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A STUDY OF THE DEPRESSOR AND PRESSOR COMPONENTS OF THE CAT'S CAROTID SINUS AND AORTIC NERVES USING ELECTRICAL STIMULI OF DIFFERENT INTENSITIES AND FREQUENCIES

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Distension of the carotid sinus or the aortic arch causes a reflex fall in blood pressure. If records of electrical activity are taken from the carotid sinus or aortic nerves during the stimulus of distension many fibres of different size can be seen to fire. It is generally assumed that these impulses in diverse barosensory fibres are all concerned in the depressor reflex, and that together they account fully for the reflex. Furthermore, it is held that the depressor effects of electrical stimulation of the carotid sinus and aortic nerves are to be attributed to excitation of barosensory fibres.

Some idea of the function of different sized fibres can be obtained from experiments in which the nerves are stimulated electrically with carefully graded intensities of stimulus. Such an experiment was carried out by Douglas, Innes & Kosterlitz (1948, 1950) on the cat's carotid sinus nerve and yielded two curious findings: first, that little depressor function could be attributed to the large barosensory fibres so prominent in action potential records (and whose receptor behaviour has frequently been studied as typifying the barosensory depressor afferent); and secondly, that much of the depressor activity of this nerve seemed to be attributable to fibres smaller than the barosensory fibres that have been seen in action potential records.

Moreover, Neil, Redwood & Schweitzer (1949*a*, *b*) have observed that while stimulation of the cat's carotid sinus or aortic nerve after chloralose usually caused a rise in blood pressure, the effect sometimes became depressor when the pulse duration of the stimulus was increased. Although these workers do not make the point, this observation also suggests the presence of comparatively small depressor fibres in these nerves.

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In the present experiments the cat's carotid sinus and aortic nerves have been excited electrically with different intensities and frequencies of stimulation. The experiments were undertaken to compare the depressor fibre constitution of the aortic nerve with that of the carotid sinus nerve, and to examine how the reflex effects of the different fibres in both nerves vary with the frequency of firing.

METHODS

The experiments were done on cats. After induction with ethyl chloride and ether they were anaesthetized with urethane 1·2 g/kg intravenously or decerebrated. Records were made on a kymograph. Systemic blood pressure was measured from a femoral artery with a mercury manometer. Respiration was recorded by a tambour and lever activated by a stethograph tied round the lower chest, or by measuring diaphragm movements with the phrenograph described by de Candole, Douglas & Spencer (1950). An estimate of respiratory movements was obtained by measuring the lever excursions. In some experiments, in order to exclude the influence of respiratory movements on blood pressure, the chest was opened on both sides and the lungs were artificially ventilated through a Starling pump. The minute volume was then adjusted so that spontaneous gentle respiratory movements of the thorax were present.

The right or left carotid sinus nerve was dissected well clear of surrounding structures. The cut central end was stimulated some 6 mm or more from the junction with the glossopharyngeal nerve. The left aortic nerve was identified as it joined the superior laryngeal nerve close to the nodose ganglion, and its cut central end was stimulated 1–2 cm below this point. Square wave stimuli were delivered through a transformer with 5 kΩ across the output. Each stimulus was visualized on a cathode-ray oscilloscope and adjusted to the desired intensity before its application. During application of the stimulus, when there was some further shorting by the nerve, the voltage fell by less than 10% of its monitored value. The frequency calibration of the stimulator was checked by photographing trains of impulses displayed on the cathode-ray oscilloscope. The frequencies used were in the series 1 shock/4 sec, 1 shock/2 sec, 1-2-4-8-16-32-64-125-250-350 shocks/sec.

RESULTS

Carotid sinus nerve

The right or left carotid sinus nerve was stimulated with shocks of varying intensity and frequency in six decerebrate and seven anaesthetized cats.

Intensity of stimulation. In two decerebrate and in three anaesthetized cats increasingly stronger stimulation at a constant frequency (64 or 125 shocks/sec) led to the following sequence in the response of the arterial blood pressure: first, a small depressor effect; then with slightly stronger stimulation a pressor response; and finally, with further increase in the intensity of stimulation, a large depressor response (Fig. 1). The initial depressor response was obtained only with stimuli which lay within a very narrow range of intensity. The extent of the fall of blood pressure was usually between 5 and 10 mm Hg. In one decerebrate cat it was 16 mm Hg. Respiration was not affected by this weak stimulation; respiratory stimulation appeared, however, with the onset of the pressor response and was maximal with a stimulus intensity that produced the maximal pressor effect. The greatest respiratory stimulant effect was always seen during application of the stimulus, but in many animals

increased respiratory movements persisted for some time after stimulation had ceased. This secondary response occurred in animals breathing normally and also in others with open chest and artificially ventilated. It was never marked in the normally breathing animals, probably because the increased amount of CO_2 washed out of the lungs during stimulation tended to reduce respiratory centre activity.

