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PROPERTIES OF VESTIBULAR NEURONES PROJECTING TO NECK SEGMENTS OF THE CAT SPINAL CORD*

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SUMMARY

1. Vestibular neurones projecting to the upper cervical grey matter (vestibulocollic neurones) were identified by localized microstimulation in the C3 segment of the cat spinal cord.

2. The neurones were found in the lateral (Deiters'), medial and descending nuclei bilaterally and projected to the spinal cord in the lateral and medial vestibulospinal tracts (LVST and MVST). Ipsilateral axons of Deiters' neurones were mostly in the LVST, axons of medial and descending neurones in the MVST; a few Deiters' neurones had axons in the MVST; some descending neurones had axons in the LVST. Most axons of contralateral neurones were in the MVST.

3. The axons of 62 % of ipsilateral vestibulocollic Deiters' neurones not only gave off a collateral to C3, but also extended as far as the cervical enlargement ('branching'); some of these neurones projected as far as the upper thoracic cord, almost none to the lumbar cord. Ipsilateral descending nucleus neurones branch in the same fashion, but there is no branching in the relatively small medial nucleus population.

4. A large majority of vestibulocollic neurones receive monosynaptic excitation from the ipsilateral labyrinth and a number are inhibited by stimulation of the contralateral labyrinth (commissural inhibition). It is possible that commissural inhibition acts on a broad population of vestibular neurones involved in the control of eye, head and trunk movement.

5. Vestibulocollic neurones do not make up a homogeneous population acting only on the neck. Instead it is likely that subpopulations, for example branching and non-branching neurones, have different functions.

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INTRODUCTION

Properties of neurones giving rise to fibres travelling within the lateral and medial vestibulospinal tracts (LVST and MVST), and connexions of these fibres at various levels of the spinal cord, have been studied extensively. Both tracts contain neurones that project to different levels of the spinal cord (Pompeiano & Brodal, 1957; Nyberg-Hansen, 1964; Nyberg-Hansen & Mascitti, 1964; Petras, 1967) and that vary in the nature of the inputs which they receive (for instance, Wilson, Kato, Peterson & Wylie, 1967*a*; Akaike, Fanardjian, Ito, Kumada & Nakajima, 1973*a*). In addition, MVST neurones may be crossed or uncrossed (Nyberg-Hansen, 1964; Akaike *et al.* 1973*a*; Wilson & Maeda, 1974), and excitatory or inhibitory (Wilson & Yoshida, 1969*b*; Akaike, Fanardjian, Ito & Ohno, 1973*b*; Wilson & Maeda, 1974). Many questions concerning the organization of VST neurones remain to be studied; investigation of specific properties of sub-populations of VST neurones may provide insight into the functions subserved by these populations.

We have examined a number of properties of VST neurones projecting to neck segments of the spinal cord (vestibulocollic neurones). These neurones were chosen for study because connexions between VST axons and motoneurones are very direct in neck segments (Wilson & Yoshida, 1969*a*, *b*; Akaike *et al.* 1973*b*) and vestibulocollic neurones are involved in specific reflexes of the neck (Suzuki & Cohen, 1964; Wilson & Maeda, 1974). These neurones are therefore most likely to constitute a specialized population with relatively unique properties. Properties examined were input from the ipsilateral as well as contralateral labyrinth, tracts in which fibres descend, location of neurones within the vestibular nuclei, and possible axon branching to more caudal levels of the spinal cord. The last is perhaps the most interesting of the properties: if vestibulocollic neurones do constitute a specialized population, their axonal distribution may be restricted exclusively to neck segments of the cord, unlike other VST neurones whose axons may branch to both cervical enlargement and lumbar cord (Abzug, Maeda, Peterson & Wilson, 1974).

Our experiments demonstrate that a substantial proportion of vestibulocollic neurones branch, but that the pattern of branching differs from that of previously studied neurones projecting to the cervical enlargement (Abzug *et al.* 1974). The neurones also receive extensive excitatory input from the ipsilateral labyrinth, and many are inhibited by stimulation of the contralateral labyrinth.

Some of these results have been presented in a preliminary communication (Wilson, Uchino, Susswein & Rapoport, 1976).

METHODS

Twenty-seven adult cats were used. In eighteen animals, preliminary surgery was performed under a halothane-nitrous oxide mixture, which was replaced by 50 mg/kg α-chloralose at the start of recording. Additional doses of chloralose (15–20 mg/kg) were given if necessary to maintain the anaesthesia. Six animals were initially anaesthetized by a 35 mg/kg I.P. injection of sodium pentobarbitone (Nembutal, Abbott Lab.) and three cats were prepared under halothane-nitrous oxide, decerebrated at the precollicular level, and then studied without anaesthesia. Results obtained using the two kinds of anaesthesia and in unanaesthetized animals were similar. All animals were paralysed by I.V. administration of gallamine triethiodide (Flaxedil; Davis and Geck) and respired artificially. Mean arterial pressure was monitored routinely from the femoral artery. When necessary, metaraminol bitartrate (Aramine; Merck, Sharp and Dohme) was administered by I.V. infusion to maintain an arterial pressure of 100 mmHg or more. Rectal temperature was maintained between 36 and 38° C.

Surgical procedures

The vestibular nerve on both sides was stimulated bipolarly in all experiments. An electrode of silver wire (0·5 mm in diameter), insulated except for the spherical tip, was placed upon the vestibular nerve, while an identical indifferent electrode was placed upon nearby bone. Stimuli were 100 μsec rectangular pulses. The C3, and sometimes C2, dorsal rami (DR) were dissected and placed on bipolar platinum hook electrodes. A dorsal laminectomy was performed from C1 to C8 to permit placement of stimulating electrodes at various cervical levels; additional smaller laminectomies were made at upper thoracic and upper lumbar levels. An occipital craniotomy was performed and the medial portion of the cerebellum was removed by suction, to facilitate visualization of the vestibular nuclei.

