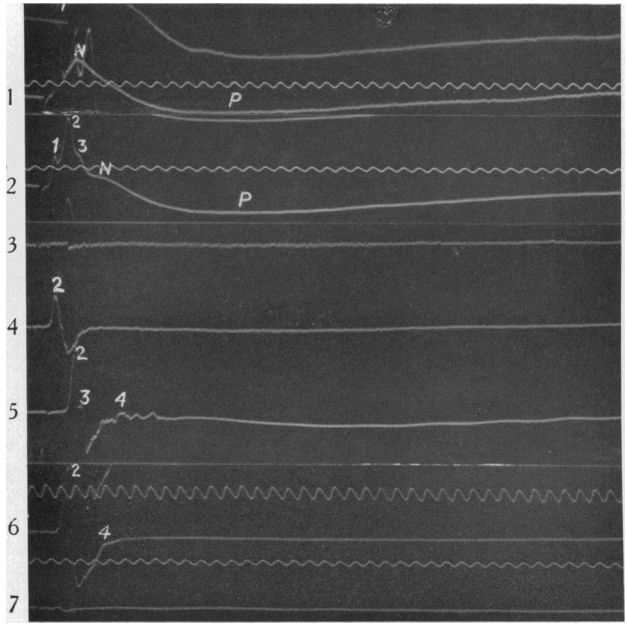

Fig. 6.

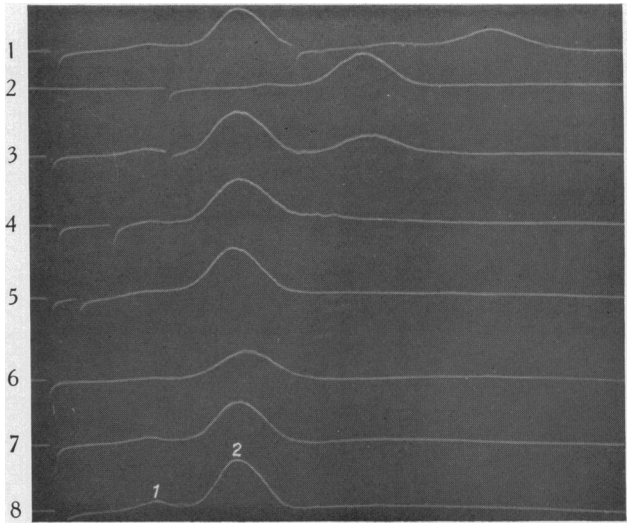

Fig. 7.