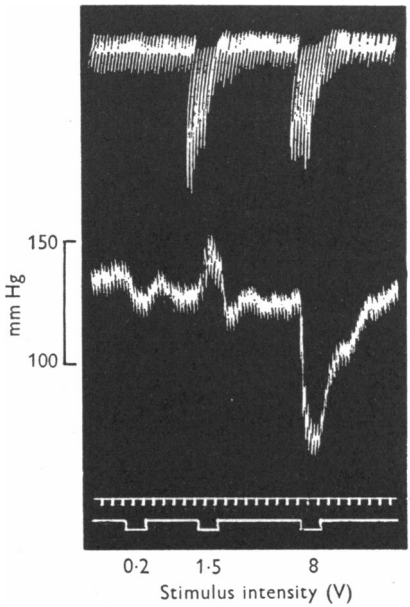

Fig. 1. Cat. Decerebrate: open chest, artificial ventilation. Tracings from above down are: (I) spontaneous respiratory movements (stethograph; inspiration downwards); (II) systemic B.P.; (III) 10 sec time marker; (IV) stimulus marker. The figure shows responses to stimulation of the right carotid sinus nerve with increasing stimulus intensities. The frequency of stimulation was 64 shocks/sec and the pulse width 0·13 msec throughout the record.

In three decerebrate and four anaesthetized cats the initial depressor response was absent: the feeblest effective stimuli resulted in a pressor response and in stimulation of respiration. Sometimes pure pressor responses did not occur but the responses were diphasic. In such instances blood pressure first rose during stimulation and then fell. When the stimulus intensity was further increased the pressor component diminished and the response became more purely depressor. In one decerebrate cat no pressor responses were seen with any of the stimuli from threshold to maximal intensity, but stimuli of intermediate intensity gave depressor responses feebler than were obtained with the weaker and stronger stimuli. The sequence of events was similar to that sometimes seen on stimulation of the aortic nerve, which is illustrated in Fig. 4.

Frequency of stimulation. The effect of varying the frequency of stimulation was tried for the three blood-pressure responses evoked by the various intensities of stimulation. The initial depressor responses to feeble stimulation were generally so small that the relationship between stimulus frequency and the blood-pressure response could be determined with accuracy in only one experiment. In this experiment, which was carried out on a decerebrate cat, the greatest fall in blood pressure was obtained with a frequency of 64 shocks/sec. Raising the frequency to 125 or 250 shocks/sec did not appreciably enhance the response. Lowering the frequency from 64 shocks/sec diminished it, and below a frequency of 16 shocks/sec no effect was detected.

With stimuli of a strength causing pronounced pressor responses detectable rises in blood pressure were usually obtained at frequencies as low as 2–4 shocks/sec. In order to obtain maximal rises the frequency had to be increased to 16 shocks/sec in some cats, and to 32 shocks/sec in others. With a further increase in the frequency of stimulation the pressor responses usually became smaller (Figs. 2, 3).

With the strong stimuli which gave maximal depressor responses, a depressor effect was usually detectable at frequencies as low as 1–2 shocks/sec; the effect increased as the frequency of stimulation increased up to about 64 shocks/sec; a further increase in frequency to 125 or 250 shocks/sec did not lead to a further increase in the depressor response.

The graphs of Fig. 3 illustrate how the different blood-pressure responses varied with the frequency of stimulation when the stimulus intensity was optimal for depressor or pressor effects. With such stimulus intensities it can be seen that frequency variation caused only quantitative differences. On the other hand, when stimuli were used of an intensity giving mixed or ill-defined responses, (e.g. between 1·5 and 8 V in Fig. 1), frequency change often caused the blood-pressure response to change in sign. In such experiments the lower frequencies caused pressor effects and the higher frequencies caused depressor effects.

With stimuli of pressor intensity the respiratory response occurring during stimulation was maximal at frequencies about 16–32 shocks/sec, and in any one cat the frequency causing a maximal respiratory response was also that which had the greatest pressor effect. Frequencies of stimulation above 32 shocks/sec tended to lessen the respiratory response and there was usually a fair parallelism between respiratory and pressor effects (Figs. 2, 3).

With stimuli of maximal depressor intensity the respiratory response to frequency changes was similar to that just described for the pressor stimulus: the frequency causing the maximal respiratory response lay again between 16 and 32 shocks/sec, i.e. at a frequency well below that causing the maximal depressor response.