Stimulating procedures

Stimulation of branches in C3 grey matter

Glass micropipettes were used, which were filled with 2M-NaCl saturated with Fast Green FCF and had resistances of 1–2 MΩ. Micropipettes were inserted into, or just dorsal to, the mononeurone pool which was activated antidromically by stimulation of the C3DR (Fig. 1). Many stimulating positions were marked by ejection of Fast Green dye (Thomas & Wilson, 1965).

A constant-current stimulator was used to apply cathodal pulses of 150–200 μsec duration, at a rate of 5/sec, through the micropipette with respect to a distant indifferent electrode. Stimulating current was monitored as the voltage drop across a 1kΩ resistor which was placed in the return path to ground. Whenever a vestibular neurone was fired by the C3 stimulus, the threshold was determined; the electrode was usually moved dorsally and ventrally in its track to obtain the lowest threshold. Criteria for determining whether neurones were responding antidromically were as described by Abzug *et al.* (1973). Almost all neurones had a sharp threshold and constant latency to above-threshold stimuli (see section 1 of Results). Whenever tested, firing followed stimulus frequencies of 250–500/sec and there was lack of temporal facilitation with activation by a train of stimuli. If a neurone fired spontaneously in response to stimulation of the vestibular nerve, then a check was made of the interval over which the occurrence of a synaptically evoked action potential blocked the appearance of a subsequent C3-evoked impulse, to see whether the cause of the blockage could be ascribed to collision of orthodromic and antidromic

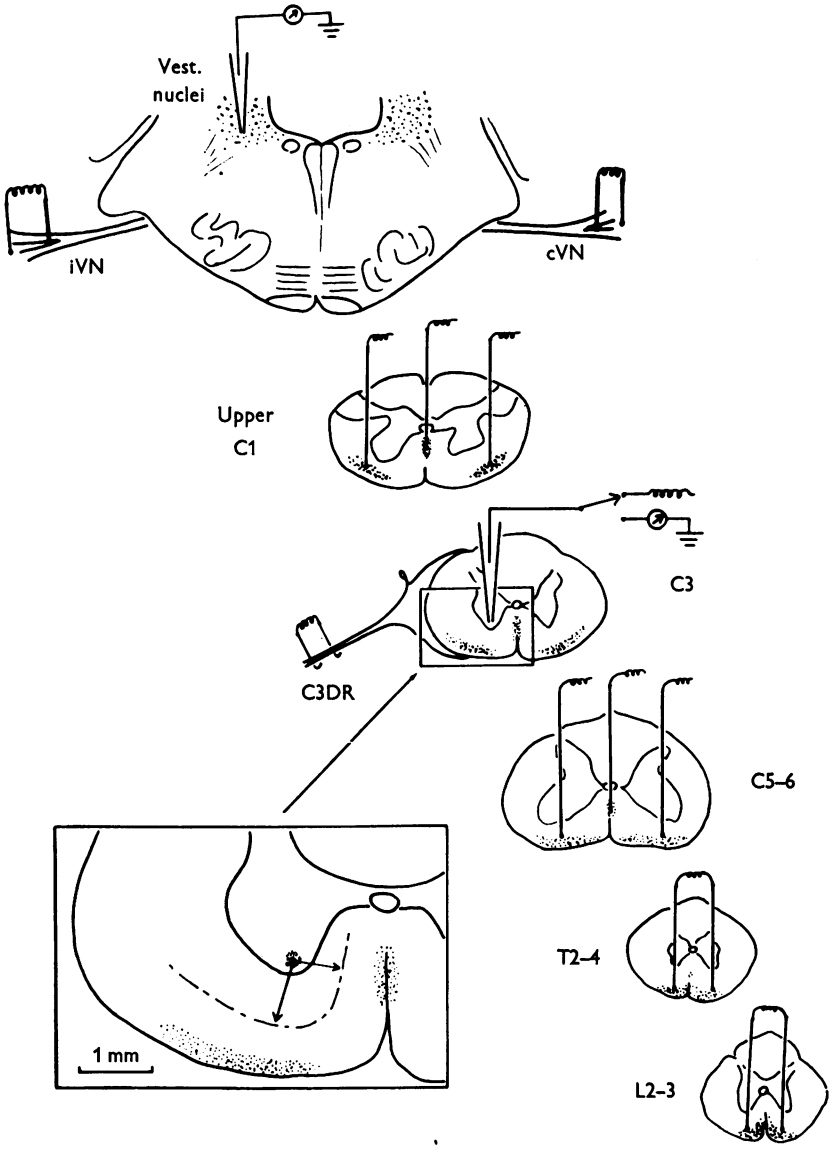

Fig. 1. Diagram of experimental arrangement. Topmost drawing shows recording micro-electrode in vestibular nuclei and stimulating electrodes on the ipsilateral and contralateral vestibular nerves (iVN and cVN). Other drawings show stimulating arrays at various spinal levels from C1 to L2–3. In all cord sections the approximate locations of the two LVSTs and MVSTs, based on Nyberg-Hansen (1964), Nyberg-Hansen & Mascitti (1964) and Petras (1967), are shown by stippling. The drawing of C3 also shows the stimulating electrode on the C3 dorsal rami (DR). The enlarged drawing of the area marked off by the rectangle in C3 shows the location of a Fast Green dye mark made at a stimulating location, as well as the measurements made for conservative estimation of the distance from the stimulating point to the two VSTs (arrows). Further details in text.

impulses (Darian-Smith, Phillips & Ryan, 1963). We also tested collision block between responses to C3 stimulation and responses to stimulation at other spinal levels that satisfied the criteria for antidromic unit activity. The maximal blocking interval between two antidromic responses is conduction time between the stimulating electrodes, plus the refractory period at the test stimulus point (Abzug *et al.* 1973).

