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THE CONDUCTION VELOCITIES OF RESPIRATORY AND CARDIOVASCULAR AFFERENT FIBRES IN THE VAGUS NERVE

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A preliminary account of the present work was given in 1952 (Paintal, 1952). Subsequent work has shown that those vagal afferent nerve fibres which were then believed to end in pulmonary vascular receptors (Whitteridge, 1948), in fact end in the right and left atria of the heart (Paintal, 1953*a*). These fibres can be divided into two groups, one of which has a presystolic series of action potentials, the other has not. These have been designated type A and type B atrial receptors respectively (Paintal, 1953*b*). The above investigations were extended, and the present paper describes the results of the study of the conduction velocity in 116 single vagal afferent nerve fibres.

METHODS

Dissection

All the experiments were performed on adult cats anaesthetized with intravenous chloralose (80 mg/kg). The general plan of dissection was the same as that described by Whitteridge (1948). After exposure of the right vagus in the neck, the margin of the skin was raised with hooks tied to dry string, and warmed (38° C) liquid paraffin B.P. poured on the area of dissection. The nerve was placed on a smooth glass plate with a black background.

For the preparation of single units the connective tissue sheath of the vagus was split and a thin bundle of fibres was separated and cut centrally near the nodose ganglion. It was then divided longitudinally into four or five smaller strands which were spread out on the glass plate with fine sharpened needles. The recording electrodes of silver wire were now inserted carefully under one nerve strand which was kept in position without attaching its free end to the distal electrode. The activity in the strand was amplified and fed to a double-beam cathode-ray tube and loud-speaker. If there was more than one active unit in the strand it was divided further with needles.

To determine the conduction velocity of the single afferent fibres, a 5 mm length of the whole vagal trunk was dissected out low down in the neck and placed on a pair of stimulating electrodes. At this stage the recurrent laryngeal nerve was cut where it lay beside the trachea in order to avoid the stimulation of the laryngeal muscles. Their action potentials would otherwise complicate the action potential record from the vagus nerve. The vagal trunk was then stimulated and the action potential so recorded was invariably compound in spite of the fact that only one single unit in the strand was responding to natural stimuli. This was to be expected, however, as there was

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usually a number of inactive live fibres in the strand containing the active one. The next and the most difficult step therefore was to try to eliminate as many of these inactive live fibres as possible by further subdivision of the already fine nerve strand. This step was accomplished by the use of a Zeiss binocular dissecting microscope giving a magnification of $\times 36$.

The nerve was subdivided by placing the sharp dissecting needles in the centre of the strand and separating the two divided parts with gentle pressure. This was repeated until single action potentials could be obtained by stimulation of the nerve trunk. After the final subdivision, and only then, the free end of the thin nerve strand was twisted around the distal electrode.

In early experiments, a thermocouple was used to measure the temperature of the nerve at three points along its length. Later this was replaced by a thermistor bridge circuit (Grieve, 1951) connected to three thermistors (Standard Telephones and Cables Ltd.) type F 2311-300. The output from the bridge was coupled to the galvanometer of a three-channel continuous recorder (Electroflo Meters Co. Ltd).

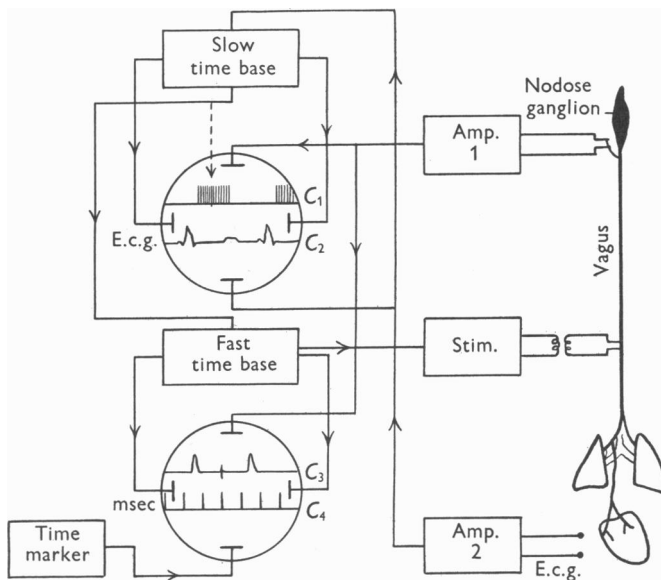


Fig. 1. Block diagram of apparatus used. C_1 , C_2 , C_3 and C_4 refer to the respective beams of the cathode-ray tubes; C_1 and C_2 both display action potentials.

The temperature of the nerve was kept at about 37°C by warmth applied to the area of dissection with a carbon lamp and by maintaining the rectal temperature at about 38°C . The importance of the latter was considerable, as the vagus between the recording and stimulating electrodes was not freed from the surrounding tissues and was thus approximately at body temperature.

The conduction distance of the action potential in the selected strand was measured with a pair of dividers from the cathode of the stimulating electrodes to the proximal recording electrode, and an endeavour was made to allow for any tortuosity in the course of the nerve between these points. These measurements were frequently checked at the end of the experiment by measuring the length of the excised vagus.

To record the intrapleural pressure a wide-bore intrapleural needle was inserted through the third or fourth intercostal space close to the sternum and connected to an air-filled mirror membrane manometer.