In most experiments respiration was excited by frequencies of stimulation

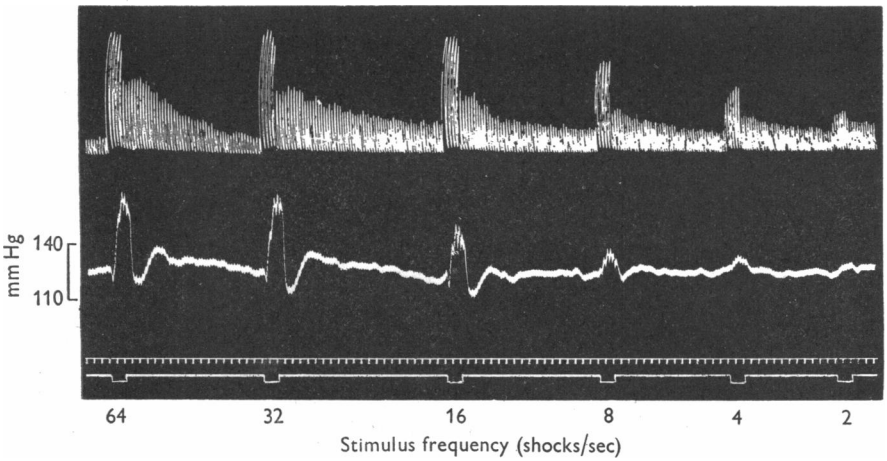

Fig. 2. Cat. Urethane: tracings from above down are: (I) respiratory movements (phrenograph; inspiration upwards); (II) systemic B.P., (III) 10 sec time marker; (IV) stimulus marker. The figure shows responses to stimulation of the right carotid sinus nerve at different frequencies with a stimulus of 4 V and 0·13 msec pulse width.

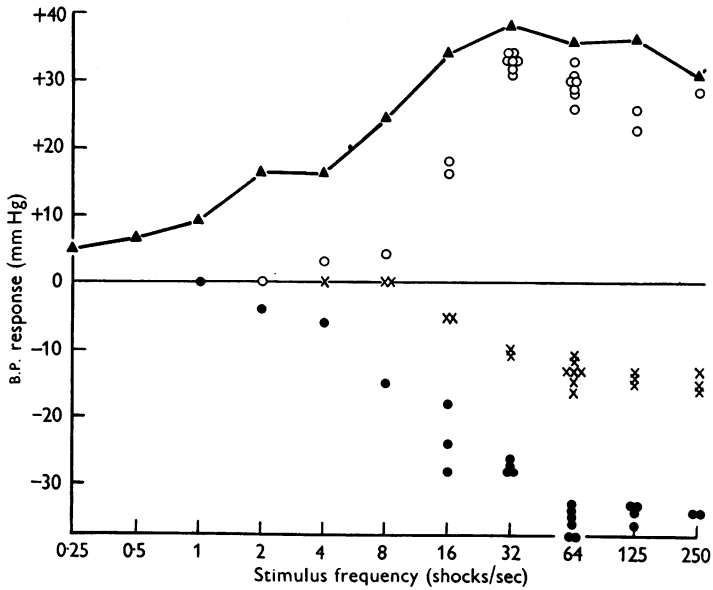

Fig. 3. Carotid sinus nerve experiments. Graph showing the variation of initial depressor (×), pressor (○) and final depressor (●) responses with frequency of stimulation. Each series was obtained using a stimulus of constant intensity. The initial depressor responses were obtained with a stimulus of 0·5 V, the pressor responses with 2·0 V and the final depressor responses with 8 V. The pulse width of each stimulus was 0·13 msec. The values (○) and (●) were from a cat under urethane. The values (×) from a decerebrate cat. The respiratory stimulant effects accompanying the pressor responses are expressed in arbitrary units by the curve ▲—▲.

below those effective in influencing the blood pressure as shown in Fig. 3, but exceptionally the blood-pressure response was the more evident at low frequencies.

The secondary respiratory response increased with frequencies higher than those optimal for the response seen during the stimulus. We have not examined closely the relationship between frequency and the magnitude of the secondary response.