Stimulation of LVST and MVST axons in C1

Monopolar stimulating electrodes were electrolytically sharpened tungsten needles, insulated to 0·3 mm of their tips. These electrodes were inserted into the caudal medulla or the C1 segment, 3–4 mm caudal to the obex, to stimulate left and right LVST and MVST fibres (Fig. 1). LVST electrodes were placed approximately 2·5 mm from mid line, while MVST electrodes were placed near the mid line. Placement of C1 electrodes was performed while recording antidromic field potentials in Deiters' nucleus and in the medial vestibular nucleus or MLF. Electrodes were positioned at points which elicited low threshold antidromic fields. It was not always possible to see individual neurone responses in, or later than, the large antidromic fields. When it was, their antidromic nature was established by their short, fixed latency.

Lower cervical, thoracic and lumbar stimulation

In most experiments, we stimulated the axons of vestibular neurones in the lower cervical, upper thoracic and upper lumbar regions of the spinal cord with tungsten electrode arrays to test whether vestibular neurones activated antidromically from the C3 grey matter also projected more caudally (Fig. 1). Low threshold positions for these electrodes were determined by recording antidromic field potentials in the vestibular nuclei. In many experiments cervical enlargement stimulation was with three monopolar electrodes. This procedure was later replaced by bipolar stimulation with two electrodes, one placed near the middle of the LVST on each side. Thoracic and lumber stimulation was always bipolar; the electrodes were placed just lateral to the LVST.

Recording and other data analysis

Glass micropipettes containing Fast Green FCF and having resistances of 1–3 MΩ were inserted into the vestibular nuclei for extracellular recording. When we studied the effect of contralateral vestibular nerve stimulation on single unit activity, data were processed on-line using a PDP-11 computer programmed to accept data for spike post-stimulus time (PST) histograms. When a PST histogram was plotted, the display included the integral of the response. To mark the location of many units Fast Green dye was ejected from the micropipette at the completion of recording.

At the end of each experiment the position of the tip of each metal electrode used for stimulation in the spinal cord was marked by lesions made by passing 20 μA of cathodal current through each electrode for 15 sec. The animal was sacrificed and the brainstem and the spinal cord were removed and fixed in 10% formol saline. 100 μm frozen sections were cut in the plane of the electrode tracks. The brain stem sections were stained by the technique of Klüver & Barerra, and histological reconstruction was used to determine the locations where vestibular neurones had been recorded. The locations of lesions in the spinal cord were determined in thionin-stained sections.

RESULTS

(1) *Localized antidromic activation of branches of vestibulospinal axons*

Axonal branches of 186 vestibular neurones were stimulated by microstimulation with a glass electrode in or near the extensor motor nuclei in C3. Extracellular records of responses of antidromically driven vestibular neurones were typically negative (for example, Fig. 3) and ranged in amplitude from 100 μV to more than 1 mV.

It was necessary to be certain that the C3 stimulus was activating a local axonal branch instead of spreading to the vestibulospinal tracts in the white matter. The distances from the stimulating point to the LVST and MVST, which are located at the periphery of the ventral white matter (Nyberg-Hansen, 1964; Nyberg-Hansen & Mascitti, 1964; Petras, 1967), were estimated conservatively by measuring to the mid-point between grey matter and edge of white matter, as shown in the enlarged drawing of C3 in Fig. 1. The shorter of the two distances was then divided by the threshold of the axon to obtain the D/T ratio (distance to threshold, Abzug *et al.* 1974). For instance, a ratio of 30:1 would mean that a 10 μA stimulus would have to spread 300 μm for excitation of axons in the tract to be a possibility. We have previously shown, by vertical movement of stimulating electrodes and by direct measurement of spread to the LVST, that in the range of stimulus strengths used in the present experiments spread is only 10–20:1 (Abzug *et al.* 1974). While spread appears to be relatively greater with very small currents (Jankowska & Roberts, 1972), the furthest estimate for spread to large axons is about 27:1 for pulses as strong as 30 μA (Fig. 11 in Gustafson & Jankowska, 1976). Our thresholds ranged from 1 to 25 μA (n = 183, mean 11·3 $\pm$ 6 S.D.). With stimuli of only a few μA, delivered in the grey matter, spread is not a factor. For the whole population of axons D/T ratios ranged from 20 to > 100:1, almost all were > 30:1 and more than half were > 50:1. Some VST fibres are located outside the areas predicted from anatomical observations (T. Hongo, personal communication) and therefore may be nearer the grey matter than we estimate. Nevertheless, considering our thresholds and the worst possible estimates of stimulus spread, it is certain that spread to the tracts is not a significant factor in our experiments.