Recording circuit

A block diagram of the electrical apparatus used is given in Fig. 1. In general, the detailed circuits of the various electronic units have been based on those described by Dickinson (1950). Basically, two resistance-capacity coupled differential amplifiers were used, the first to amplify the action potentials of the nerve fibres and the second to amplify the e.c.g. lead 1.

A slow time-base, with a sweep duration of 0.2–0.5 sec, was used to display the action potentials and the e.c.g. on the first double-beam cathode-ray tube. This time-base could be triggered by the QRS complex of the e.c.g., displaying the relation of the volleys of action potentials to the cardiac cycle. Any part of the action potential record displayed on the slow sweep could also be displayed on a second cathode-ray tube with a much faster time-base, from which the electrical stimulus to the nerve trunk could also be triggered. The second beam of the second cathode-ray tube was used to mark milliseconds.

These 'sweep expansion' facilities were obtained by driving a Schmitt trigger from the anode of the Miller valve of the first time-base, and using the output of the trigger to start the second time-base. By this means any part of the slow sweep on the first tube could be examined in much greater detail on the second one, thus enabling a simultaneous comparison of the spontaneous and evoked action potentials from nerve stimulation to be made.

The stimulator driven by the fast time-base provided stimuli which could be varied in duration and intensity. The usual duration of the stimuli was about 150 μ sec and the intensity ranged from 10 to 30 V.

To take a photographic record (4:1 reduction) the spots on the first tube were stopped and the repetition frequency of the sweeps on the second tube reduced so that no overlapping of the sweeps occurred on the photographic record. With this procedure, a record of the responses of fibres to natural stimuli could be taken simultaneously with records of the conduction time of impulses set up by electrical stimuli.

Sources of errors

An estimate of the errors arising from measurements of conduction distance was obtained by comparing the lengths of nerves measured *in situ* with those measured after excision. The difference between the two measurements was never greater than 3%—the determinations being made to the nearest 0.5 mm with an average inter-electrode distance of 50 mm.

The errors due to variations in the temperature of the nerve were small since the greater part of the vagus was not separated from the surrounding tissues and was thus kept at body temperature; the temperature of the paraffin bath was usually $37 \pm 1^\circ$ C.

However, in a few cases the temperature varied by more than $\pm 1^\circ$ C and in order to correct for this the Q_{10} of conduction was determined for the cat's vagus. The vagus was separated completely from the surrounding tissues between the stimulating and recording electrodes. Using a fixed stimulus and a fixed conducting distance, the variations in the conduction velocity of the different components of the compound action potentials were determined at different temperatures of the paraffin. Under these conditions, i.e. with some blood supply of the nerve still remaining, the Q_{10} was 1.3. Where necessary, therefore, the conduction velocities have been corrected, using this value, to 37° C.

The conduction time was measured on the records, with a travelling microscope. The reference points were the beginning of the stimulus escape and the beginning of the negative inflexion of the monophasic spike. For each afferent fibre three measurements were made in three different sweeps to allow for variations in the shock response time. In cases where the variation in conduction time for individual measurements exceeded 4%, the final result was estimated by averaging five determinations in different sweeps. No allowance was made for the shock response time as was done by Hunt & Kuffler (1951). The duration of the shock response time is variable (Blair & Erlanger, 1936). The stimulus used, however, was always well above threshold, so that this cause of error in the measured conduction velocity should be small. Further, as the conduction distance was usually above 50 mm any error caused by this factor would have been significant only in the fastest fibres.

In some records in which the single unit preparations were very thin the action potential was triphasic with an initial small short-lived positive deflexion. In such experiments the conduction time was also measured up to the beginning of the negative inflexion of the spike. In other records the action potential consisted of an almost pure positive deflexion and in these the conduction time was measured up to the beginning of the positive deflexion. These changes in the action potential were believed to be due to injury in the nerve fibres just before they reached the recording electrodes since the positive deflexion could be converted into a negative one by shifting the recording electrodes slightly nearer the nerve trunk. Measurements of conduction time under the two conditions cited revealed no significant difference (Fig. 2). However, any error arising from this cause did not affect the conduction velocity determinations in the slowly conducting fibres, while in those with a faster conduction velocity, care was taken to reduce this error to a minimum.

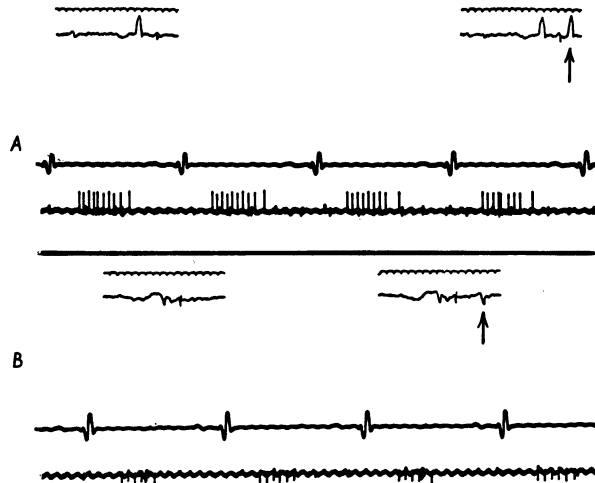


Fig. 2. Conduction velocity of a depressor fibre. Records *A* and *B* are from the same fibre. In both, the upper two traces are records of single sweeps consisting of time marks in milliseconds and conduction time measurement respectively, these read from right to left; below these are respectively, e.c.g., and impulses in a single unit. These read from left to right. In *A* the spikes are negative; in *B*, positive. The conduction velocities as determined in *A* and *B* are respectively 26.0 and 26.5 m/sec. Conduction distance = 58 mm. Arrows indicate position of impulses from receptor activity.