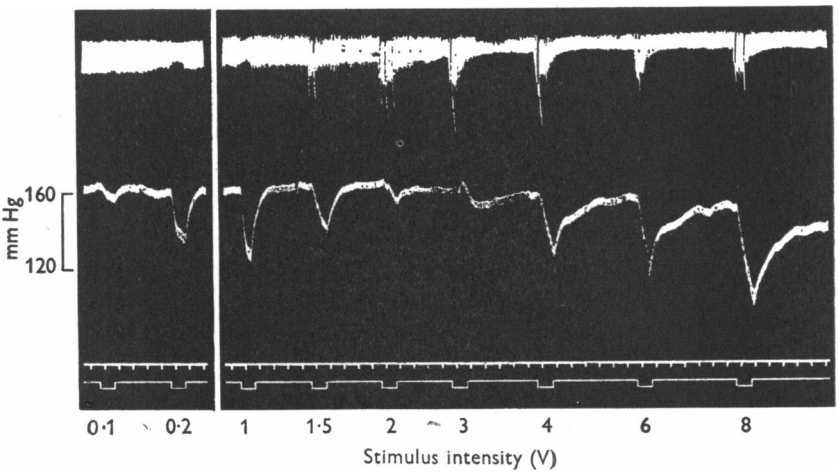

Fig. 4. Cat. Urethane: tracings from above down are: (I) respiratory movements (stethograph, inspiration downwards); (II) systemic B.P.; (III) 10 sec time marker; (IV) stimulus marker. The figure shows responses to stimulation of the left aortic nerve with increasing stimulus intensities. The frequency of stimulation was 64 shocks/sec throughout, and the pulse width 0·13 msec.

Aortic nerve

The left aortic nerve was stimulated with shocks of various intensities and frequencies in four decerebrate and nine anaesthetized cats.

Intensity of stimulation. Stimuli of constant frequency (either 64 or 125 shocks/sec) were again used. Apart from the fact that strong depressor activity was obtained with weak stimulation in all experiments, the results obtained on aortic nerve stimulation were similar to those found on carotid sinus nerve stimulation. In two decerebrate and five anaesthetized cats increasing the intensity of stimulation produced first depressor; then pressor; and finally, depressor responses. The small depressor effects obtained by stimulation at threshold intensity grew with increasing stimulus strength to become falls of 20–40 mm Hg. Further increase in stimulus strength either caused this depressor effect to diminish (Fig. 4) or, more often, to change to a pressor response as in the carotid sinus nerve experiments illustrated

by Figs. 1 and 2. Finally, with strong stimulation, clear-cut depressor responses were again produced. Sometimes these responses were preceded by a small pressor effect.

With the initial depressor response produced by feeble stimulation there was either no change in respiration or a decrease in the amplitude of the respiratory movements. However, on increasing the stimulus strength to cause the initial depressor response to diminish, or to evoke a pressor response, an excitant effect on respiration was always observed.

As was found in the carotid sinus nerve experiments, the growing pressor effect of the stimulus was paralleled by the growing respiratory stimulant effect; so that when the stimulus was maximally pressor (or minimally depressor in those experiments such as shown in Fig. 4 where there was not a frank pressor response) it was also maximally excitant on respiration. The strong stimulation causing depressor effects did not cause any further appreciable change in the strong respiratory response.

Frequency of stimulation. With feeble stimuli, sufficiently strong to elicit a depressor response but not strong enough to stimulate respiration, the threshold frequency varied in different cats from 8 shocks/sec to 32 shocks/sec and was usually 16 shocks/sec. With increasing frequency the depressor response grew and reached its maximum at a frequency of 64–125 shocks/sec. Increase in the frequency to 250 shocks/sec did not cause any further change in the response. A typical experiment is illustrated in Fig. 5 (crosses).

With stimuli of intermediate intensity causing maximal pressor responses, effects were obtained at frequencies from 2 shocks/sec to 250 shocks/sec. Maximal responses were obtained with frequencies from 16 to 32 shocks/sec. Frequencies higher than this tended to diminish the response. A representative experiment is illustrated in Fig. 5 (open circles).

With strong stimuli, depressor responses were obtained with frequencies as low as 1 shock/sec and reached a maximal value at 64 or 125 shocks/sec (Fig. 5, closed circles).

With stimuli of intensity between those found to give clear-cut pressor or depressor effects, variation in the sign of the response occurred with variation in frequency as happened in the carotid sinus nerve experiments. Once more, the higher frequencies favoured the appearance of depressor responses and the lower frequencies pressor responses. The effect is illustrated by Fig. 6.

The influence of the frequency of stimulation on the respiratory effects of stimuli of intermediate or strong intensities was similar to that found in the carotid sinus nerve experiments. With both intensities of stimulation the threshold for respiratory effects was about that for the blood-pressor responses, and the maximal respiratory effect was seen when the frequency of stimulation was about 16–32 shocks/sec.