The latency of antidromic responses of vestibular neurones occasionally decreased in duration by as much as a few tenths of a msec as the stimulus strength was increased from threshold; such large changes are probably due to stimulation of different branches (cf. Jankowska & Roberts, 1972). The minimal latency, usually measured at 1·5T, ranged from 0·7 to > 2 msec (n = 186; mean 1·2 $\pm$ 0·3 S.D.), with no difference between neurones located in the ipsi- and contralateral nuclei. Taking synaptic delay into

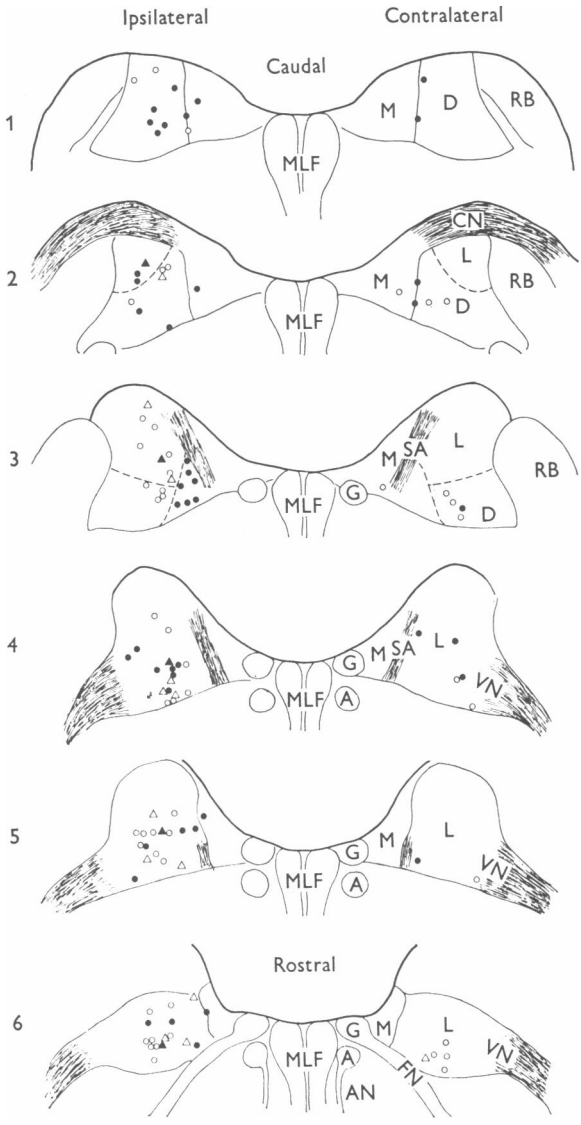

Fig. 2. Locations of vestibulocollic neurones in the vestibular nuclei. Six transverse sections through the vestibular nuclei are shown with locations of neurones recorded in many experiments. Circles and triangles are neurones receiving and lacking mono- or disynaptic input from the ipsilateral labyrinth. Open symbols are neurones with axons projecting as far as C5–7, filled symbols are neurones activated antidromically only from C3. Abbreviations: A, abducens nucleus; AN abducens nerve; CN, cochlear nerve; D, descending nucleus; FN, facial nerve; G, genu of facial nerve; L, lateral (Dieters') nucleus; M, medial nucleus; MLF, medial longitudinal fasciculus; RB, restiform body; SA, stria acoustica; VN, vestibular nerve.

consideration, the early latencies are compatible with the latencies of monosynaptic potentials evoked in C3 motoneurones by stimulation of the vestibular nuclei: 1·0–1·5 msec for e.p.s.p.s evoked by Deiters' stimulation and 1·1–1·5 msec for i.p.s.p.s evoked by medial nucleus stimulation (Wilson & Yoshida 1969*a*, *b*).

(2) *Location of vestibulocollic neurones in the vestibular nuclei*

The location of 141 antidromically activated neurones was determined either from a dye mark placed at the recording site or by reference to a nearby mark. All the locations are shown in Fig. 2, which consists of cross-sections through the vestibular nuclei at six different levels. Section 1 shows the medial and descending nuclei caudal to Deiters' nucleus, whose dorsocaudal area first appears in section 2. The rostral end of the descending nucleus is seen in section 3, where the part of the medial nucleus under the stria acoustica borders on both the descending and Deiters' nuclei. Deiters' nucleus gets larger in sections 4 and 5, before its rostralmost part is reached in section 6. These last three sections also contain much of the rostral part of the medial nucleus. It must be appreciated that the Figure summarizes the results of many experiments. Because of variations in the shape of individual sections, the placement of recording sites is sometimes approximate. Furthermore, the number of marks at any one level or in a particular nucleus does not necessarily reflect the normally occurring density of neurones, because more tracks were made in some regions than in others.

Fig. 2 shows that, as expected from anatomical and physiological studies, the ipsilateral Deiters', medial and descending nuclei all project to the C3 grey matter. A small number of neurones was found in the caudal superior nucleus, near the border of Deiters' nucleus; we assume that these are Deiters' neurones. From the somatotopic organization of Deiters' nucleus described by Pompeiano & Brodal (1957), vestibulocollic neurones would be expected mainly in the rostroventral part of the nucleus. In fact, while they are almost lacking in the dorsal region of the mid-rostral part of the nucleus they are present not only rostroventrally but also in the middle of the nucleus (section 4) and even dorsocaudally (sections 2, 3), a region associated with lumbar-projecting neurones and others with long axons. Many of these dorsal and caudal neurones project only to the neck (Fig. 2) and almost none have axons that reach lumbar levels (section 4). This provides further evidence for the view that the somatotopic organization of Deiters' nucleus is rather blurred (Wilson, 1972).

It is known that the MVST is bilateral (Nyberg-Hansen, 1964) and that neck motoneurones receive disynaptic input from the contralateral labyrinth (Akaike *et al.* 1973*b*; Wilson & Maeda, 1974). Fig. 2 shows that

vestibular neurones projecting contralaterally to C3 are also found in the lateral (see also Yoshida, 1924), medial and descending nuclei. The small number of neurones in the contralateral nuclei in Fig. 2 is partly due to the small number of experiments in which they were sampled (nine, as opposed to twenty-three for the ipsilateral nuclei).