RESULTS

In the determination of the conduction velocity of particular afferent fibres, it was necessary to prove that the fibre in which impulses were set up by receptor activity was the same as the one in which impulses were produced by electrical stimulation of the nerve trunk. In order to establish this point the following criteria were used: (1) The action potential produced by electrical stimulation should be simple and all-or-none in character. (2) It should resemble the action potentials produced by receptor activity (viz. of the single unit) in amplitude and form. (3) It should disappear, or be reduced in size and conduction velocity, if it were so timed that it fell within the absolute or relative refractory period, respectively, of an impulse set up by the activity of a receptor.

Figs. 2 and 3 show single-action potentials produced by artificial stimulation, which were ascertained to be all-or-none by varying the intensity of the stimulus and noting any change in the amplitude of the action potential. It is possible, however, that two afferent fibres with the same conduction velocity and threshold to stimulation could occur. The action potential would be clearly summated and larger than the spikes due to receptor activity. This could be tested by further subdivision of the strand.

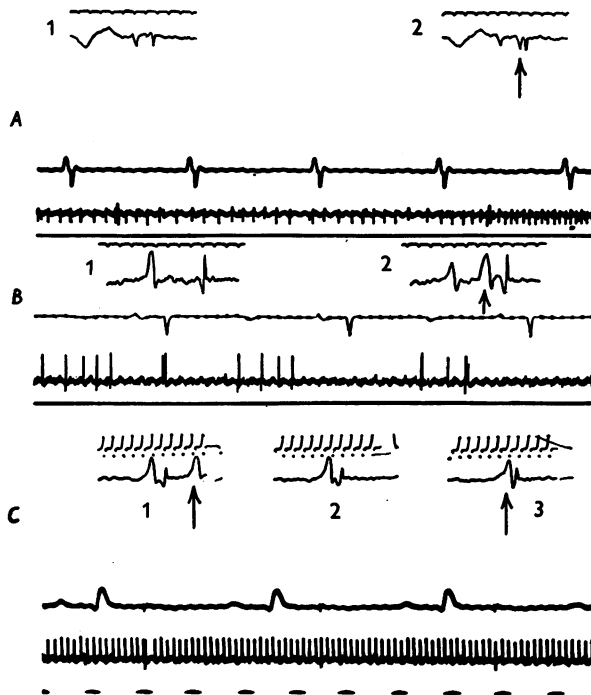


Fig. 3. The effect of setting up evoked impulses during the refractory period after impulses due to receptor activity. *A* and *C*, records from pulmonary stretch receptors, *B* is a record from a right atrial receptor type B. In each record the two upper traces, to be read from right to left, are of time in milliseconds and of action potentials for conduction time determinations. The two lower traces are of the e.c.g. and of action potentials in a single unit, and read from left to right. In *C*, time in $\frac{1}{10}$ sec. In *A*, sweep 2, the evoked impulse falls late in the relative refractory period due to an impulse set up by the receptor, and its conduction rate is reduced from 31 to 25 m/sec. In *B*, sweep 2, the evoked impulse is slowed and reduced in size by the impulse from the receptor. In *C*, sweep 3, the evoked impulse is absent in the third sweep as it has fallen in the absolute refractory period. All arrows indicate position of impulses set up by receptor activity.

Figs. 2 and 3 show the occurrence of natural and evoked action potentials in the same sweep. Measurements of their amplitude and duration show that they are identical. Further, their rate of rise and fall is the same as can be shown by superimposing one on the other.

In most fibres, and particularly those with a cardiac rhythm in which the position of a volley of impulses in the cardiac cycle is approximately constant, it is possible to set up an electrical stimulus one or two milliseconds before the expected time of arrival of a natural impulse at the recording electrode. This natural impulse has left a state of absolute or relative refractoriness in its own fibre under the stimulating electrodes (Adrian, 1921; Gasser & Grundfest, 1936). In successful experiments this led to the complete disappearance of the evoked spike (Fig. 3C) provided the fibre was totally refractory, or to a reduction in its amplitude and conduction velocity if it fell within the relative refractory period (Fig. 3A, B). When such proof was obtainable it was not considered necessary to have a single fibre preparation, provided the compound action potential consisted of not more than two or three discrete and easily identifiable components.

Conduction velocity of pulmonary stretch afferent fibres

The conduction velocities of sixty-seven pulmonary stretch afferent fibres were determined. Two types were distinguished according to the rate of adaptation of their endings to a rapid and sustained inflation of the lungs by positive pressure. Fibres coming from the slowly adapting receptors (Fig. 12) described in detail by Adrian (1933) formed the large majority and, as observed by him, many of these fibres showed discharges on deflation as well as on inflation.

The rapidly adapting receptors of Knowlton & Larrabee (1946) activated the remaining ten stretch fibres. Only units with an adaptation index of 100, namely those dropping out of action by the end of the first second of inflation, have been classified as rapidly adapting receptors. All the rapidly adapting receptors encountered here had a high threshold to inflation.

The frequency distribution of their conduction velocity and the relevant data pertaining to them are given in Fig. 4 and Table 1 respectively. The mean conduction velocity of these fibres was 25 m/sec, which is less than the mean for the fibres from slowly adapting stretch receptors. The difference is, however, not highly significant as the figures of Knowlton & Larrabee (1946) would suggest, but it is possible that examination of a large number of fibres from rapidly adapting receptors might yield a significant difference.