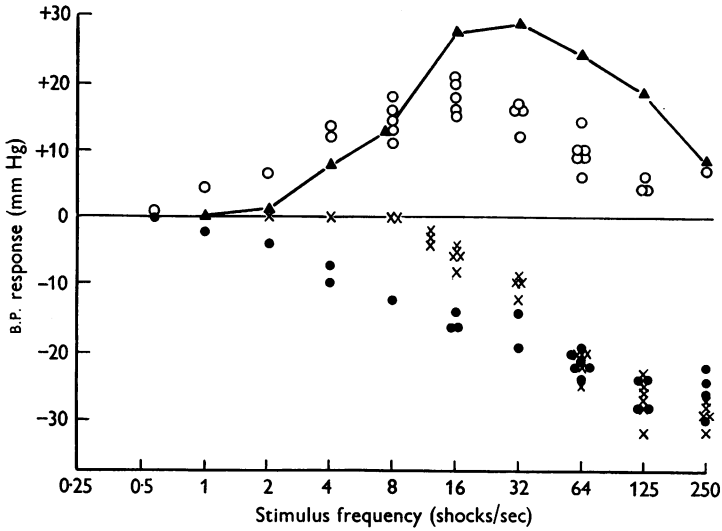

Fig. 5. Aortic nerve experiments. Graph showing the variation of initial depressor (×), pressor (○) and final depressor (●) responses with frequency of stimulation. Each series was obtained using a stimulus of constant intensity. The initial depressor responses were obtained with a stimulus of 1 V, the pressor responses with 4 V and the final depressor responses with 8 V. The pulse width of each stimulus was 0·13 msec. The values were all obtained on urethanized cats, (×) and (●) from one cat and (○) from another cat. The respiratory stimulant effects accompanying the pressor responses are expressed in arbitrary units by the curve ▲—▲.

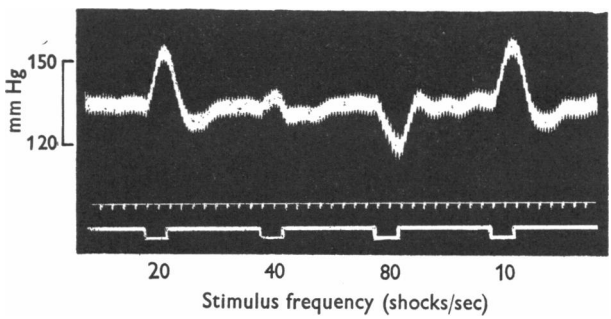

Fig. 6. Cat. Urethane: open chest, artificial ventilation. Tracings from above down are: (I) systemic B.P.; (II) 10 sec time marker; (III) stimulus marker. The figure shows reversal of the response to stimulation of the left aortic nerve with change in frequency. The intensity of the stimulus (6 V and 0·13 msec pulse width) lay between those optimal for pressor and for final depressor responses.

Effects of hypo- and hyperventilation on the responses of the carotid sinus and aortic nerves. The blood-pressure responses to sinus or aortic nerve stimulation were found to depend to a certain extent on the state of ventilation of the animal. In one urethanized cat the response to stimulation of the left aortic nerve changed from depressor to pressor as the experiment progressed without the intensity or frequency of the stimulus being altered. This happened when the artificial ventilation had become insufficient, and very vigorous breathing movements were present. When ventilation was increased the effect of aortic

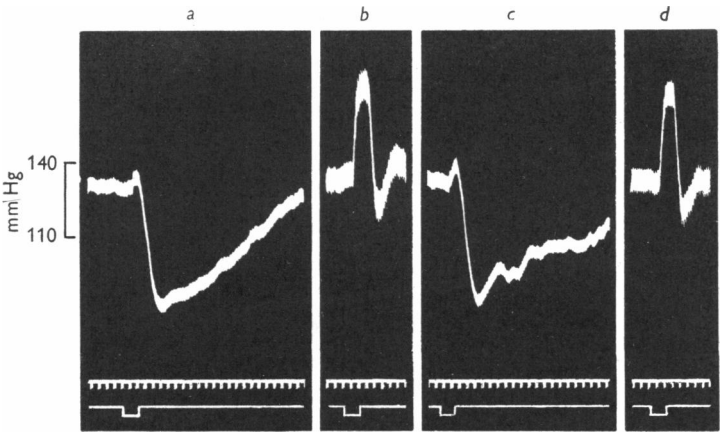

Fig. 7. Cat. Urethane: open chest, artificial ventilation. Tracings from above down are: (I) systemic B.P.; (II) 10 sec time marker; (III) stimulus marker. The figure shows the effect of changes in ventilation on the responses to stimulation of the left aortic nerve at 8 V and 0·5 msec pulse width at 64 shock/sec. During records (*a*) and (*c*) the cat was over-ventilated, during (*b*) and (*d*) it was under-ventilated. The responses were obtained consecutively.

nerve stimulation became again depressor, and by varying the degree of ventilation, the response to the same stimulus could be made pressor or depressor (Fig. 7). In four other cats the effect of under- and over-ventilation was tried. In two anaesthetized cats no reversal of any of the responses could be brought about by changes in ventilation, but in one anaesthetized cat and one decerebrate cat, a reversal of the effect of aortic nerve stimulation occurred when a stimulus was used just strong enough to elicit a final depressor response. Stronger stimuli giving more pronounced depressor responses gave no pressor effects even on gross under-ventilation.

