Archives Italiennes de Biologie, 129: 21-41, 1991.

THE ROLE OF DIFFERENT SIZE VESTIBULOSPINAL NEURONS IN THE STATIC CONTROL OF POSTURE

O. POMPEIANO

Dipartimento di Fisiologia e Biochimica, Università di Pisa, Via S. Zeno 31, 56100 Pisa, Italy

INTRODUCTION

The lateral vestibular nucleus or nucleus of Deiters (also called «grosszelliger Vestibulariskern») has been originally described as a structure characterized by the presence of giant cells (14). In a cytoarchitectonic study of the vestibular nuclei, made in collaboration with Alf Brodal (5), we outlined this magnocellular vestibular area (Fig. 1). However, large size multipolar cells were found not only in the lateral vestibular nucleus (LVN), but also in the rostral part of the descending vestibular nucleus (Fig. 1d). The original figure published by Deiters' in his volume on «Untersuchungen über Gehirn und Rüchenmark des Menschen und der Säugethiere» (14) is of interest in this respect, since it shows that the large-size vestibular neurons are surrounded by numerous longitudinally running fiber bundles cut transversally (Fig. 2). These bundles, also outlined in Fig. 1d, correspond to the descending vestibular root fibers, and can be taken as a criterion to unequivocally outline the descending vestibular nucleus (5).

According to Monakow (45, and later) the nucleus of Deiters represents the part of the vestibular nuclear complex in which giant cells undergo chromatolysis following lesions at rostral levels of the spinal cord, thus contributing to the vestibulospinal (VS) projection. This criterion has been shared by Pompeiano and Brodal (51), who found that retrograde changes which occurred after interruption of the upper cervical cord affected large-size neurons located in the LVN, but not in the rostral part of the descending vestibular nucleus. This negative result was attributed to the fact that efferent fibers, contributing to the descending medial lingitudinal fasciculus, give rise to ascending branches whose anatomical integrity prevents the occurrence of the retrograde degeneration.

The idea that the nucleus of Deiters contains only giant cells has been modified by observations made in our cytoarchitectonic study (5), showing that in addition to large multipolar neurons, there are medium-size, frequently oval or spindle-shaped, as well as small-size neurons (Fig. 1). This finding was also confirmed in our experimental anatomical study, showing that degenerated VS neurons of all size were intermingled throughout the LVN; however, the giant cells were relatively more numerous and somewhat larger in the caudal part of this structure than in the rostral part, where the smaller cells are more abundant (51). These differences may bear some relation to the somatotopical arrangement of the origin of the VS fibers, since the rostroventral part of the LVN (rvLVN), which projects

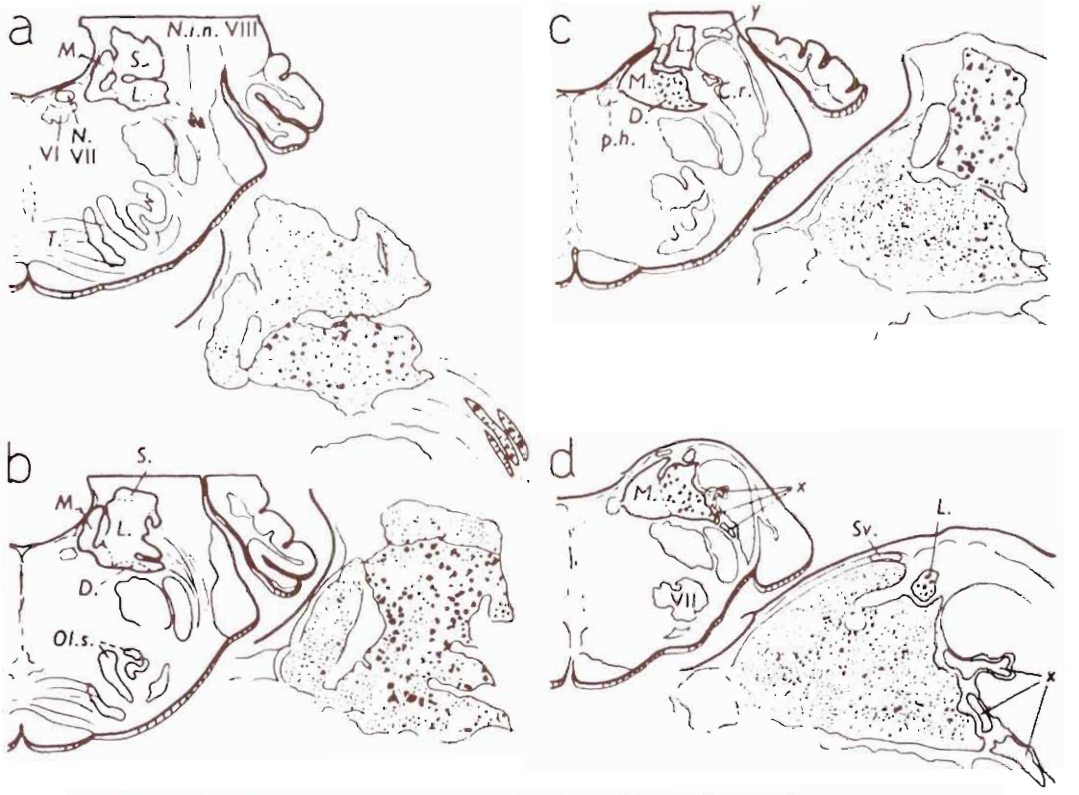

Fig. 1 – *Topography and cytoarchitecture of the lateral vestibular nucleus in the cat as seen in a series of drawings corresponding to transverse thionine stained sections of the medulla taken at equal intervals and labelled progressively from rostral (a) to caudal (d) direction.*

The rings in the descending nucleus represent the fiber bundles of the spinal (descending) root of the vestibular nerve. C.r., corpus restiforme; D., descending vestibular nucleus; L., lateral vestibular nucleus of Deiters; M., medial vestibular nucleus; N.i.n. VIII, nucleus interstitialis nervi vestibuli; N. VII, cranial nerve VII; Ol.s., oliva superior; p.h., nucleus praepositus hypoglossi; S., superior vestibular nucleus; T., trapezoid body; VI, VII, cranial motor nerve nuclei; x,small-celled group x; y, small-celled group y.
From Brodal and Pompeiano (ref. 5, Fig. 1).

to the cervical segments of the spinal cord, contains a relatively large number of small-size neurons, while the dorsocaudal part of the LVN (dcLVN), which projects to the lumbosacral segments of the spinal cord, is made chiefly of giant cells (51). However, after section of the spinal cord at T_{12}-L_1, which interrupts VS axons projecting to the lumbo-sacral segments of the spinal cord (lVS neurons), not only giant, but also medium-and small-size cells of the LVN were affected by retrograde degeneration. In analogy to this finding are the results of histological observations showing that lVS neurons cover a wide spectrum of axonal conduction velocity, as evaluated by antidromic activation of the corresponding axons from the lumbar cord (2, 37, 62).

Since the LVN excites mono- and polysynaptically the α- (26, 43, 64) as well

as the γ-motoneurons innervating the ipsilateral limb extensors (9, 25; cf. 48), we decided to investigate the role that different size lVS neurons exert on static posture.

Functional properties related to cell size have been originally investigated in hindlimb extensor motoneurons, where a set of experimental evidence has led to the formulation of the «size principle» according to which the smaller the size of the motoneurons, the lower is the threshold and the more effective is the corresponding proprioceptive input in exciting them (29, 30, 32, 33, 44, 58; cf. 31, 60). However, the discharge of a given population of neurons in response to a given input can be determined not only by neuronal properties related to cell size (cf. 31, 60), but also by a differential distribution of the relevant input system on different neuronal groups (cf. 6, 7).