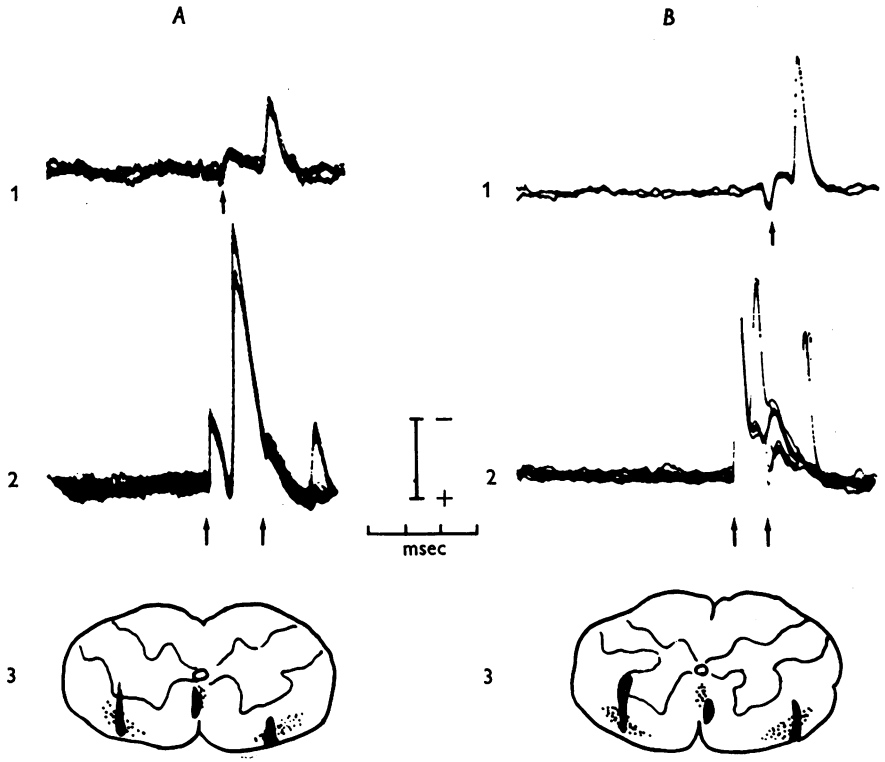

Fig. 3. Projection of vestibulocollic neurones in LVST and MVST. The Deiters' neurone in *A* was activated antidromically from the left (ipsilateral) C3 grey matter with a 10 μA shock (*A*1; T = 5·5 μA). It was blocked by a preceding 20 μA stimulus to the area of the left LVST (*A*2); when the neurone failed to fire on top of the antidromic field potential, there was a response to the C3 stimulus. A 250 μA stimulus to the MVST had no effect. The descending nucleus neurone in *B* was fired antidromically from right (contralateral) C3 with a 12 μA shock (*B*1; T = 5 μA). The neurone sometimes responded to an 80 μA stimulus to the area of the MVST and when it did the response to the C3 stimulus was blocked (*B*2). An 800 μA stimulus to the LVST on either side had no effect. In all traces arrows indicate time of stimulus. *A*3 and *B*3 show approximate locations of VSTs (dots) and lesions marking stimulating locations in the two experiments. Calibration 250 μA for *A*, 500 μA for *B*.

(3) *Projection of vestibulocollic neurones in the LVST and MVST*

Whether the axon of a vestibulocollic neurone was in the LVST or MVST was determined in some cases by stimulation at C5–7, and in later experiments by stimulation of the caudal medulla or of C1. At the C5–7 level the LVST has shifted somewhat medially (Nyberg-Hansen & Mascitti, 1964; Petras, 1967; Fig. 1). It was nevertheless possible to classify some axons because they responded only to stimulation of a lateral or of a medial, somewhat dorsal, electrode. With C1 stimulation we classified an axon as being in the LVST or MVST if only one electrode excited the axon, or if there was a difference in threshold of at least 3:1 (Akaike *et al.* 1973*a*). The responses of two neurones are illustrated in Fig. 3. The antidromic response of the neurone in *A* was blocked by collision only by stimulation of the left LVST, at a threshold of 20 μA; the ratio of MVST to LVST threshold was at least 13:1. For the neurone in Fig. 3*B*, which responded only to MVST stimulation at 80 μA, the threshold differential was at least 10:1. For the whole population the mean differential exceeded 10:1.

TABLE 1. Classification of projection of neurones of identified location in the vestibular nuclei

Location	Left LVST	MVST	Right LVST
Ipsilateral (left) nuclei			
Rostral Deiters	11	4	
Middle Deiters	2	1	
Caudal Deiters	8	1	
Descending	4	3	
Medial	0	4	
Contralateral (right) nuclei			
Deiters	2	5	1
Descending	0	6	0
Medial	0	3	0

Neurones in the table were classified by stimulation at C1 (49) or C5–7 (6).

38 neurones of known location were classified by tract projection and the results are summarized in Table 1. Although the numbers are small, several points are clear. As expected (Pompeiano & Brodal, 1957; Wilson, 1972) axons in the LVST arise mainly from neurones in the ipsilateral Deiters' nucleus. Some LVST neurones, however, are in the descending nucleus, which may also contribute to this tract. Ipsilateral MVST neurones are found not only in the medial and descending nuclei (Nyberg-Hansen, 1964; Wilson, 1972) but also in Deiters' nucleus. The presence of MVST neurones in Deiters' nucleus was earlier demonstrated

by Akaike *et al.* (1973*a*) and Akaike (1973), who were not able to discriminate between axons in the ipsilateral and contralateral MVST. Our results confirm that there are MVST neurones in Deiters' nucleus and show that some of them project to the ipsilateral cervical cord.