DISCUSSION

Assuming that the excitability of nerve fibres varies with their diameters, our results indicate that in the aortic nerve there are three distinguishable groups: large depressor fibres, smaller pressor fibres, and depressor fibres similar in

size or yet smaller than the pressor fibres. Fig. 8 shows a schematic fibre population which could account for our results. The aortic nerve of the cat is known to contain barosensory fibres responsible for the classical depressor reflex on change of the intra-aortic pressure, and chemosensory fibres from the aortic glomus whose activity causes a rise in blood pressure and stimulation of respiration (Gernandt, 1946). Hence the simplest explanation of our results is that the pressor effects (always accompanied by increased respiration) are attributable to the chemosensory fibres and the depressor effects to barosensory fibres. The depressor effects seen with feeble stimulation and the pressor effects with somewhat stronger stimulation tally with Paintal's (1953) conduction velocity experiments, which have shown the presence of large barosensory and somewhat smaller chemosensory fibres in the cat's vagus and depressor nerves.

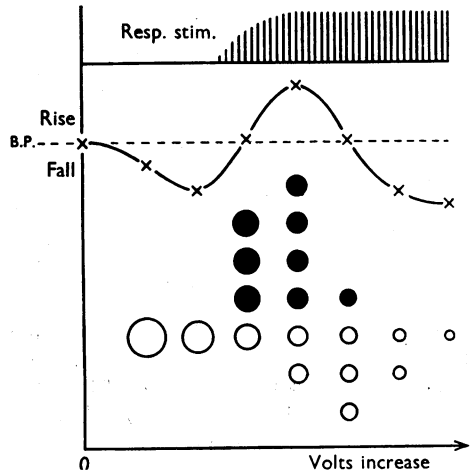

Fig. 8. A summary of the respiratory and blood-pressure responses seen on stimulating the aortic nerve with increasingly stronger stimuli, and a schematic nerve fibre population which could account for the effects. The closed circles (●) represent pressor (and respiratory excitant) fibres. The open circles (○) represent depressor fibres.

His study, however, revealed no small barosensory fibres which would account for the depressor effects we have seen with high intensity stimulation. His experiments do not exclude the possibility that these small depressor fibres are barosensory for they may be smaller than any of those from which Paintal was able to record. Recently small depressor fibres have been found in the rabbit's aortic nerve whose excitability threshold is similar to that of the cat's aortic nerve fibres, and which conduct at 1 m/sec or less (Douglas, Ritchie & Schaumann, 1956), whereas the smallest fibres observed by Paintal conducted at 3 m/sec. On the other hand, these small depressor fibres in the cat's aortic nerve may not be barosensory fibres, but fibres serving one or

other of the well-known reflexes from the heart and lungs reviewed by Dawes & Comroe (1954). The demonstration that there are small depressor fibres of similar excitability in the carotid sinus nerves would, however, favour the view that they are baroreceptors—or at least fibres originating in the great vessels.

To judge from the effects of electrical stimulation, the carotid sinus nerve would seem to differ from the aortic nerve in its fibre constitution. The absence of substantial depressor responses to weak stimulation, which was noted previously by Douglas *et al.* (1950), has been confirmed in the present investigation: and any doubt that the earlier result was attributable to anaesthesia or inappropriate stimulus frequency has been removed. In other respects, the results obtained on stimulating the two nerves are similar. Using the same conditions of stimulation the stimulus intensities evoking the pressor (respiratory excitant) and high threshold depressor reflex were indistinguishable. The carotid sinus nerve, like the aortic nerve, is known to contain large barosensory fibres. It is not at all obvious why we should be able to demonstrate with any regularity depressor activity attributable to large fibres only in the aortic nerve. Perhaps the answer lies in the number of large fibres being different in the two nerves. De Castro's (1951) figures suggest that less than 3% of the medullated fibres in the carotid sinus nerve are of the very large type. We have no figures for the cat's aortic nerve, but it may contain more. There is an alternative possibility, namely, that the large barosensory fibres in the carotid sinus nerve are not depressor, but play a part in the other effects of sinus distension on central nervous activity (see Koch, 1931; Schweitzer, 1936; Dell, 1952). It is noteworthy that among splanchnic afferents it is the largest which induce the primary cortical responses (Amassian, 1951).