It is known that the LVN receives a monosynaptic and polysynaptic excitatory input from the ipsilateral labyrinth (1, 36, 46, 62; cf. 63), through primary afferents originating mainly from macular receptors (22, 57, 59). On the other hand the same structure receives a monosynaptic inhibitory influence from the ipsilateral cortex of the cerebellar vermis (cf. 35), via a direct cerebellar corticovestibular projection (cf. 11). There is also evidence that the LVN of both sides are interconnected by a crossed inhibitory pathway, characterized by VS neurons of one side

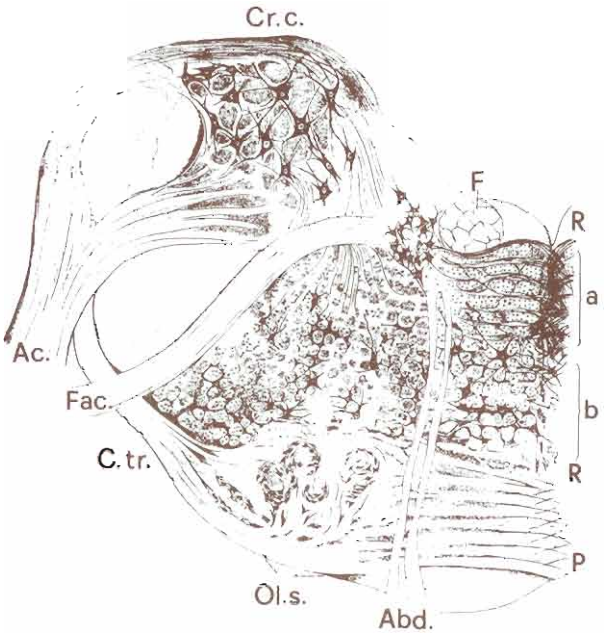

Fig. 2 – *Drawing of a transverse section of the medulla showing the large-size neurons in the vestibular complex, as originally illustrated by Deiters.*

Abbreviations taken from the legend of the original figure: Abd., abducens nerve; Ac., acoustic nerve; Cr.c., crura cerebelli; C.tr., trapezoid body; F, genu facialis; Fac., facial nerve; Ol.s., superior olive; P, pyramid; RR, raphe, dorsal (a) and ventral (b).

From Deiters (ref. 14, Fig. 14).

acting on neurons of the crossed spino-reticulocerebellar pathway (12, 15, 40) and then, through the corticocerebellar loop, on the contralateral LVN (cf. 49). These findings explain why the tonic contraction of limb extensors increases after ablation of the cerebellar vermis (cf. 16), but decreases after interruption of the ipsilateral vestibular nerve. In this instance a postural asymmetry occurs, characterized mainly by hypotonia in the ipsilateral limb extensors and hypertonia in the contralateral ones (cf. 54, 55). These postural deficits, however, disappear with the time after the lesion, due to compensation of the vestibular syndrome (cf. 19, 20, 42, 54-56).

With the experiments reviewed in the present report we tried to compare the resting discharge of different size lVS neurons, recorded in decerebrate cats (52), with that obtained either after bilateral ablation of the medial corticonuclear zone of the cerebellum (4) or after unilateral acute (aVN) or chronic vestibular neurectomy (cVN) (53). We could then obtain information, as to whether the excitatory labyrinth input and the inhibitory cerebellar input were either homogeneously distributed among the different size lVS neurons or differently oriented towards particular groups of neurons.

METHODS

All the experiments were performed on precollicular decerebrate cats operated under ether anaesthesia. Among these experiments 14 cats had the cerebellum intact (52), while 19 cats had been submitted to bilateral ablation of the cerebellar vermal cortex and the fastigial nuclei, which were aspired until the floor of the fourth ventricle (4). Finally a third group of experiments were performed in 29 cats after unilateral vestibular neurectomy (53); in particular, in 14 cats this lesion was made after decerebration, while in 15 cats the animals were first submitted to unilateral vestibular neurectomy under pentobarbital anaesthesia (Nembutal 35 mg/Kg, i.v.) and then decerebrated 2-4 months after vestibular deafferentation. In the first instance a postural asymmetry occurred, characterized mainly by a decreased postural activity in the ipsilateral limbs and an increased activity in the contralateral limbs. In the second instance a compensation of the postural deficits appeared, which was characterized by the disappearence of the asymmetric changes in posture and stretch reflexes produced in the four limbs by the acute vestibular lesion. Moreover, decerebration performed in chronically compensated animals did not elicit a postural asymmetry of the four limbs as described after aVN.

The animals were immobilized with pancuronium bromide (Pavulon, Organon, Oss, NL, 0.6 mg/Kg/h, i.v.) and artificially ventilated. The experimental procedures, methods of recording extracellular unit activity and marking the location of the recording sites have already been described in detail in the original papers. In particular, lVS neurons were antidromically identified by single shock stimulation of the spinal cord between T_{12}-L_2. Most of these neurons were found to be histologically located in the dcLVN, rather than in the rvLVN. Each unit was first tested under resting conditions of the animal. In particular, raw spike train data from segments of spontaneous background discharges of lVS neurons were recorded on magnetic tape. The mean resting discharge rate (imp./sec) was evaluated over a period of time sufficiently long to allow the recording of 400 to 4000 spikes. In addition, the mean interspike interval, standard deviation interval and coefficient of variation (CV), defined as the standard deviation of interspike intervals divided by the interval mean, were calculated following the criteria described previously (4).

Each unit was also tested during rotation about the longitudinal axis of the whole animal at the standard parameters of 0.026 Hz, ± 10° peak amplitude, thus leading to sinusoidal stimulation of labyrinth receptors. Responsive units were those which showed a sinusoidal modulation of their firing rate in relation to the animal displacement, as defined previously (4). In these instances the mean discharge rate or base frequency (imp./sec) evaluated for each unit during animal tilt at the parameters indicated above, closely corresponded to the mean discharge frequency of the same unit recorded at rest.

RESULTS

1. *Resting discharge and conduction velocity of lVS neurons in decerebrate cats with the cerebellum intact.*

The results were obtained in decerebrate cats with the cerebellum intact (52). Among 129 LVN neurons antidromically activated by electrical stimulation of the spinal cord between T_{12} and L_1 (lVS neurons), 110 were spontaneously active, while the remaining 19 were silent. Resting discharge rates, evaluated for 108 out of 110 lVS neurons, ranged from 0.7 to 66.7 imp./sec and averaged 24.5 ± 15.7, S.D. imp./sec. The mean discharge rate obtained for the units responsive to standard parameters of animal tilt (21.3 ± 15.4, S.D. imp./sec, n = 68) was lower than that of the units unresponsive to tilt (29.9 ± 14.7, S.D., imp./sec, n = 40), the difference being statistically significant (t-test, $P < 0.01$). In agreement with the known anatomically projections from the Deiters' nucleus, 85 (78.7%) lVS neurons were located histologically in the dcLVN, while only 23 (21.3%) neurons were located in the rvLVN.

If we consider the total population of lVS neurons exhibiting a background discharge at rest, a clear-cut positive correlation was found between the mean interval and the standard deviation interval of the examined spike trains. The variability of the resting discharge, measured by the coefficient of variation (CV), as defined in the methods, ranged from 0.12 to 2.28 and averaged 0.56 ± 0.49, S.D. (n = 108). Moreover, 35 out of the 108 units (32.4%) showed a CV higher than 0.575. A prominent negative correlation was found between the CV of the spike train and the mean resting discharge rate; in fact, the higher the CV, reflecting a more irregular unit discharge, the lower was the mean background discharge (paired rank, $P < 0.001$; n = 108). Similar results were obtained for the two populations of units responsive and unresponsive to vestibular stimulation.

Across the whole population of lVS units, the axonal conduction velocity varied in an unimodal fashion from 33.8 to 124.8 m/sec, with an average value of 90.0 ± 21.5, S.D. m/sec. Moreover, the antidromic units responsive to vestibular stimulation showed on the average a faster conduction velocity than the unresponsive ones, thus being presumably larger in size (93.9 ± 19.3, S.D. m/sec, n = 68 against 80.7 ± 22.8, S.D. m/sec, n = 40; *t* test between the means, $P < 0.01$) (Fig. 3A, inset, striped and white columns, respectively).