A number of fibres from slowly and rapidly adapting pulmonary stretch receptors also showed a superimposed cardiac rhythm, as was first described by Adrian (1933). Presumably, as suggested by him, these receptors are situated near the root of the lungs and are deformed by the pulsation of the heart and great vessels. The mean conduction velocity of seven of these fibres is 26 m/sec, the difference from the mean of the stretch afferent fibres not being significant.

TABLE 1. Conduction velocities of vagal afferent fibres

Type of afferent fibre	No. of fibres	Range (m/sec)	Mean (m/sec)	S.D.
Pulmonary stretch (slowly adapting)	57	14-59	36	10
Pulmonary stretch (rapidly adapting)	10	16-37	25	7
Systemic arterial pressure receptors	16	12-53	33	11
Right atrial type A	8	13-27	20	5
Right atrial type B	11	8-23	13	5
Left atrial type B	4	15-26	20	4
Chemoreceptor	3	7-12	10	2
Afferent fibres stimulated by phenyl diguanide	4	3-9	6	2

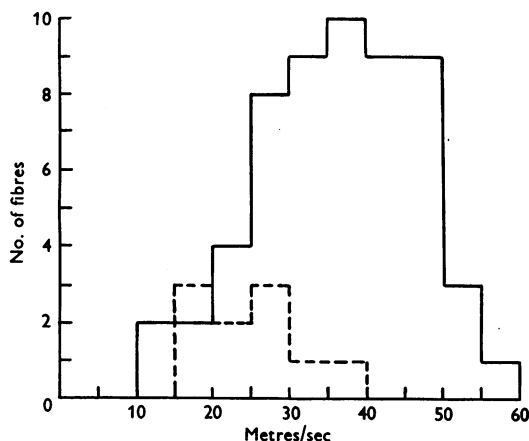


Fig. 4. Frequency distribution of the conduction velocities of pulmonary stretch afferent fibres. Solid line, slowly adapting receptors; interrupted line, rapidly adapting receptors.

The conduction velocity of type B right and left atrial receptors

Altogether, the conduction velocity of eleven fibres from the right atrium and four fibres from the left atrium have been determined. These afferent fibres have a number of characteristic features and their identification is usually fairly simple (Fig. 5). A detailed description of their behaviour and method of identification appears elsewhere (Paintal, 1953*b*).

Of those arising from the right side, the first nine were regarded as coming from pulmonary vascular receptors (Whitteridge, 1948), before it was known that such fibres originate in the right atrium of the heart. These fibres were therefore identified by the criteria described by Whitteridge (1948) and by Pearce & Whitteridge (1951). The remaining two, in addition to being identified by the usual criteria, were shown to be excited by receptors in the right atrium in the course of experiments with the chest open (Paintal, 1953*b*). All four fibres from the left atrium were traced to their origin after opening the chest. The conduction velocity of one of these was measured by direct

measurement of the cathode-ray tube trace as no photographic records could be made of it.

Although the conduction velocities of fibres from both atria cover nearly the same range (Fig. 6), their means are different (Table 1) but the difference is not highly significant. The entire range of conduction velocity of the type B atrial afferent fibres is about a third of that of the pulmonary stretch afferent fibres.

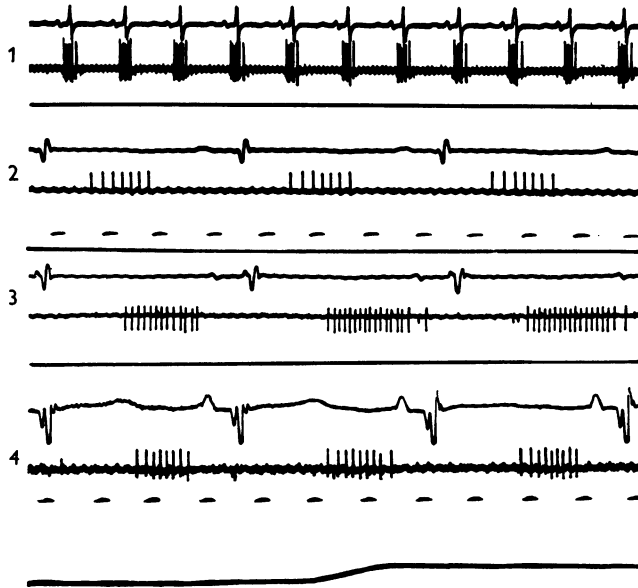


Fig. 5. Records from single receptors: (1) right atrial receptor type A; (2) systemic arterial pressure receptor; (3) right atrial receptor type B; (4) left atrial receptor type B. In addition in (4) there is time in $\frac{1}{10}$ sec and the intrapleural pressure record, inspiration downward. All records show the e.c.g. and the action potential record.

Conduction velocities of nerve fibres from systemic arterial pressure receptors

These afferent fibres are easily distinguished from others with a cardiac rhythm when their activity is heard on the loudspeaker. They are characterized by the early systolic volley (Fig. 5) which begins between 36 and 38 msec after the Q wave of the e.c.g. and reaches its peak frequency between 53 and 106 msec. This is very much earlier than the volley from the atrial type B receptors and provides one means of distinguishing the two. Another difference is that the activity of their receptors is much less influenced by respiratory movements. Further, maximum activity occurs during expiration usually at its beginning, whereas maximum activity in the right atrial receptors occurs at the height of inspiration.