Table 1 also shows that fibres from the contralateral vestibular nuclei are almost entirely in the MVST, as expected from the fact that the LVST is ipsilateral (Nyberg-Hansen & Mascitti, 1964; Brodal, 1974) and that potentials evoked in neck motoneurones by contralateral vestibular nerve stimulation are abolished by MLF lesions (Akaike *et al.* 1973*b*; Wilson & Maeda, 1974).

(4) *Further destination of axons of vestibulocollic neurones*

Neurones activated by antidromic stimulation at C3 were tested to see if their axons also branched more caudally in the spinal cord. All neurones were tested by stimulation at C5–7, but not all were tested at either or both lower levels (thoracic and lumbar). Of the whole population of neurones, some were activated antidromically only from the C3 grey matter. Others, such as the neurone of Fig. 4 that branched to the thoracic cord, were also activated from more caudal tract electrodes. A summary of branching, by nuclei, is given in Table 2.

On the ipsilateral side 62% of Deiters' neurones branch to the cervical enlargement, only half that number to Th3–4, and almost none to the lumbar cord. This branching pattern is very different from that of Deiters' neurones projecting to the grey matter of the cervical enlargement, 50% of which branch to upper lumbar levels (Abzug *et al.* 1974). Fig. 2 shows that neurones branching to C5–7 (open symbols) are present at all rostrocaudal levels of Deiters' nucleus, as are non-branching neurones (filled symbols). Branching of Deiters' neurones is not restricted to those with axons in the LVST: three quarters MVST neurones also branched, but only as far as C5–7. Table 2 shows that descending nucleus neurones branch as extensively as those in Deiters' nucleus, but there is no branching of medial nucleus neurones.

Contralaterally the ratio of branching to non-branching Deiters' neurones is very similar to that in the ipsilateral nucleus, but there is a much smaller projection to Th3–4. There is considerable branching of descending nucleus neurones, and one branching neurone in the small medial nucleus population.

From the latency of the responses to stimulation of C3 and of C5–7 it is possible to draw some inferences about the branching of axons. As might be expected, the C5–7 latency is usually equal to or somewhat longer than the C3 latency: the conduction time in approximately 30 mm of VST must be compensated to a considerable extent by slowed conduction in the C3 branch. For several neurones,

however, the C5–7 latency is much longer than expected. A likely explanation of this observation is that such neurones may make up a population with extensive upper cervical branching and only a smaller-diameter, slowly conducting, axon projecting more caudally.

TABLE 2. Branching of vestibulocollic neurones

	C3 only	C5–7	Th3–4	L1–4
Ipsilateral				
Deiters	28/73 (38)	45/73 (62)	14/46 (30)	2/49 (4)
Descending	10/23 (44)	13/23 (56)	1/7 (14)	0/9 (0)
Medial	11/11 (100)	0/11 (0)	0/5 (0)	0/6 (0)
Contralateral				
Deiters	5/14 (36)	9/14 (64)	1/12 (8)	0/14 (0)
Descending	5/8 (62)	3/8 (38)	0/8 (0)	0/8 (0)
Medial	3/4 (75)	1/4 (25)	0/4 (0)	0/4 (0)

Table includes neurones accurately located with reference to marks, as well as some whose location was known only approximately. Numbers in parentheses are percentages.

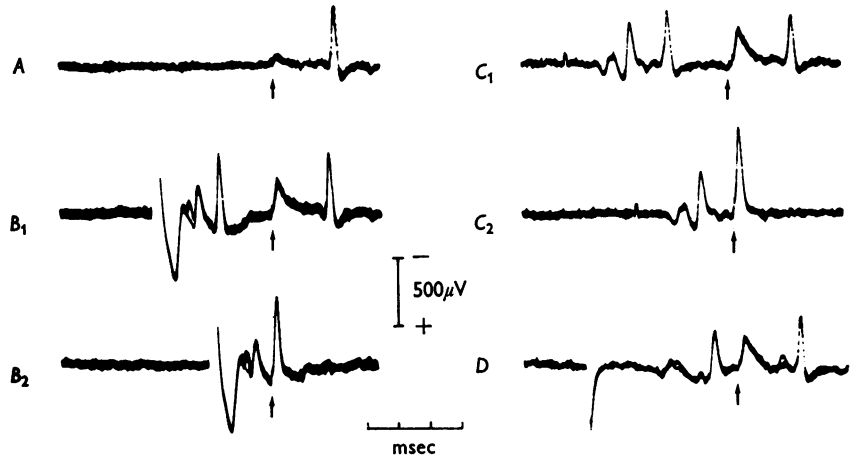

Fig. 4. Branching of a vestibulocollic neurone. This neurone, located at the border of Deiters' nucleus and the rostral medial nucleus, was activated antidromically from the ipsilateral C3 grey matter with a 4 μA shock, A; T = 2 μA. A shock to the VSTs in C5 also fired this neurone (B_1). When the interstimulus interval was 3·6 msec, there was a response to both stimuli (B_1), but when the interval was reduced to 2 msec the second response was blocked by collision (B_2). There was also a response to stimulation at Th3 (C_1) with collision block at a short interstimulus interval (C_2). Stimulation at L2 caused an antidromic field potential, but did not fire the neurone (D). Arrows indicate stimuli, where necessary.