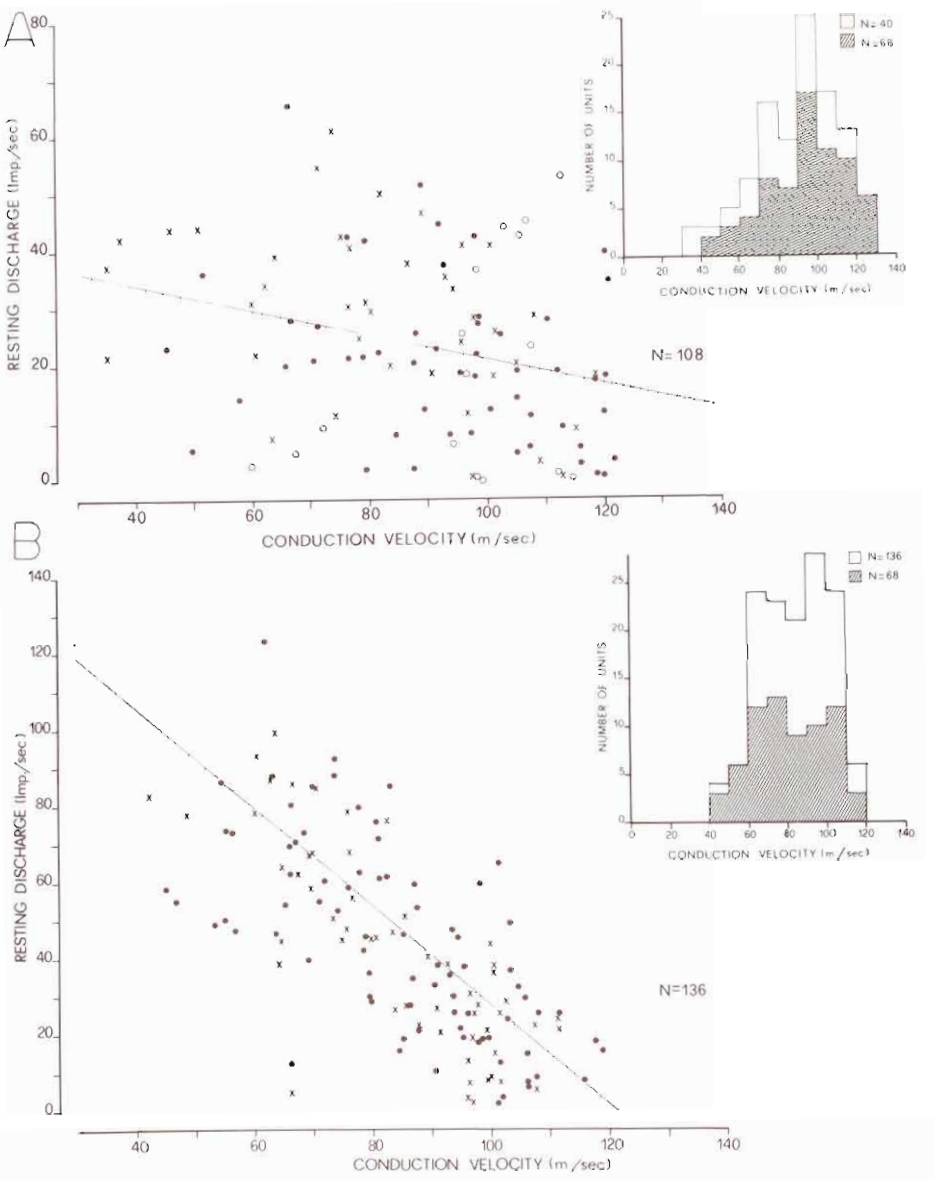

Fig. 3 – *Relation between resting discharge rate of IVS neurons and conduction velocity of the corresponding axons in decerebrate cats before and after partial cerebellectomy.*

A: the graph shows the data of 108 antidromically identified IVS neurons firing at rest in decerebrate cats with the cerebellum intact. Symbols represents units either responsive (●,○; n = 68) or unresponsive (x,n = 40) to roll tilt of the animal at standard parameters; responsive units are indicated by filled or empty circles if located in the dorsocaudal and the rostroventral part of the LVN, respectively. A slight negative correlation was found between the unit resting discharge rate and the conduction velocity of the corresponding VS axon (paired rank, $P < 0.01$; n = 108). *Inset*: distribution of conduction velocity in the general population of IVS units, including 68 responsive (striped columns) and 40 unresponsive units (white columns).

B: the graph shows the data of 136 IVS neurons firing at rest in decerebrate cats after ablation of the cerebellar vermal cortex and fastigial nuclei. Symbols represent units either responsive (●; n = 80) or unresponsive (x; n = 56) to animal tilt. Responsive and unresponsive units were located in both the dorsocaudal and the rostroventral part of the LVN. A prominent negative correlation was found between the unit resting discharge rate and the conduction velocity of its axon (paired rank, $P < 0.001$, n = 136). Inset: distribution of conduction velocity in the general population of IVS units (white columns, n = 136) and the population of units used to evaluate the coefficient of variation (striped columns, n = 68).

From Pompeiano et al. (ref. 52, Fig. 2) and Boyle and Pompeiano (ref. 4, Fig. 3).

If we consider now the whole population of responsive and unresponsive IVS neurons, a *slight* negative correlation was found between the unit resting discharge rate and the conduction velocity of the axons, so that the faster the conduction velocity of its axon, the lower was the unit discharge rate at rest (paired rank, $P<0.01$; $n=108$) (Fig. 3A). Similar results were also obtained for the unresponsive units (paired rank, $P<0.01$, $n=40$), but not for the responsive units (same test, $P<0.05$; $n=68$).

A more detailed comparison for the recorded units was obtained by subdividing the IVS neurons into two groups according to their axonal conduction velocity (either $\leqslant$ 90 m/sec, slow units or $>$ 90 m/sec, fast units). As expected from the results reported above, the resting discharge rate evaluated for all the responsive and unresponsive neurons was slightly but significantly higher for the slow ($n=47$) than for the fast IVS units ($n=61$) (*t* test between the means, $P>0.01$; Fig. 5B). This held true also for the IVS units which were unresponsive to labyrinth stimulation ($n=23$, slow and $n=17$, fast units), but not for the remaining units responsive to vestibular stimulation ($n=24$, slow and $n=44$, fast units).

2. *Resting discharge and conduction velocity of IVS neurons in decerebrate and partially cerebellectomized cats.*

Experiments were performed in decerebrate cats in which the cerebellar vermal cortex and the fastigial nuclei were removed (4). Among 136 LVN neurons antidromically activated by electrical stimulation of the spinal cord between L_1 and L_2 (IVS neurons) the resting discharge rate ranged from 1.7 to 123.2 imp./sec, and averaged 44.1 ± 23.8, S.D. imp/sec; similar values were also obtained for the responsive as well as for the unresponsive units. As shown in the control experiments, the IVS units recorded after partial cerebellectomy were mainly located in the dcLVN ($n=107$, 78.7%) than in the rvLVN ($n=29$, 21.3%).

The mean discharge rate of all the units indicated above was higher than that obtained in preparations with the cerebellum intact. A comparable range and mean value (46.5 ± 21.9, S.D. imp./sec) of discharge rate were also obtained from a smaller population of 68 units whose discharge was examined in more detail. Among these units, 41 responded to standard parameters of roll tilt while the remaining 27 units were unresponsive.

If we consider these two populations of units, a high degree of correlation was observed between the mean interval and the standard deviation interval of the examined spike trains. The variability of discharge, measured by the CV, ranged from 0.078 to 1.09 and averaged 0.26 ± 0.20, S.D. ($n=68$). The mean value was lower than that obtained from IVS neurons recorded in decerebrate cats with the cerebellum intact (0.56 ± 0.49, S.D.; $n=108$). Moreover, the proportion of units showing a CV higher than 0.575 was smaller in partially cerebellectomized animals (6/68, i.e. 8.8%) than in preparations with the cerebellum intact (35/108, i.e. 32.4%). A continuous relation was found between the interval distri-

bution of the spike train and the mean resting discharge rate; in fact, the higher the CV, reflecting a more irregular unit discharge, the lower was the mean background discharge (paired rank, $P<0.001$, $n=68$).

Among the whole population of lVS units ($n=136$), the axon conduction velocity varied in an unimodal fashion from 45.0 to 118.7 m/sec, with an average value of 84.3 ± 17.1, S.D. m/sec (Fig. 3B, inset, white columns); comparable values (81.9 ± 18.3, S.D. m/sec) were also obtained from the population of 68 units described above (Fig. 3B, inset, shaded columns).

If we consider the whole population of responsive and unresponsive lVS units, a *prominent* negative correlation was found between the unit resting discharge rate and the conduction velocity of the corresponding axon. In general, the faster the conduction velocity of the axon, the lower was the unit discharge rate at rest (paired rank, $P<0.001$, $n=136$) (Fig. 3B). Similar results were also obtained for both the responsive and the unresponsive units.

If we subdivide the lVS units responsive to animal tilt into two groups according to their axonal conduction velocity (either $\leqslant$ 90 m/sec, slow units or $>$ 90 m/sec, fast units), it appears that the resting discharge rate was much higher for the slow ($n=45$) than for the fast lVS neurons ($n=35$), and similar result was also obtaimed for the unresponsive units (*t* test between the means. $P<0.0001$; Fig. 5A). This difference was more prominent than that obtained in preparations with the cerebellum intact, where it involved only units unresponsive to labyrinth stimulation. It seems, therefore, that the release phenomenon, characterized by the increased resting discharge following partial cerebellectomy, affected the slow units rather than the fast units (compare Fig. 5, A with B).

3. *Resting discharge and conduction velocity of lVS neurons in decerebrate cats after ipsilateral vestibular neurectomy.*

The experiments were performed either in decerebrate cats submitted to unilateral acute vestibular neurectomy (aVN) or in cats decerebrated 2-4 months after unilateral chronic vestibular neurectomy (cVN).