Our results on stimulation of the carotid sinus and aortic nerves in cats under urethane anaesthesia or decerebrate, in which a pattern of three distinct responses was usually obtained, are in contradiction to those seen by Neil *et al.* (1949*a*, *b*) on the decerebrate or normally nembutalized cat. They found only depressor effects in such preparations. The fact that they did not observe the pressor responses to intermediate intensity stimulation or the depressor effects with feeble stimulation in such preparations is probably due to their using different stimulus parameters. Their observation, that a pressor response to carotid sinus nerve or aortic nerve stimulation occurring in chloralose anaesthesia sometimes changed to a depressor response when the pulse duration of the stimulus was increased, might have its explanation in the pattern of depressor and pressor fibres which is envisaged for the carotid sinus nerve in the present paper and in the previous papers by Douglas *et al.* (1948, 1950) and the fibre population which we now suggest for the aortic nerve. However, the effect they obtained on the aortic nerve was caused by changing from a 1 msec pulse delivered 70 times per second, to 10 msec pulse delivered at

the same rate (that is to say, that during the period of stimulation current was passed for 10 out of each 14 msec). Such stimulation readily produces repetitive firing in the fibres in this nerve (Douglas & Ritchie, unpublished), and the reversal they observed may be due to this rather than to excitation of fibres with a higher threshold.

The relationship found between the frequency of stimulation and the depressor effects obtained with feeble stimulation probably gives a good indication of the frequency/response characteristic of the large fibre depressor mechanism. These fibres apparently only cause depressor effects when excited at frequencies about 16 shocks/sec, and exert their greatest effect at 64–125 shocks/sec.

The frequency/response characteristics of the smaller fibre mechanisms are not so readily determined, for these, whether pressor or depressor, have not been selectively stimulated. Some insight into their characteristics can be arrived at, however, by comparing the curves obtained at intermediate or high intensity stimulation with those obtained using feeble stimulation. When this is done, it becomes clear that the pressor fibres which are excited with intermediate intensity of stimulation exert their effects on blood pressure and respiration at comparatively low rates of firing. The lowest rate at which these effects were seen was about 1–4 shocks/sec and optimal responses were obtained at about 16 shocks/sec. The latter figure may underestimate the true optimum, for higher frequencies certainly introduce increasing depressor activity from the large fibres which are simultaneously stimulated. However, the optimal frequency for the pressor fibres is certainly less than that for the large depressor fibres, for pressor responses were often replaced by depressor effects on increasing the rate of stimulation.

It is obviously not safe to try to extract much information on the behaviour of the small fibre depressor mechanism from the curves obtained with yet higher intensity stimulation which involves the simultaneous excitation of the three functional fibre groups, but the comparatively low frequency required to give depressor effects with such stimuli suggests that these small depressor fibres are effective at considerably lower frequencies than the large depressor fibres. This raises the possibility that the different-sized depressor fibres are associated with the production of effects within the central nervous system with different time constants, the large fibres giving short-lived effects and the small fibres causing longer lasting changes. It should be pointed out that so far there is no direct evidence that the receptor functions of the large and small barosensory fibres are essentially different (Landgren, 1952).

It is an old observation that the vasomotor response to stimulation of a somatic nerve may vary in sign with the frequency of the stimulus (Gruber, 1917; Ashkenaz, 1939), and that low-frequency stimulation favours depressor effects and high-frequency pressor effects. Our results show that the response

to stimulation of the carotid sinus and aortic nerves may also change with stimulus frequency, but in the opposite way; for pressor responses to low-frequency stimulation are changed to depressor effects when the stimulus frequency is increased. The phenomenon of 'reversal' in somatic nerve experiments has been ascribed to greater temporal summation of impulses at higher frequency or to the presence in these nerves of fibres of different qualities (see Gordon, 1943). The reversals seen on changing the frequencies of stimulation of the carotid sinus and aortic nerves seem to result from the different frequency/response characteristics of the depressor and pressor mechanisms associated with these nerves. Frequency change had no qualitative effect when depressor fibres alone were excited, nor when a preponderance of pressor fibres or small depressor fibres were excited, but only when the intensity of the stimulus was in a range which stimulated a balance of the depressor and pressor fibres.

That the blood-pressure response to stimulation of the aortic nerve might, as we observed, be changed from a fall to a rise by underventilation seems most likely owing to asphyxia disturbing the balance between the pressor and depressor components simultaneously excited, in such a way as to favour the former. An analogous observation was made by Stewart & Pike (1907). They found that after clipping the arteries to the brain, to cause the disappearance of the depressor effect of central vagus stimulation, and subsequently restoring circulation, the first effect of vagal stimulation during recovery was a pressor response and only later did the normal depressor effect reappear.

SUMMARY

1. The blood pressure and respiratory responses to electrical stimulation of the carotid sinus and aortic nerves have been studied in cats under urethane anaesthesia or decerebrated. The effects of stimuli from threshold to maximal effective intensities and of frequencies of 1 shock/4 sec to 350 shocks/sec were determined.

2. Stimulation of the aortic nerve at low intensity caused a substantial depressor effect accompanied by no change or a slight inhibition of respiration. Somewhat stronger stimulation usually led to pressor effects accompanied by respiratory excitation. Still stronger stimulation caused the reappearance of depressor effects without further perceptible change in respiration.