(5) *Input from the labyrinth to vestibulocollic neurones*

Ipsilateral excitation. As can be seen in Fig. 2 (filled and open circles) a great majority of those neurones that were tested for input from the labyrinth were excited mono or disynaptically; in Deiters' nucleus this was the case at all rostrocaudal levels.

An example of response to vestibular nerve stimulation is illustrated in Fig. 5. This neurone responded at low multiples of N_1 threshold (near $1{\cdot}8N_1T$), and with stronger shocks clearly fired monosynaptically on top

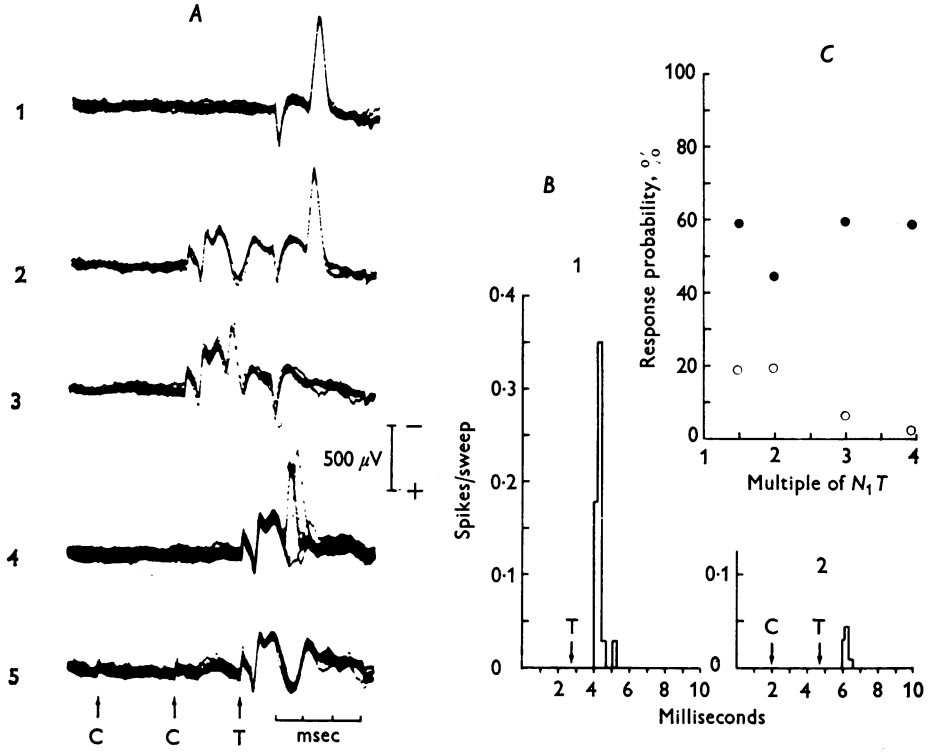

Fig. 5. Commissural inhibition of vestibulocollic neurones. *A* illustrates the response of a neurone in contralateral rostral Deiters' nucleus. The antidromic response in 1 was evoked by a 12 μA stimulus to C3 (T = 8 μA). A weak shock to the labyrinth ipsilateral to the neurone evoked only a field potential (2), but a stimulus $2N_1T$ fired the neurone, blocking the antidromic response (3). The response to labyrinth stimulation at $1{\cdot}8N_1T$ (4) is inhibited by two conditioning stimuli to the other labyrinth (5). *B* and *C* illustrate inhibition of another neurone, in the rostral descending nucleus. The PST histogram in *B*1 (100 sweeps) show the unconditioned response, that in *B*2 the result of a single conditioning stimulus at $1{\cdot}5N_1T$. The graph in *D* shows effect of increases in the strength of the conditioning stimulus. Filled circles unconditioned, open circles conditioned responses.

of the N_1 potential (not illustrated). At appropriate condition-test intervals (Fig. 5*A*3) the synaptic response blocked the antidromic response by collision. With some neurones that had higher thresholds it was possible to observe collision block by labyrinth-evoked activity, but not to determine unambiguously if the neurone responded mono- or disynaptically. Table 3 summarizes the behaviour of those neurones whose responses could be classified.

TABLE 3. Effect of vestibular nerve stimulation on vestibulocollic neurones

	Ipsilateral		Contralateral
	Excitation	Monosynaptic excitation	Commissural inhibition
Ipsilateral neurones			
Deiters	53/76 (70)	39/65 (60)	3/14 (21)
Descending	20/24 (83)	14/19 (74)	4/7 (57)
Medial	11/11 (100)	9/9 (100)	3/4 (75)
Contralateral neurones			
Deiters	12/14 (86)	12/14 (86)	5/6 (83)
Descending	7/7 (100)	6/7 (86)	2/5 (40)
Medial	4/4 (100)	4/4 (100)	3/4 (75)

The first two columns are for excitation of neurones by stimulation of the labyrinth ipsilateral to them, the third for inhibition from the contralateral labyrinth. Numbers in parentheses are percentages.

A very high fraction of ipsilaterally and contralaterally projecting neurones in all nuclei received monosynaptic excitation strong enough to make them fire: the range is from 60% in ipsilateral Deiters' to 100% in the medial nucleus. Within Deiters' nucleus 57% of caudal neurones (12/21), 40% of neurones in the middle of the nucleus (4/10) and 68% (23/34) of rostroventral neurones were excited. The values in the medial and descending nuclei are somewhat higher than those obtained earlier for spinal projecting neurones identified by MLF stimulation at C3 (Wilson, Wylie & Marco, 1967*b*, 1968). The values for Deiters' are somewhat higher (ipsilateral) or much higher (contralateral) than those for Deiters' neurones projecting to the cervicothoracic cord (51%, Wilson *et al.* 1967*a*). The cervicocollic projection obviously receives a strong input from the ipsilateral labyrinth; there is no difference between input to branching and non-branching neurones.