Acute vestibular neurectomy (aVN). Among the 170 LVN neurons recorded after ipsilateral aVN, 134 neurons were used for evaluating the mean discharge rate of the corresponding units in the animal at rest (53). The discharge rate of all these units ranged from 0.44 to 76.9 imp./sec, and averaged 26.7 ± 19.4, S.D. imp./sec. Similar results wer also obtained for the antidromic ($n=65$) and the non-antidromic LVN neurons ($n=69$), as identified by stimulating the spinal cord between T_{12} and L_1. Moreover, no significant difference in resting discharge rate was found between the neurons responsive to standard parameters of tilt and those unresponsive.

The variability of resting discharge of these units, measured by the CV, ranged from 0.08 to 1.09 and averaged 0.52 ± 0.29, S.D. ($n=134$). A comparable mean value was obtained for both the antidromic ($n=65$) and non-antidromic units ($n=69$);

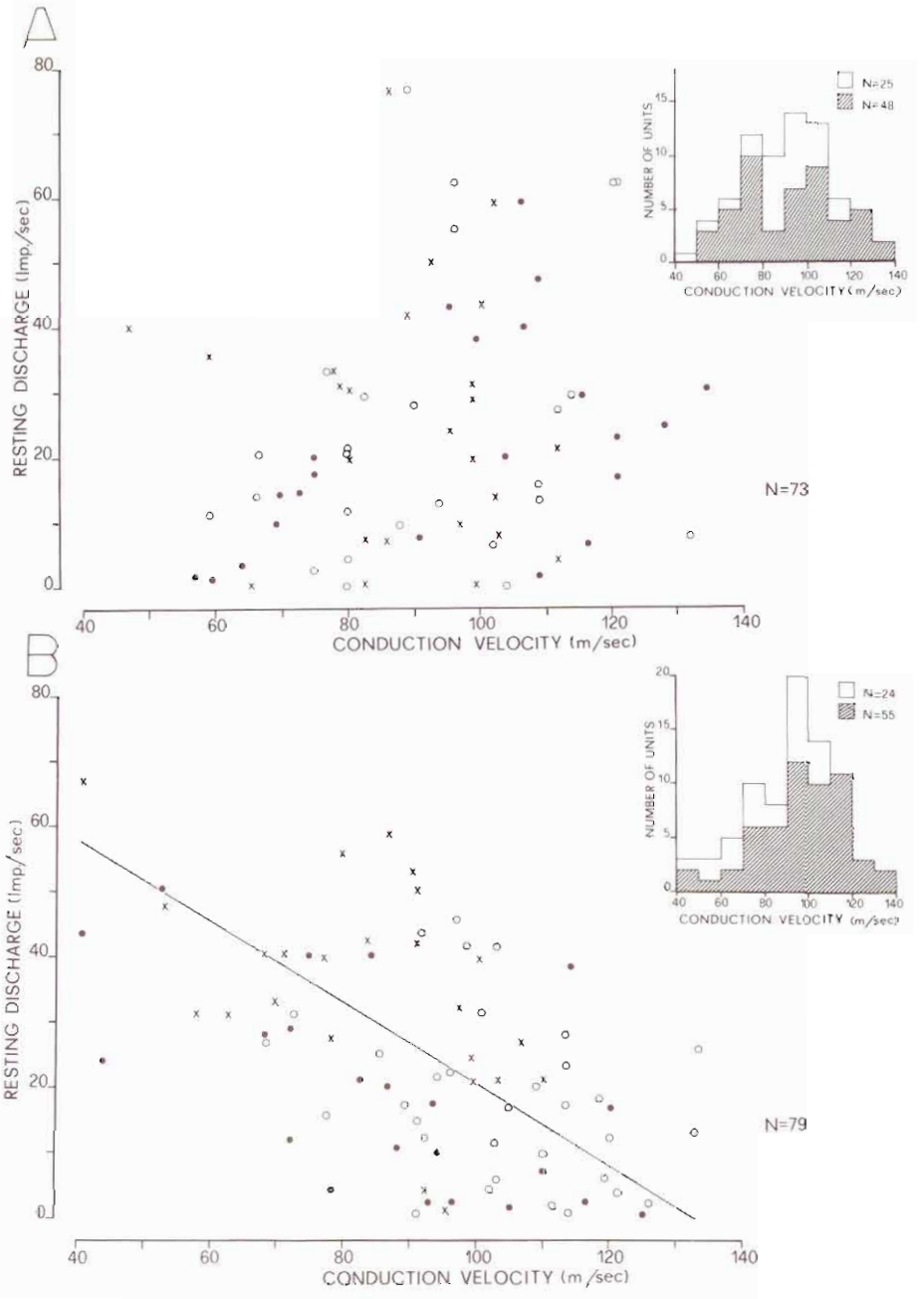

Fig. 4 - *Relation between resting discharge rate of lVS neurons and conduction velocity of the corresponding axons in decerebrate cats after ipsilateral aVN or cVN.*

A: graph showing data of 73 antidromically identified lVS neurons recorded after aVN. Filled circles (n = 22) and open circles (n = 26) represent units responsive to standard parameters of roll tilt of the animal which were located in the dorsocaudal and the rostroventral part of the LVN, respectively; crosses (n = 25) were units unresponsive to animal tilt. There was no correlation between the unit resting discharge rate and the conduction velocity of the corresponding VS axon. Inset: distribution of conduction velocity in the two populations of lVS units responsive (striped columns; n = 48) and unresponsive to labyrinth stimulation (white columns; n = 25).

B: graph showing data of 79 lVS neurons recorded after cVN. Among units responsive to roll tilt of the animal, 22 (filled circles) and 33 (open circles) were located in the dorsocaudal and the rostroventral part of the LVN, respectively; the remaining 24 units (crosses) were unresponsive to animal tilt. A negative correlation was found beween the unit resting discharge rate and the conduction velocity of the corresponding VS axon (*t* test, $P < 0.001$; n = 79). Inset: distribution of conduction velocity of the lVS units responsive to labyrinth stimulation (striped columns; n = 55) and those unresponsive (white columns; n = 24).

From Pompeiano et al. (ref. 53, Fig. 4).

in addition, 45 out of 134 units (33.6%) showed a CV higher than 0.575. A strong negative correlation was found between the CV of the spike train and the mean resting discharge rate, the higher the CV, reflecting a more irregular unit discharge, the lower was the mean discharge rate (t test, $P<0.001$, $n=134$). These findings, which affected both responsive and unresponsive units, did not greatly differ from those obtained in control experiments with the vestibular nerves intact.

Across the population of tested units ($n=73$), the axon conduction velocity varied in a unimodal fashion from 46.9 to 134.7 m/sec, with an average value of 92.2 ± 19.1, S.D. m/sec; comparable values were obtained from the two populations of responsive ($n=48$) and unresponsive units ($n=25$) (Fig. 4A, inset, striped and white columns, respectively).

Interestingly, no significant relation was found between unit resting discharge rate and axonal conduction velocity (Fig. 4A). This was in contrast to the results obtained in control experiments, where a slight negative correlation was found between these two parameters; in particular, the faster the conduction velocity of VS axon, the lower was the unit discharge rate at rest. Moreover, similar results were obtained for the responsive and the unresponsive units.

The lack of correlation between resting discharge and conduction velocity of the units described above depended on the fact that the average resting discharge of the slow units (conduction velocity $\leqslant 90$ m/sec) decreased after aVN, whereas that of the fast units (conduction velocity >90 m/sec) slightly increased with respect to the control values (compare Fig. 5, C with B).

Chronic vestibular neurectomy (cVN). Among the 189 LVN neurons recorded after cVN, 161 neurons were used to evaluate the mean discharge rate in the animal at rest (53). The discharge rate of all these units ranged from 0.6 to 71.4 imp./sec and averaged 25.6 ± 17.5, S.D. imp./sec. Both antidromic ($n=72$) and non-antidromic LVN neurons ($n=89$) showed comparable values. However, the resting discharge rate of all the neurons responsive to standard parameters of tilt (22.4 ± 16.9, S.D. imp./sec; $n=114$) was on the average lower than that of the unresponsive neurons (33.4 ± 16.8, S.D. imp./sec; $n=47$), the difference being statistically significant (t test, $P<0.001$).

The coefficient of variation (CV) ranged from 0.06 to 1.25 and averaged 0.50 ± 0.31, S.D. ($n=161$). A comparable mean value was found for both the antidromic ($n=72$) and the non-antidromic units ($n=89$). The number of regularly discharging units increased after cVN; in particular, the proportion of units with CV ranging between 0 and 0.35 increased while that of the units with a CV between 0.35 and 0.57 decreased in chronic with respect to acute preparations (Yates corrected χ^2, $P<0.054$ for the former group of units, $P<0.05$ for the latter group).