Inflation of the lungs with positive pressure reduces the activity of the systemic arterial receptors, and this does not return to its original height for several heart beats after inflation has ceased. This is one of the most consistent features of these receptors, distinguishing them from type B right atrial receptors in which maximum activity always returns within two or three heart beats. On the other hand, this behaviour is similar to that of the left atrial type B receptor. The frequency distribution of their conduction

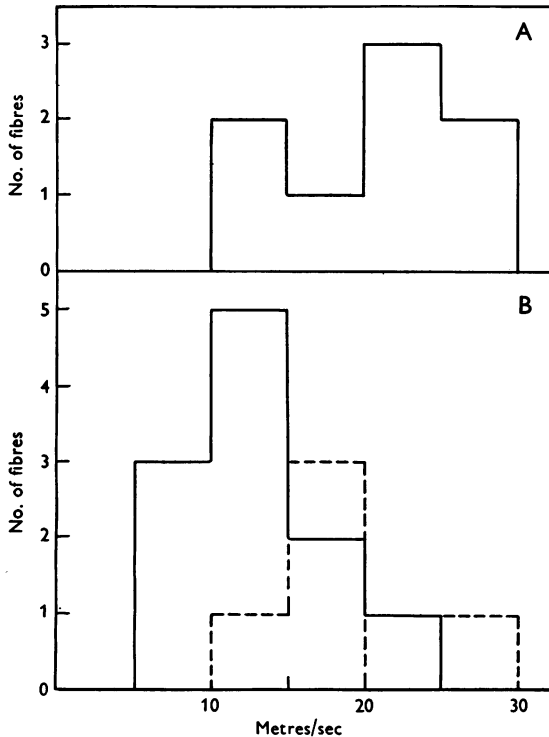


Fig. 6. Frequency distribution of the conduction velocities of atrial afferent fibres. *A*, right atrial type A; *B*, atrial type B. In *B*, the solid and interrupted lines represent right and left atrial type B fibres respectively.

velocities is shown in Fig. 7. From Fig. 7 it is clear that the range 12–53 m/sec is as wide as that of the pulmonary stretch fibres. Their mean of 32 m/sec (Table 1) is also near to the corresponding value for the pulmonary stretch afferent fibres which is 36 m/sec and both are much higher than the mean for type B right atrial afferent fibres which is about 13 m/sec. From the data given, it can be calculated that the difference in means for the arterial pressure receptors and right atrial type B fibres is statistically highly significant.

There is no significant difference between the conduction velocities of the fibres from arterial pressure receptors which are found in the depressor nerve

and those which occur in the vagus, the mean of both groups being 33 m/sec. It therefore appears that the assumption of Bishop, Heinbecker & O'Leary (1934) that fibres from the arterial pressure receptors in the vagus are larger than those found in the depressor nerve itself, cannot be confirmed.

When the activity in these systemic arterial pressure afferent fibres is plotted as frequency of discharge against time, the constancy of behaviour of different fibres is well illustrated. The similar shapes of the histogram and the narrow range of time (54 msec) within which the peak frequency occurs is remarkable. This is in contrast to the curves for the activity in type B atrial afferent fibres which show a wide variation in shape and temporal configuration.

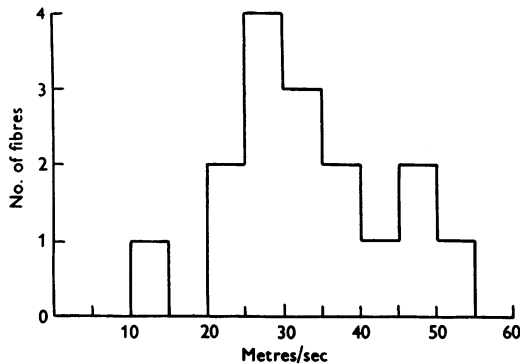


Fig. 7. Frequency distribution of the conduction velocities of fibres from systemic arterial pressure receptors.

Conduction velocity of type A right atrial afferent fibres

It has been possible to determine the conduction velocities of only eight of these. This was partly because, in the absence of a venous pressure record in the earlier experiments, it was only possible to identify five of them with certainty. Some other fibres examined may also have been right atrial afferent fibres with an atypical discharge.

The criteria used in the identification of type A atrial afferent fibres were that they should show a characteristic presystolic burst (Fig. 5) with or without *c* and *v* volleys, presumably in time with the *a*, *c* and *v* waves respectively of the venous pressure curve, and that they should increase in activity during inspiration and during suction of air out of the lungs, i.e. conditions leading to increased venous return. Further, they should show a reduction in activity on positive-pressure inflation of the lungs. In the case of three fibres the right atrial pressure was recorded simultaneously, thus giving additional confirmation of their identity.

The observation of Whitteridge (1948) that *v* volleys show greater changes with respiration than do the *a* volleys is confirmed.

The frequency of discharge in the *a* burst can be very high; for the fibre shown in Fig. 5 the peak frequency reached was 405 impulses/sec.

The frequency distribution of the conduction velocities is plotted in Fig. 6. The range of conduction velocity (Table 1) overlaps that of the left atrial type B fibres and their means are identical as well. It also overlaps that of the fibres from type B receptors of the right atrium, although the means for the two groups are different; the difference, however, is not highly significant statistically.