Commissural inhibition. Stimulation of the contralateral labyrinth inhibits the activity of many vestibular neurones, as first described by Shimazu & Precht (1966). We looked for commissural inhibition of vestibulocollic neurones, as illustrated in Fig. 5. The excitatory ipsilateral

response of the neurone of Fig. 5*A* (3, 4) was abolished by two weak preceding shocks to the contralateral vestibular nerve (Fig. 5*A*5). Many neurones were also studied as the one in Fig. 5*B*, *C*. The excitatory response is displayed as a PST histogram in Fig. 5*B*1; it is sharply depressed by a conditioning shock of $1{\cdot}5N_1T$ delivered to the contralateral vestibular nerve (Fig. 5*B*2). Fig. 5*C* shows that the inhibition, already strong at $1{\cdot}5N_1T$, is complete at $4N_1T$. The threshold of inhibition was not always measured precisely but it was usually less than $4\ N_1T$ and often between 1 and $2N_1T$. The test response was always labyrinth-evoked: antidromic spikes were not abolished by contralateral stimulation even in cases where commissural inhibition of synaptic responses was present. With strong contralateral stimuli vestibular neurones may be not only inhibited, but also excited by reticular pathways (Shimuzu & Precht, 1966). We have seen such excitation, but have not studied it systematically.

The fraction of inhibited neurones is quite sizeable, as shown in Table 3. On the ipsilateral side the fraction is higher in the medial and descending nuclei than in Deiters'. Inhibition seems to be more common in contralateral neurones, because of the relatively greater number of inhibited Deiters' neurones.

DISCUSSION

The vestibulocollic neurones that we studied were activated antidromically by localized microstimulation, mainly in the C3 grey matter. Stimulation near and dorsal to the motor nuclei could activate axon branches making synaptic contact with nearby motoneurones or interneurones, or branches headed dorsally towards different interneurone populations; some branches may make functional contact with more than one category of target neurones. Our results indicate that this population is heterogeneous in its properties: neurones are widely distributed throughout the vestibular nuclei, and do not uniformly branch caudally or receive ipsilateral or contralateral labyrinthine input. This shows that the vestibulocollic projection does not act only on the neck segments of the spinal cord and that its different subgroups are likely to serve more than one function.

The pronounced input to vestibulocollic neurones from the ipsilateral labyrinth, the distribution of these neurones in the ipsilateral and contralateral vestibular nuclei, and the contribution of the various nuclei to the LVST and MVST have been considered in Results. Some other properties deserve further discussion.

Commissural inhibition. This inhibition has been found for vestibular neurones projecting to the extraocular nuclei (Maeda, Shimazu & Shinoda, 1971; Baker, Precht & Llinàs, 1972) and Maeda, Shimazu & Shinoda

(1972) have suggested that it is an important factor in the generation of rhythmic eye movements (nystagmus). There is also, however, commissural inhibition of vestibular neurones projecting to the flocculus (Shinoda & Yoshida, 1975; Wilson, Maeda, Franck & Shimazu, 1976). Earlier observations demonstrated that some spinal-projecting vestibular neurones also could be inhibited by stimulation of the contralateral vestibular nerve (Wilson, Wylie & Marco, 1968) and our results (Table 3) show that a substantial fraction of vestibulocollic neurones receive commissural inhibition. It may be that commissural inhibition has several functional roles. On the other hand, it may only influence a mixed population of neurones all involved in the regulation of eye, head and trunk movement. One test of this notion would be to see whether the inhibition affects vestibular neurones projecting to the lumbar enlargement.

Branching. Since the demonstration that some vestibulospinal axons may branch to widely different levels of the spinal cord (Abzug *et al.* 1974), similar branching has been observed in other descending tracts: reticulospinal (Peterson, Maunz, Pitts & Mackel, 1975) and pyramidal (Shinoda, Arnold & Asanuma, 1976). Our results provide further information about branching in the vestibulospinal system.

The vestibulocollic projection contains many branching neurones. As pointed out in Results our branching Deiters' population is very different from that studied by Abzug *et al.* (1974). Vestibulocollic neurones give off branches to the upper cervical grey matter and their long branch almost never reaches upper lumbar levels. The Deiters' neurones of Abzug *et al.* (1974) give off branches to the grey matter of the cervicothoracic enlargement, and at least half have a long branch that reaches lumbar levels. Together, these results demonstrate that the vestibulospinal tracts do not contain just one group of branching neurones that project diffusely to all spinal levels. Instead there are at least two groups, with different projection zones. In other words, there is organization in the branching pattern of VST neurones.

Possible functional correlates of branching. It is likely that branching and non-branching neurones have different functions. Although both groups of neurones receive ipsilateral vestibular afferents to the same extent, there may be a distinction in the type of vestibular afferent they receive, or in the distribution of other inputs, for example from the cerebellum. The two populations of neurones may also have different targets. One possibility would be that non-branching vestibulocollic neurones act in a relatively localized region, in neck segments only, and directly on motoneurones. Branching neurones would act more diffusely, via interneurones at different spinal levels. This appears not to be the case, however, because synapses with motoneurones are made by both branching

and non-branching axons (manuscript in preparation). Further experiments are required to detect any relation between input to vestibulocollic neurones, presence or absence of branching, and efferent connexions.

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