As shown after aVN a strong correlation was found after cVN between the CV and the mean resting discharge rate, the higher the CV, reflecting a more irregular unit discharge, the lower was the mean background discharge (t test, $P<0.001$, $n=161$). Similar results were found for both responsive and unresponsive units.

Across the population of tested units ($n=79$), the axon conduction velocity varied

in a unimodal fashion from 40.4 to 133.3 m/sec, with an average value of 92.7 ± 20.0, S.D. m/sec; comparable values were obtained for the responsive (n = 55) and the unresponsive VS units (n = 24) (inset of Fig. 4B, striped and white columns, respectively).

In contrast to the results obtained after aVN, a negative correlation was observed in the group of chronic experiments between unit resting discharge rate and conduction velocity of the corresponding VS axon (Fig. 4B). In particular, the faster the conduction velocity of its axon, the lower was the unit discharge rate at rest (*t* test, $P < 0.001$; n = 79). This finding, which affected both the responsive and unresponsive units, is similar to that obtained in control experiments with the vestibular nerves intact (52). The correlation between resting discharge and conduction velocity of the units was due to a partial recovery of the resting discharge of the slow units, while the average resting discharge of the fast units decreased

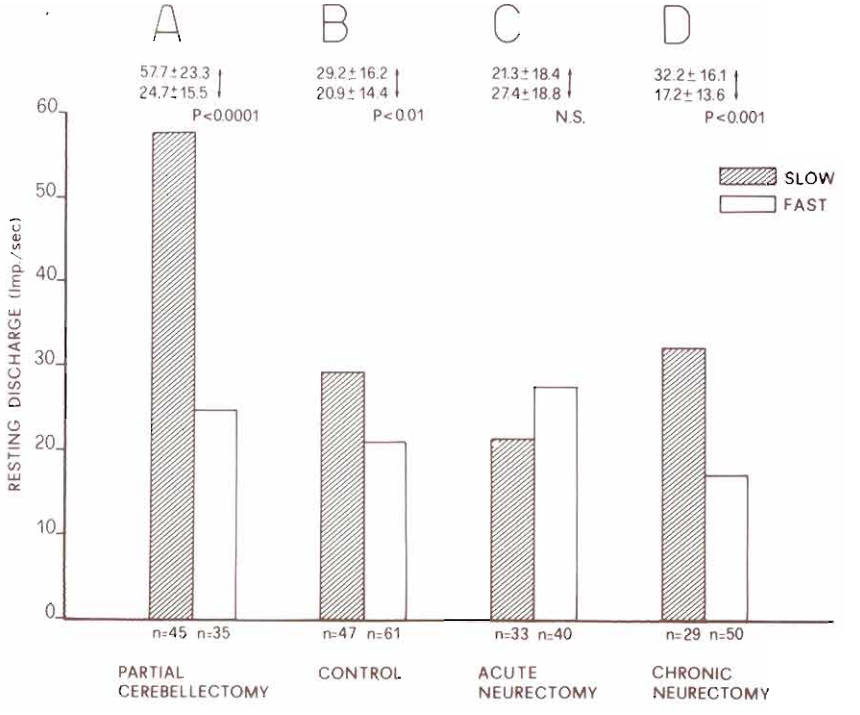

Fig. 5 – *Resting discharge rate of slow- and fast-conducting IVS neurons.*

The results obtained in precollicular decerebrate cats with the cerebellum and the vestibular nerves intact (B) were compared with those elicited either after partial cerebellectomy (A) or after ipsilateral aVN (C) or cVN (D). Striped and white columns refer to the two populations of slow and fast IVS neurons, whose conduction velocity of axons ranged between 40-90 and 91-140 m/sec, respectively. For each experimental condition, the means ± S.D. of the resting discharge rates (in imp./sec) evaluated for slow and fast units are given.

Arrows indicate the differences of means that are statitically significant; N.S., no significant difference (*t* test). The number of recorded units is indicated below each column; in A only the units responsive to sinusoidal roll tilt of the animal at standard parameters were evaluated, while in B, C, D both responsive and unresponsive units were used for the analysis.

with respect to the value obtained after aVN (compare Fig. 5D, with C). As a result of this finding, the difference between slow and fast lVS neurons became statistically significant (*t*-test between the means, $P<0.001$; Fig. 5D).

DISCUSSION

In the experiments reported above, the spontaneous discharge of the LVN neurons, antidromically activated by stimulation of the spinal cord at T_{12}-L_2 (lVS neurons), has been related to cell size inferred on the basis of the conduction velocity of their axons. Observations made on a different neuronal model, i.e. the α-motoneurons, had in fact shown that a close relation exists between the conduction velocity of axons and cell size as measured by input resistance and conductance (38, 39) and by direct histological measurements of dye-injected neurons (3, 13, 39).

The resting discharge of lVS neurons was recorded in decerebrate cats with the cerebellum intact (52), and the results obtained were compared with those occurring either after partial cerebellectomy, which suppressed the tonic inhibitory influence normally exerted by the cerebellar vermis on the underlying LVN neurons (4), or after unilateral acute or chronic vestibular deafferentation, which deprived the corresponding LVN neurons of a tonic excitatory drive (53).

This comparison is justified by the fact that in all these groups of experiments: 1) a large and comparable number of identified lVS neurons was recorded, 2) the corresponding vestibulospinal axons covered the same range of conduction velocity and had comparable mean values; and, finally, 3) the majority of these neurons were histologically located in the dcLVN, in agreement with the pattern of somatotopical organization of the VS projection, showing that this part of Deiters nucleus projects to the lower segments of the spinal cord (51).

Comparison of the results obtained in preparations with and without the cerebellum.

In precollicular decerebrate cats with the cerebellum intact (52) the resting discharge of all the spontaneously firing lVS neurons (24.5 ± 15.7, S.D. imp./sec; $n=108$) was significantly lower than that obtained in partially cerebellectomized animals (44.1 ± 23.8, S.D. imp./sec; $n=136$).

In both groups of experiments the recorded lVS neurons formed a continuoum between the more regularly discharging units, characterized by an higher firing rate and a low coefficient of variation (CV) and the more irregularly discharging units characterized by a low firing rate and a high CV. However, as expected, the CV obtained in preparations with the cerebellum intact was on the average higher (0.56 ± 0.49, S.D.) than that obtained in partially cerebellectomized preparations (0.26 ± 0.20, S.D.), thus reflecting a more irregular unit discharge. These characteristics of resting discharge and CV of the lVS neurons can easily be understood, since in preparations with the cerebellum intact these neurons are under the tonic inhibitory control of the cerebellar vermis.

It is of interest that in the latter preparations a *slight* negative correlation was found between the resting discharge of all the recorded lVS neurons and the conduction velocity of the corresponding axons, so that the faster the conduction velocity, the lower was the unit discharge rate at rest (paired rank, $P<0.01$; Fig. 3A). This relationship, which reflects comparable physiologic properties of different size vestibular afferents (23; cf. 63, for ref.), involved the units unresponsive to standard parameters of tilt rather than those responsive. It appears, therefore, that the lVS neurons responsive to tilt are more prominently inhibited by the cerebellar cortex than the unresponsive units. These findings differ from those obtained after partial cerebellectomy (4), where a *prominent* negative correlation was found between resting discharge and conduction velocity of all the lVS units including both the responsive and unresponsive ones (paired rank, $P<0.001$; Fig. 3B). In this instance the lVS neurons discharging regularly at high rate were characterized by a slower conduction velocity of their axons (smaller units), while the units discharging irregularly at low rate by a faster conduction velocity (larger neurons).

If we assume that the excitatory pathways responsible for the background dicharge of the lVN neurons are homogeneously distributed among the lVS neurons, thus making an equal number of synaptic contacts with different size lVS neurons, then the results described above in cerebellectomized animals would be in agreement with the «size principle», which states that the smaller the size of the neurons the higher is the input resistance, so that the lower is the threshold and the more effective is the corresponding input in exciting them. A similar conclusion was originally proposed for the proprioceptive input exerting an excitatory influence on hindlimb extensor motoneurons (29, 30, 32, 33, 44, 58; cf. 31, 60). In Deiters' nucleus the excess of excitation exerted by the afferent volleys impinging on the lVS neurons in the animal at rest would be smaller in large-size neurons, since they fired at lower rate, than in small-size neurons, which fired at higher rate.