3. These effects were considered to indicate the presence in the aortic nerve of large depressor fibres, small pressor fibres, and small depressor fibres—these last being probably yet smaller than the pressor fibres.

4. Experiments have been made to test how the initial depressor, the pressor and the final depressor responses obtained with the different intensities of stimulation of the aortic nerve vary with the frequency of the stimulus. The low threshold depressor effect was just detectable at about 16 shocks/sec and

maximal at 64–125 shocks/sec. The pressor effects were first obtained with rates of 1–4 shocks/sec and were maximal at about 16 shocks/sec. The depressor effect from high intensity stimulation was threshold at 1–2 shocks/sec and maximal at about 64–125 shocks/sec. These results indicate differences in the frequency/response characteristics of the reflexes served by different fibres.

5. The respiratory responses accompanying the pressor and the final depressor effects from aortic nerve stimulation usually showed two phases; a primary phase during the application of the stimulus, and a secondary response occurring after stimulation had ceased. The primary phase was threshold at about 1 shock/sec and maximal at about 16 shocks/sec. The secondary phase continued to grow with increasing frequencies above 16 shocks/sec.

6. The results obtained on electrical stimulation of the carotid sinus nerve were similar, except that depressor activity attributable to large fibres was not regularly obtained.

REFERENCES

AMASSIAN, V. E. (1951). Cortical representation of visceral afferents. *J. Neurophysiol.* **14**, 433–444.

ASHKENAZ, D. M. (1939). Nerve impulse frequency and its relation to vasomotor reflexes. *Amer. J. Physiol.* **125**, 119–129.

DE CANDOLE, C. A., DOUGLAS, W. W. & SPENCER, K. E. V. (1950). A method of recording movements of the diaphragm. *J. Physiol.* **111**, 21–22*P*.

DE CASTRO, F. (1951). Sur la structure de la synapse dans les chemocepteurs, leur mécanisme d'excitation et rôle dans la circulation sanguine locale. *Acta physiol. scand.* **22**, 14–43.

DAWES, G. S. & COMROE, J. H. (1954). Chemoreflexes from the heart and lungs. *Physiol. Rev.* **34**, 167–201.

DELL, P. (1952). Corrélations entre le système végétatif et le système de la vie de relation mésencéphale, diencéphale et cortex cérébral. *J. Physiol., Paris*, **44**, 471–557.

DOUGLAS, W. W., INNES, I. R. & KOSTERLITZ, H. W. (1948). Changes in arterial blood pressure caused by electrical stimulation of the sinus nerve in cats. *J. Physiol.* **108**, 17*P*.

DOUGLAS, W. W., INNES, I. R. & KOSTERLITZ, H. W. (1950). The vasomotor responses due to electrical stimulation of the sinus and vagus nerves of the cat and their modification by large doses of sodium pentobartitol (nembutal). *J. Physiol.* **111**, 215–230.

DOUGLAS, W. W., RITCHIE, J. M. & SCHAUMANN, W. (1956). *J. Physiol.* **132**, 187–198.

GERNANDT, B. E. (1946). A study of the respiratory reflexes elicited from the aortic and carotid bodies. *Acta physiol. scand.* **11**, Suppl. 35.

GORDON, G. (1943). The mechanism of the vasomotor reflexes produced by stimulating mammalian sensory nerves. *J. Physiol.* **102**, 95–107.

GRUBER, C. M. (1917). The response of the vasomotor mechanism to different rates of stimuli. *Amer. J. Physiol.* **42**, 214–227.

KOCH, E. (1931). *Die reflektorische Selbststeuerung des Kreislaufes.* Dresden: T. Steinkopff.

LANDGREN, S. (1952). On the excitation mechanism of the carotid baroceptors. *Acta physiol. scand.* **26**, 1–34.

NEIL, E., REDWOOD, C. R. M. & SCHWEITZER, A. (1949*a*). Pressor responses to electrical stimulation of the carotid sinus nerve in cats. *J. Physiol.* **109**, 259–271.

NEIL, E., REDWOOD, C. R. M. & SCHWEITZER, A. (1949*b*). Effects of electrical stimulation of the aortic nerve on blood pressure and respiration in cats and rabbits under chloralose and nembutal anaesthesia. *J. Physiol.* **109**, 392–401.

PAINTAL, A. S. (1953). The conduction velocity of respiratory and cardiovascular afferent fibres in the vagus nerve. *J. Physiol.* **121**, 341–359.

SCHWEITZER, A. (1936). *Die irradiation autonomer Reflexe.* Basel: S. Karger.

STEWART, G. N. & PIKE, F. H. (1907). Resuscitation of the respiratory and other bulbar nervous mechanisms with special reference to the question of their automaticity. *Amer. J. Physiol.* **19**, 328–359.