Fig. 8. A chemoreceptor fibre (large diphasic action potential). *A*, before; *B*, during; and *C*, after administration of nitrogen for 2 min by a respiratory pump. From above downwards, e.c.g. and impulses in nerve fibres. The e.c.g. shows changes in *B*.

Conduction velocity of chemoreceptor afferent fibres

The main reason for the difficulty in the isolation of single chemoreceptor units is that they are normally inactive, and it has therefore been possible to determine the conduction velocity of only three chemoreceptor afferent fibres. This was so, in spite of the fact that the majority of the afferent fibres from the aortic body are contained in the right vagus and aortic nerves (Neil, Redwood & Schweitzer, 1948).

In the isolation of single units the fibres were dissected as already described. If, on recording, there were occasional irregular impulses, their possible origin from chemoreceptors was tested by the administration of pure nitrogen for a brief period through a respiratory pump (Starling 'Ideal'). If a continuous discharge appeared during nitrogen inhalation and ceased a few minutes after it was discontinued, this was regarded as evidence that the nerve strand contained a chemoreceptor afferent fibre (Fig. 8). This test was based on the observation of Landgren & Neil (1951) that a continuous discharge is produced in the aortic chemoreceptor afferent fibres by the administration of a high concentration of N₂ (96% with 4% O₂). The administration of N₂ was found to cause marked changes in the e.c.g. The mean conduction velocity of three chemoreceptor afferent fibres, two from the depressor nerve and one from the vagus, is given in Table 1.

The range of the conduction velocities appears to be rather narrow but this might be due to the small number of fibres isolated. It will be noted that the mean conduction velocity of the chemoreceptor fibres is not significantly different from the mean conduction velocity of the type B right atrial afferent fibres.

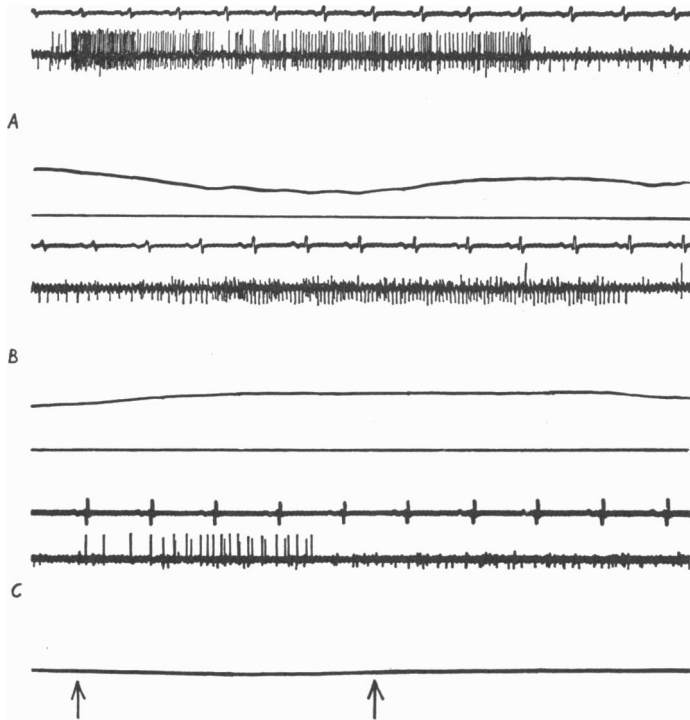


Fig. 9. Fibres firing on suction of air from the trachea. From above downwards in *A*, *B* and *C* are e.c.g., impulses in a fibre and intrapleural pressure, inspiration downwards. *A* and *B* are continuous records of deflation and inflation respectively. The fibre with large spikes is active only during deflation and the small one, a pulmonary stretch fibre, fires on inflation. In *C*, during inspiration (between arrows) two pulmonary stretch fibres are active; during expiration only the fibre with small spikes is active.

Conduction velocity of afferent fibres stimulated by deflation

Although a search for these fibres was started only in the latter part of the present investigation, five fibres were found to become active during suction of air from the trachea, and not during inflation (Fig. 9 *A*, *B*). It was possible, however, to determine the conduction velocity of only two of these.

The behaviour of the five fibres is presented in Table 2.

Fig. 9 *C* shows the record from a multi-fibre preparation during a normal respiratory cycle. The pulmonary stretch fibre with the large spike becomes active during inspiration, while the fibre with the small spikes shows marked

activity during expiration. It appears, therefore, that some receptors do become more active during normal expiration.

TABLE 2. Afferent fibres stimulated by deflation

Serial no.	Response to inflation	Response to deflation	Activity during expiration	Conduction velocity (m/sec)
1	-	+	+	32
2	-	+	-	.
3	-	?	+	.
4	-	+	-	2.8
5	-	+	+	.

Afferents stimulated by phenyl diguanide

These afferent fibres are normally inactive, and respond by a continuous discharge to an intravenous injection of 100 μ g phenyl diguanide (Dawes & Mott, 1950). They are discussed in detail elsewhere (Paintal, 1953c). The data pertaining to their conduction velocities appear in Table 1.

These fibres could be those responsible for the inhibition of respiration produced by the drug, since this reflex effect is abolished by cooling the vagi to about 3° C (Dawes, Mott & Widdicombe, 1951), a temperature which is probably effective in blocking fibres with such low conduction velocities.

DISCUSSION

On the assumption that thermal blocking of conduction in vagal nerve fibres varies according to the conduction velocity (Pearce, 1951), the results presented here are in general agreement with those of Torrance & Whitteridge (1947).