The reduced slope of the regression line relating the resting discharge of the VS neurons to the conduction velocity of the corresponding axon in the experiments with the cerebellum intact (53), with respect to the cerebellectomized preparations (4), may depend on the fact that in the former preparations the small-size lVS neurons are subject to a prominent tonic inhibitory influence of the cerebellum, thus firing at a lower rate, in contrast to the large size lVS neurons, whose resting discharge was on the average almost comparable to that obtained after cerebellar ablation. In other words, the lower resting discharge rate of the small size lVS neurons in the control experiments with respect to the cerebellectomized preparations can be attributed to a greater inhibitibility which affects this population of neurons as a result of the tonic discharge of the related Purkinje cells.

If the distribution of the corticocerebellar inhibition within the lVN were largely uniform and independent of cell size, as postulated for the distribution of the excitatory synapses responsible for the resting discharge in the animal at rest, one would expect that large neurons with their small safety margin would be more sensitive to corticocerebellar inhibition than small neurons; the greater safety margin of the latter would, in fact, protect against corticocerebellar inhibition. This

situation would then be comparable to that postulated for the distribution of recurrent inhibition within a motor nucleus, which also could be independent of cell size (10, 30, 33, 34), an hypothesis supported by the fact that in experiments of ventral root stimulation large motoneurons appeared to be more susceptible to recurrent inhibition of stretch-evoked responses than smaller neurons (10,33; cf. 44). Unfortunately, electrical stimulation of ventral roots does not represent an appropriate tool to study the recurrent inhibitory circuitry, since antidromic stimulation affects Renshaw cells driven by both agonist and antagonist motoneurons. Thus mutual interaction among different populations of Renshaw cells can hardly be avoided (cf. 50).

Contrary to the hypothesis reported above, we found that in our experiments small-size lVS neurons were apparently more sensitive to corticocerebellar inhibition than large-size neurons. This finding is compatible with the results of recent experiments showing that Renshaw cells anatomically linked with extensor motoneurons, and ortodromically activated during vibration of the homonymous muscle, produced a more prominent recurrent inhibition on small motoneurons than on large motoneurons (61). Moreover, experiments of intracellular recording demonstrated that recurrent inhibitory postsynaptic potentials (IPSPs) were generally larger in slow-twitch than fast-twitch motoneurons (21).

The greater susceptibility of the small-size lVS neurons to corticocerebellar inhibition with respect to large-size neurons can in part be explained by difference in their input resistence, as shown for the α-motoneurons (8). There is in fact evidence that in small tonic motoneurons, that have higher input resistance than larger motoneurons (38), the same inhibitory input may elicit larger IPSPs (18, 41). An additional possibility, however, is that the Purkinje cells of the cerebellum make an higher number and density of inhibitory synaptic contacts on small-size lVS neurons which counteract the eccess of input excitation, in contrast to the large-size lVS neurons, where the number and density of inhibitory synaptic contacts are apparently unable in the animal at rest to overcome the weak excitatory input impinging upon them. Moreover, the inhibitory synapses could be located closer to the cell body and/or proximal dendrites of small-size than of large-size neurons.

It is of interest that in preparations with the cerebellum intact slow-conducting lVS neurons of comparable size, although firing at low rate, showed different discharge rates. This finding suggested that the corticocerebellar inhibitory influence on neuronal discharge did not equally affect the small-size lVS neurons, in analogy with the observation that the firing rate of small-size motoneurons can also be differentially affected by an inhibitory segmental input (28, 61). The observation, however, that slow-conducting lVS neurons of comparable size did not show the same discharge rates in cerebellectomized cats (4) indicates that even the excitatory input is not apparently homogeneously distributed among the same population of small-size lVS neurons. Similar results were also obtained at the level of small motoneurons of comparable size, which could have different firing rates in response to a given proprioceptive input (27, 61).

A final comment concerns the fact that, independently upon their responsiveness

to labyrinth stimulation, the small-size lVS neurons contribute not only to the postural activity in normal decerebrate cats but also, by increasing their discharge rate, to the prominent rigidity which occurs after partial cerebellectomy. Since the tonic contraction of limb extensors which occurs in decerebrate preparations involves the γ-system (γ-rigidity, 24), while that which occurs after cerebellectomy affects only the α-system (α-rigidity, 47), we may postulate that in the former preparation the moderate discharge of the small-size lVS neurons activates not only the small tonic α-motoneurons, but also the static γ-motoneurons innervating the limb extensors. After cerebellectomy, however, the increased discharge of the same population of lVS neurons would lead to recruitment of larger size α-motoneurons. Since these units are particularly effective in driving the recurrent inhibitory system, which acts preferentially on small-size motoneurons including those of the γ-type (cf. 50), one may understand why the increased rigidity which occurs after cerebellectomy is associated with a γ-paralysis (24).

Comparison of the results obtained in preparations with ipsilateral acute and chronic vestibular neurectomy.

A comparison between the effects of unilateral acute or chronic vestibular deafferentation on different size lVS neurons is justified only if the loss of descending influences from the forebrain following decerebration does not lead to decompensation of the postural deficits which occur after acute vestibular deafferentation. In our experiments, no postural asymmetry was observed when precollicular decerebration was performed in chronically operated, compensated animals (53). This finding is in agreement with the results of experiments showing that the tonic vestibular reflexes acting on the limbs were normal when the labyrinth of one site had been destroyed at least five weeks prior to decerebration (17). It appears, therefore, that decerebration does not interfere with the plastic events that are involved in the recovery and stabilization of a symmetric posture and normal labyrinth reflexes after unilateral chronic vestibular deafferentation.

Let us consider now the changes in the resting discharge of different size lVS neurons following ipsilateral acute and chronic vestibular deafferentation and relate these changes with the postural deficits as well as with the compensatory events occurring in these experiments (53). It is of interest that after aVN and cVN the resting discharge as well as the CV of the recorded lVS neurons were on the average comparable to the values obtained in the preparation with the vestibular nerves intact. However, while after aVN the proportion of regularly discharging units (characterized by a high firing rate and a CV between 0 and 0.35) was smaller than that of the irregular units (characterized by a low firing rate and a CV between 0.35 and 0.57), the situation reversed after cVN.

A further finding concerns the relation between resting discharge rate of lVS units and conduction velocity of the corresponding axons. In control experiments (52) units discharging more regularly at a high rate were characterized by a slower

conduction velocity of their axons (smaller neurons), while units discharging irregularly at a low rate by a faster conduction velocity (larger neurons). This relationship was lost after aVN, as a result of a depression in firing rate of the small neurons. However, the activity of the small neurons partly recovered after cVN, while large neurons tended to decrease their firing rate, so that the resting discharge rate was again negatively correlated with the axonal conduction velocity (53).

It has been postulated in the previous section that the excitatory input, which is responsible for the background discharge of the vestibular nuclear neurons in the animal at rest, makes an equal number of synaptic contacts on different-size lVS neurons projecting to the lower segments of the spinal cord. If we consider that the ipsilateral labyrinth input greatly contributes to the background of excitation, then the prominent decrease in resting discharge of small-size VS neurons, which occurs after ipsilateral aVN, would be in agreement with the «size principle» which states that the smaller the size of the neurons, the more effective is the corresponding input in exciting them (29, 30, 32, 33, 44, 58; cf. 31, 60). In agreement with the «size principle» is also the fact that after cVN, the recovery in resting discharge affects mainly the small-size neurons. Therefore, the postural hypotonia that occurs in the limbs ipsilateral to the side of the acute labyrinthine deafferentation depends mainly on disfacilitation of small-size lVS neurons; on the other hand, the compensation of the postural deficits occurring after cVN is, in part at least, due to partial recovery in the background discharge of these neurons.

In conclusion, it appears that among all the recorded lVS neurons, those of small-size are particularly involved in the static changes in posture which occur either after cerebellectomy or after aVN. The same neurons also intervene in the recovery process which occurs in chronically deafferented and compensated animals. It is worth mentioning that the LVN neurons contribute not only to the static,but also to the dynamic control of posture. A detailed analysis of experimental findings reported in a previous study (53) clearly indicates that in addition to the small-size lVS neurons, the large-size neurons are particularly influenced by sinusoidal stimulation of labyrinth receptors, thus intervening in the dynamic postural changes during the vestibulospinal reflexes.

SUMMARY

1. In addition to giant cells, originally described by Deiters, the lateral vestibular nucleus contains also medium- and small-size cells (5). The role that these neurons exert in the static control of posture has been investigated in precollicular decerebrate cats in which the resting discharge of spontaneously active vestibulospinal neurons projecting to lumbosacral segments of the spinal cord (lVS neurons) has been related to the cell size inferred on the basis of the conduction velocity of their axons.

2. In control experiments, the lVS neurons with slower axonal conduction velocity and, by inference, having thinner axons and smaller cell bodies differred from

those having faster conduction velocity by displaying a higher resting discharge rate and a relatively regular interspike interval distribution, i.e. a lower coefficient of variation (CV).

3. The resting discharge of the lVS neurons, which corresponded on the average to 24.5 ± 15.7, S.D. imp./sec, in control experiments, increased significantly to 44.1 ± 23.8, S.D. imp./sec after ablation of the cerebellar vermis and the fastigial nuclei, leading to a great increase in postural activity, while the proportion of regularly discharging units (with the lowest CV) increased. Moreover, the negative correlation between resting discharge of all the recorded lVS neurons and the conduction velocity of the corresponding axons, which was quite slight in the experiments with the cerebellum intact, greatly increased after partial cerebellectomy. This finding was due to a prominent increase in resting discharge of the small-size lVS neurons, while the discharge of the large-size lVS neurons was, on the average, comparable to that obtained in the controls. It appears, therefore, that the cerebellum exerts a prominent tonic inhibitory influence on the small-size lVS neurons, which are thus responsible for the great increase in decerebrate rigidity after cerebellectomy.

4. The resting discharge rate of the lVS neurons was not, on the average, greatly modified after ipsilateral acute (aVN) and chronic vestibular neurectomy (cVN) with respect to the controls. However, the proportion of regularly discharging units (with the lowest CV) decreased after aVN, but increased after cVN. The relation found in control experiments, i.e. the faster the conduction velocity of VS axon the lower was the unit discharge at rest, was lost after aVN, due to a decrease in resting discharge rate of the slow neurons. The mean discharge rate of these units, however, recovered after cVN, so that the negative correlation between resting discharge rate and axonal conduction velocity was reestablished. It appears, therefore, that the small-size VS neurons intervene in the development and compensation of the static postural deficits which occur following ipsilateral vestibular deafferentation.

5. We conclude that, in the animal at rest, the small-size lVS neurons are particularly sensitive to the tonic inhibitory input originating from the cerebellar vermis, as well as to the tonic excitatory input originating from the ipsilateral vestibular receptors, thus being involved in the static postural deficits which occur after partial cerebellectomy or after acute vestibular deafferentation. The same neurons are also involved in the compensation of the postural asymmetry following chronic vestibular deafferentation.

Acknowledgements. — The experiments reviewed in the present report were supported by the National Institute of Neurological and Communicative Disorders and Stroke Research Grant NS 07685 and by a grant from the Ministero dell'Università e della Ricerca Scientifica, Roma, Italy. The author wishes to acknowledge the assistance of Mrs. S. Pompeiano in the preparation of this manuscript.

REFERENCES

1. AKAIKE, T. Neuronal organization of the vestibulo-spinal system in the cat. *Brain Res.*, **259**: 217-227, 1983.
2. AKAIKE, T., FANARDJIAN, V. V., ITO, M., KUMADA, M. and NAKAJIAMA, H. Electrophysiological analysis of the vestibulospinal reflex pathway of rabbit. I. Classification of tract cells. *Exp. Brain Res.*, **17**: 477-496, 1973.
3. BARRET, J. N. and CRILL, W. E. Specific membrane resistance resistivity of dye-injected cat motoneurons. *Brain Res.*, **28**: 556-561, 1971.
4. BOYLE, R. and POMPEIANO, O. Relation between cell size and response characteristics of vestibulospinal neurons to labyrinth and neck inputs. *J. Neurosci.*, **1**: 1052-1066, 1981.
5. BRODAL, A. and POMPEIANO, O. The vestibular nuclei in the cat. *J. Anat., Lond.*, **91**: 438-454, 1957.
6. BURKE, R.E. On the central nervous system control of fast and slow twitch motor units. Pp. 69-94. In: Desmedt, J. E. (Ed.) *New Development in Electromyography and Clinical Neurophysiology*. Vol. 3, Karger, Basel, 1973.
7. BURKE, R. E. The role of synaptic organization in the control of motor unit activity during movement. Pp. 61-67. In: Granit, R. and Pompeiano, O. (Eds.) *Reflex Control of Posture and Movement*. Vol. 50. *Progress in Brain Research*. Elsevier, Amsterdam, 1979.
8. BURKE, R. E. and RUDOMIN, P. Spinal neurons and synapses. Pp. 877-944. In: Kandel, E. R. (Ed.) *Handbook of Physiology*. Sect. 1. *The Nervous System*. Vol. 1. *Cellular Biology of Neurons*. Part. 1. Bethesda. American Physiological Society, 1977.
9. CARLI, C., DIETE-SPIFF, K. and POMPEIANO, O. Responses of the muscle spindles and of the extrafusal fibres in an extensor muscle to stimulation of the lateral vestibular nucleus in the cat. *Arch. ital. Biol*, **105**: 209-242, 1967.
10. CLAMANN, H. P., GILLIES, I. D. and HENNEMAN, E. Effects of inhibitory inputs on critical firing level and rank order of motoneurons. *J. Neurophysiol.*, **37**: 1350-1360, 1974.
11. CORVAJA, N. and POMPEIANO, O. Identification of cerebellar corticovestibular neurons retrogradely labelled with horseradish peroxidase. *Neuroscience*, **4**: 507-515, 1979.
12. COULTER, J. D., MERGNER, T. and POMPEIANO, O. Effects of static tilt on cervical spinoreticular tract neurons. *J. Neurophysiol.*, **39**: 45-62, 1976.
13. CULLHEIM, S. Relations between cell body size, axon diameter and axon conduction velocity of cat sciatic α-motoneurons stained with horseradish peroxidase. *Neurosci. Lett.*, **8**: 17-20, 1978.
14. DEITERS, O. *Untersuchungen über Gehirn und Rüchenmark des Menschen und der Säugethiere*. Braunschweig, Friedrich Vieweg und Sohn, XVII-318 pp., 1865.
15. DENOTH, F., MAGHERINI, P. C., POMPEIANO, O. and STANOJEVIĆ, M. Responses of Purkinje cells on the cerebellar vermis to neck and macular vestibular inputs. *Pflügers Arch.*, **381**: 87-98, 1979.
16. DOW, R. S. and MORUZZI, G. *The Physiology and Pathology of the Cerebellum*. Minneapolis, The University of Minnesota Press, XVI-675 pp., 1958.
17. DUTIA, M. B. and ROSENBERG, J. R. The reappearance of nystagmus in chronic hemilabyrinthectomized cats following precollicular decerebration. *J. Physiol., Lond.*, **313**: 27P-28P, 1980.
18. ECCLES, J. C., ECCLES, R. M., IGGO, A. and ITO, M. Distribution of recurrent inhibition among motoneurons. *J. Physiol., Lond.*, **159**: 479-499, 1961.
19. FLOHR, H., BIENHOLD, H., ABELN, W. and MACSKOVICS, I. Concepts of vestibular compensation. Pp. 153-172. In: Flohr, H. and Precht, W. (Eds.) *Lesion-induced Neuronal Plasticity in Sensorimotor Systems*. Berlin, Springer Verlag, 1981.
20. FLUUR, E. Vestibular compensation after labyrinthine destruction. *Acta Otolaryngol., Stockh.*, **52**: 367-375, 1960.