The degree of overlap of the conduction velocities of different vagal afferent fibres is considerable, the pulmonary stretch fibres cover the entire range of conduction velocities for the depressor and venous afferent fibres and a considerable part of the range of the type B atrial afferent fibres as well. This wide range of conduction velocities has been seen in individual cats (Fig. 10), for the same type of afferent fibres.

From these facts it is unlikely that blocking conduction by pressure or cold could be selective for any one group of afferent fibres; the reflex changes brought about by any cold block would be the result of action on more than one type of fibre. This should be borne in mind in the interpretation of reflex changes produced by applying a vagal block and either stimulating the vagal trunk distal to it (Head, 1889; Hammouda & Wilson, 1935) or stimulating the receptors themselves by various drugs (Dawes *et al.* 1951).

The conduction velocities of fibres from stretch and depressor receptors do not correspond to their fibre diameters as given by Heinbecker & O'Leary (1933), namely 10–4 μ for stretch and 6–3 μ for depressor fibres. Further, no

confirmatory evidence could be obtained for the suggestion of Bishop *et al.* (1934) regarding the existence in the vagus of depressor fibres larger than those found in the depressor nerve itself. In a similar connexion the conclusion of Nonidez (1941) that, in general, the fibres ending as receptors in the large veins are thicker than those which end as pressoreceptors in the arch of the aorta, is also not supported by this work.

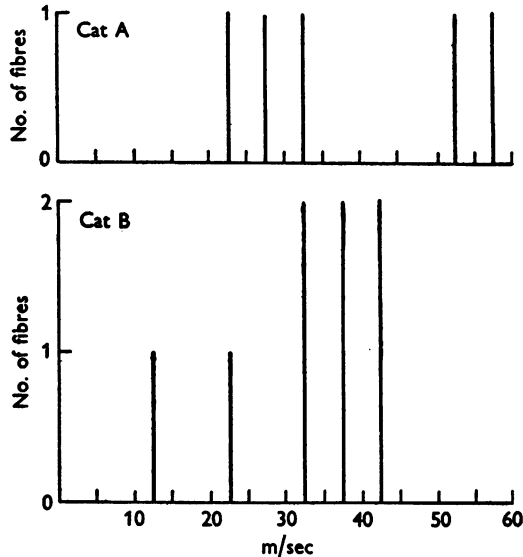


Fig. 10. Distribution of the conduction velocities in pulmonary stretch fibres in two cats. Note the wide range in individual cats.

In respect of their conduction velocities there does not appear to be a significant difference between the fibres from slowly adapting and rapidly adapting pulmonary stretch receptor fibres (Knowlton & Larrabee, 1946). Recently, Widdicombe (1952) has shown that rapidly adapting stretch receptors are to be found in the trachea and larger bronchi. It has been suggested that they are concerned with the cough reflex.

The observation of Adrian (1933) that forced deflation calls a new set of endings into play finds confirmation in the present investigation. However, contrary to his conclusion, evidence has been presented here to show that some receptors do discharge impulses during normal expiration (Fig. 9C) and that both rapidly and slowly adapting types respond to forced deflation. The conduction velocity of one fibre (2.8 m/sec) suggests that some of these afferent fibres possess the characteristics required to account for some of the results of Hammouda & Wilson (1935) in experiments involving cooling of the vagi. It is, however, difficult to account for the acceleration of respiration on inflation

of the lungs according to their views, on the basis of the deflation receptors described, as none of these responded to an inflation. This needs to be investigated further.

An analysis of the compound action potentials of the vagus on the basis of the conduction velocities of the afferent fibres and histological evidence provided by previous investigators would now seem possible.

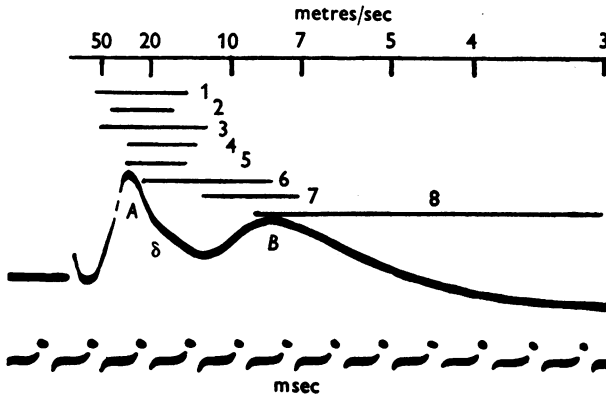


Fig. 11. Compound action potential of the vagus with the range of conduction velocities of various afferent fibres superimposed on it. (1) slowly adapting pulmonary stretch fibres, (2) rapidly adapting pulmonary stretch fibres, (3) depressor fibres, (4) type A right atrial fibres, (5) type B left atrial fibres, (6) type B right atrial fibres, (7) chemoreceptor fibres, (8) fibres firing on injection of 100 μ g phenyl diguanide intravenously. Conduction distance, 33 mm. Note the presence of different types of afferent fibres in the δ and B elevations.

Heinbecker (1930) concluded from a comparative study of the vagus and sympathetic nerves of the cat and turtle that the A and B elevations contained visceral afferent fibres, while the B₂ (with a conduction velocity of about 10 m/sec) and C elevations were composed of autonomic motor fibres. The B₁ and B₂ elevations of Heinbecker correspond to the A γ and B elevation, respectively of Erlanger & Gasser (1937). Recently, Middleton, Middleton & Grundfest (1950) inferred that part of the γ elevation with a conduction velocity of 18 m/sec and the B elevation with a maximum conduction velocity of 14.3 m/sec were composed of an influx of sympathetic preganglionic fibres.