21. FRIEDMAN, W. A., SYPERT, G. W., MUNSON, J. B. and FLESHMAN, J. W. Recurrent inhibition in type-identified motoneurons. *J. Neurophysiol.*, **46**: 1349-1359, 1981.
22. GACEK, R. R. The course and central termination of first-order neurons supplying vestibular end organs in the cat. *Acta Otolaryngol., Stockh., Suppl*, **254**: 1-66, 1969.
23. GOLDBERG, J. M. and FERNANDEZ, C. Conduction times and background discharge of vestibular afferents. *Brain Res.*, **122**: 545-550, 1977.
24. GRANIT, R. *The Basis of Motor Control.* London and New York, Academic Press, VI-346 pp., 1970.
25. GRILLNER, S., HONGO, T. and LUND, S. Descending monosynaptic and reflex control of γ-motoneurones. *Acta physiol. scand.*, **75**: 592-613, 1969.
26. GRILLNER, S., HONGO, T. and LUND, S. The vestibulospinal tract. Effects on alpha motoneurones in the lumbosacral cord in the cat. *Exp. Brain Res.*, **10**: 94-120, 1970.
27. HARRIS, D. A. and HENNEMAN, E. Identification of two species of alpha motoneurons in cat's plantaris pool. *J. Neurophysiol.*, **40**: 16-25, 1977.
28. HARRIS, D. A. and HENNEMAN, E. Different species of alpha motoneurons in same pool: Further evidence from effects of inhibition on their firing rates. *J. Neurophysiol.*, **42**: 927-935, 1979.
29. HENNEMAN, E. Relations between size and neurons and their susceptibility to discharge. *Science*, **126**: 1345-1346, 1957.
30. HENNEMAN, E., CLAMANN, H. P., GILLIES, J. D. and SKINNER, R. D. Rank order of motoneurons within a pool: Law of combination. *J. Neurophysiol.*, **37**: 1338-1349, 1974.
31. HENNEMAN, E. and MENDELL, L. M. Functional organization of motoneuron pool and its inputs. Pp. 423-507. In: Brooks, V. B. (Ed.) *Handbook of Physiology*. Sect. 1. *The Nervous System*. Vol. II. *Motor Control*. Part 1. Bethesda, American Physiological Society, 1981.
32. HENNEMAN, E., SOMJEN, G. and CARPENTER, D. O. Functional significance of cell size in spinal motoneurons. *J. Neurophysiol.*, **28**: 560-580, 1965.
33. HENNEMAN, E., SOMJEN, G. and CARPENTER, D. O. Excitability and inhibitibility of motoneurons of different sizes. *J. Neurophysiol.*, **28**: 599-620, 1965.
34. HULTBORN, H., PIERROT-DESEILLIGNY, E. and WIGSTRÖM, H. Distribution of recurrent inhibition within a motor nucleus. *Neurosci. Lett., Suppl.*, **1**: S97, 1978.
35. ITO, M. *The Cerebellum and Neural Control.* New York, Raven Press, XVII-580 pp., 1984.
36. ITO, M., HONGO, T. and OKADA, Y. Vestibular evoked postsynaptic potentials in Deiters' nucleus. *Exp. Brain Res.*, **7**: 214-230, 1969.
37. ITO, M., HONGO, T., YOSHIDA, M., OKADA, Y. and OBATA, K. Antidromic and transsynaptic activation of Deiters' neurones induced from the spinal cord. *Jap. J. Physiol.*, **14**: 638-658, 1964.
38. KERNELL, D. Input resistance, electrical excitability and size of ventral horn cells in cat spinal cord. *Science*, **152**: 1637-1640, 1966.
39. KERNELL, D. and ZWAAGSTRA, B. Input conductance, axonal conduction velocity and cell size among hindlimb motoneurones of the cat. *Brain Res.*, **204**: 311-326, 1981.
40. KUBIN, L., MAGHERINI, P. C., MANZONI, D. and POMPEIANO, O. Responses of lateral reticular neurons to sinusoidal stimulation of labyrinth receptors in decerebrate cat. *J. Neurophysiol.*, **44**: 922-936, 1980.
41. KUNO, M. Excitability following antidromic activation in spinal motoneurones supplying red muscles. *J. Physiol., Lond.*, **149**: 374-393, 1950.
42. LACOUR, M. and XERRI, C. Vestibular compensation: new perspectives. Pp. 240-253. In: Flohr, H. and Precht, W. (Eds.) *Lesion-induced Neuronal Plasticity in Sensorimotor Systems*. Berlin, Springer-Verlag. 1981.
43. LUND, S. and POMPEIANO, O. Monosynaptic excitation of alpha-motoneurones from supraspinal structures in the cat. *Acta Physiol. Scand.*, **73**: 1-21, 1968.

44. MENDELL, L. M. AND HENNEMAN, E. Terminals of single Ia fibers: location, density and distribution within a pool of 300 homonymous motoneurons. *J. Neurophysiol.*, **34**: 171-187, 1971.
45. MONAKOW, C. V. Experimenteller Beitrag zur Kenntnis des Corpus restiforme, des «äusseren Acusticuskerns» und deren Beziehungen zum Rückenmark. *Arch. Psychiatr. Nervenkr.*, **14**: 1-16, 1983.
46. PETERSON, B. W. Distribution of neural responses to tilting within vestibular nuclei of the cat. *J. Neurophysiol.*, **33**: 750-767, 1970.
47. POMPEIANO, O. Alpha types of «release» studied in tension-extension diagrams from cat's forelimb triceps muscle. *Arch. ital. Biol.*, **98**: 92-117, 1960.
48. POMPEIANO, O. Vestibulospinal relations: vestibular influences on gamma motoneurons and primary afferents. Pp. 197-232. In: Brodal, A. and Pompeiano, O. (Eds.) *Basic Aspects of Central Vestibular Mechanisms*. Vol. 37. *Progress in Brain Research*. Amsterdam, Elsevier, 1972.
49. POMPEIANO, O. Neck and macular labyrinthine influences on the cervical spinoreticulocerebellar pathway. Pp. 501-514. In: Granit, R. and Pompeiano, O. (Eds.) *Reflex Control of Posture and Movement*. Vol. 50. *Progress in Brain Research*. Amsterdam, Elsevier, 1979.
50. POMPEIANO, O. Recurrent inhibition. Pp. 461-557. In: Davidoff, R. A. (Ed.) *Handbook of the Spinal Cord*. Vols. 2 and 3. *Anatomy and Physiology*. New York, Basel, Marcell Decker, 1984.
51. POMPEIANO, O. and BRODAL, A. The origin of vestibulospinal fibres in the cat. An experimental study with comments on the descending medial longitudinal fasciculus. *Arch. ital. Biol.*, **95**: 166-195, 1957.
52. POMPEIANO, O., MANZONI, D., MARCHAND, A. R. and STAMPACCHIA, G. Effects of roll tilt of the animal and neck rotation on different size vestibulospinal neurons in decerebrate cats with the cerebellum intact. *Pflügers Arch.*, **409**: 24-38, 1987.
53. POMPEIANO, O., XERRI, C., GIANNI, S. and MANZONI, D. Central compensation of vestibular deficits. II. Influences of roll tilt on different-size lateral vestibular neurons after ipsilateral labyrinth deafferentation. *J. Neurophysiol.*, **52**; 18-38, 1984.
54. PRECHT, W. Characteristics of vestibular neurons after acute and chronic labyrinthine destruction. Pp. 451-462. In: Kornhuber, H. H. (Ed.) *Handbook of Sensory Physiology*. Vol. VI/2. *Vestibular System*. Part 2. *Psychophysics, Applied Aspects and General Interpretations*. Berlin, Springer Verlag, 1974.
55. SCHAEFER, K.-P. and MEYER, D. L. Compensation of vestibular lesions. Pp. 463-490. In: Kornhuber, H. H. (Ed.) *Handbook of Sensory Physiology*. Vol. VI/2. *Vestibular System*. Part 2. *Psychophysics, Applied Aspects and General Interpretations*. Berlin, Springer-Verlag. 1974.
56. SCHAEFER, K.-P. and MEYER, D. L. Aspects of vestibular compensation in guinea pigs. Pp. 197-207. In: Flohr, H. and Precht, W. (Eds.) *Lesion-induced Neuronal Plasticity in Sensorimotor Systems*. Berlin, Springer-Verlag, 1981.
57. SIEGBORN, J. and GRANT, G. Brain stem projections of different branches of the vestibular nerve. An experimental study by transganglionic transport of horseradish peroxidase in the cat. I. The horizontal ampullar and utricular nerves. *Arch. ital. Biol.*, **121**: 237-248, 1983.
58. SOMJEN, G., CARPENTER, D. O. and HENNEMAN, E. Responses of motoneurons of different sizes to graded stimulation of supraspinal centers of the brain. *J. Neurophysiol.*, **28**: 958-965, 1965.
59. STEIN, B. M. and CARPENTER, M. B. Central projections of portions of the vestibular ganglia innervating specific parts of the labyrinth of the rhesus monkey. *Amer. J. Anat.*, **120**: 281-318, 1967.
60. STUART, D. G. and ENOKA, R. M. Motoneurons, motor units, and the size principle.

Pp. 471-517. In: Rosenberg, R. N. (Ed.) *The Clinical Neurosciences*. Sect. 5. *Neurobiology* (Willis W. D., Ed.). New York, Churchill Livingstone, 1983.

61. WAND, P., POMPEIANO, O. and FAYEIN, N. A. Impulse decoding process in extensor motoneurones of different size during the vibration reflex. *Arch. ital. Biol.*, **118**: 205-242, 1980.
62. WILSON, J., KATO, M., PETERSON, B. W. and WYLIE, R. M. A single-unit analysis of the organization of Deiters' nucleus. *J. Neurophysiol.*, **30**: 603-619, 1967.
63. WILSON, V. J. and MELVILL-JONES, G. *Mammalian Vestibular Physiology*, New York, Plenum Press, XI-365 pp., 1979.
64. WILSON, V. J. and YOSHIDA, M. Comparison of effects of stimulation of Deiters' nucleus and medial longitudinal fasciculus on neck, forelimb and hindlimb motoneurons. *J. Neurophysiol.*, **32**: 743-758, 1969.