An examination of Fig. 11 shows that the conclusions of these two groups of authors are not entirely correct. That both the A and B elevations contain visceral afferent fibres is borne out by the variety and large numbers of afferent fibres that have been isolated and found to have corresponding velocities. On the other hand, although it is highly probable that part of the B elevation is composed of preganglionic efferent fibres, it would appear that the contribution by visceral afferent fibres is considerable. There are a number of different types of afferent fibre in this group, namely pulmonary stretch, depressor,

right atrial type A, right and left atrial type B, chemoreceptor, fibres firing on deflation, and those with unknown function stimulated by phenyl diguanide. In addition, one-fifth of all afferent fibres encountered have a conduction velocity below 15 m/sec, which, according to the figures given by Middleton *et al.* would all fall in the *B* elevation. It would, therefore, appear unnecessary to postulate an external influx from sympathetic preganglionic fibres to account for the practically unchanged *B* elevation after a supranodosal section.

Histological evidence of Dickinson (unpublished observations) and Daly & Evans (1953) support these conclusions; they found that the greatest number of afferent fibres are in the 2–4 μ groups. On these grounds, as suggested by Whitteridge (1952), it would be more appropriate to designate part at least of the *B* elevation of the vagus as δ_2 .

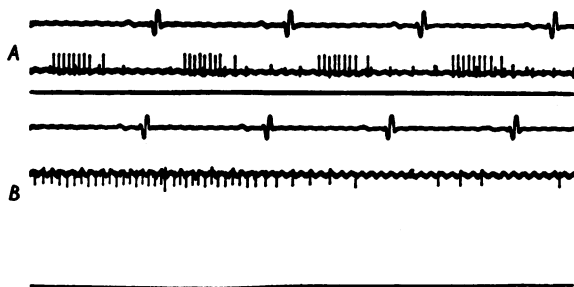


Fig. 12. Spike size and conduction velocity. From above downwards, e.g., record of nerve impulses; and in *B* intrapleural pressure, inspiration downwards. In *A*, a depressor fibre (large spike) and a stretch fibre with (small spike) a cardiac rhythm can be seen; *B*, record of the same stretch fibre after further subdivision of the nerve strand. The conduction velocities of the depressor and stretch fibres were 26 and 39 m/sec respectively.

Wyss & Rivkine (1950) observed that on stimulating the central end of the cervical vagus nerve a strong inspiratory reaction with marked acceleration of breathing was associated with activity of slow fibres of the B_1 type (presumably δ). From Fig. 11 it is obvious that the δ elevation is a composite one, pulmonary stretch, depressor, right atrial type A, and right and left atrial type B, afferent impulses being represented in it. The appearance of this elevation implies, therefore, that some or all of these afferent fibres may have been stimulated, the resulting reflex changes being due to the composite effect of several functionally different types of afferent fibre. Caution is therefore necessary in the interpretation of results obtained by this approach.

It is generally recognized that the amplitude of the action potential varies as the size of the fibre (Gasser & Grundfest, 1939), and therefore as its conduction velocity. Exceptions to this rule, however, have been observed on many occasions. An example is shown in an experiment (Fig. 12) in which it was found by later subdivision that the pulmonary stretch fibre with the

smaller spike had a conduction velocity of 39 m/sec and the depressor fibre with a large spike, a conduction velocity of 26 m/sec. The spike size and conduction velocity relation need not therefore hold true when action potentials from fine dissected strands are being recorded. This is mentioned, as conclusions about the size of fibres have often been drawn purely from measurements of the relative sizes of the spike (Jarisch & Zotterman, 1948).

SUMMARY

1. A method of determining the conduction velocities in single respiratory and cardiovascular afferent fibres is described.

2. The conduction velocities of 116 respiratory and cardiovascular vagal afferent fibres have been determined and it has been shown that:

(a) The range of conduction velocities of the thoracic vagal afferent fibres extends from about 2 to 61 m/sec.

(b) The mean conduction velocity of fibres from slowly adapting pulmonary stretch receptors is 36 m/sec; from rapidly adapting stretch receptors, 25 m/sec; from depressor receptors, 33 m/sec; from right atrial type A receptors, 20 m/sec; from left atrial type B receptors, 20 m/sec; from right atrial type B receptors, 13 m/sec; from chemoreceptors, 10 m/sec; and from receptors firing on injection of phenyl diguanide, 6 m/sec.

(c) There is a considerable overlap of the conduction velocities of the different afferent fibres.

(d) There is a group of slowly conducting fibres with conduction velocities of 2–10 m/sec consisting mainly of unidentified fibres, in addition to deflation, chemoreceptor afferent fibres, and of fibres stimulated by an intravenous injection of 100 μ g phenyl diguanide.

3. The compound action potential of the vagus has been analysed, and it has been shown that a number of afferent fibres are represented in each of its components. The contributions by different afferent fibres to the *B* elevation is considerable.

4. The value of studying reflex changes by stimulating the vagus or using differential nerve blocks has been discussed in the light of the present results.

5. The relation between spike size and conduction velocity does not always hold, when action potentials are recorded in very fine nerve strands.

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