maintained. Tracings *3*, *4*, *5* and *6* illustrate that the occurrence of spontaneous activity of the collicular cell is associated with a decrease in activity of the geniculate neuron. In tracings *4* and *6* (Figure 1, *B*, *C*), it is shown that occasionally the collicular neuron presented a spike at 70 msec which coincided with a delayed discharge (tracing *3*) or a complete absence of activity (tracing *5*) in the geniculate neuron.

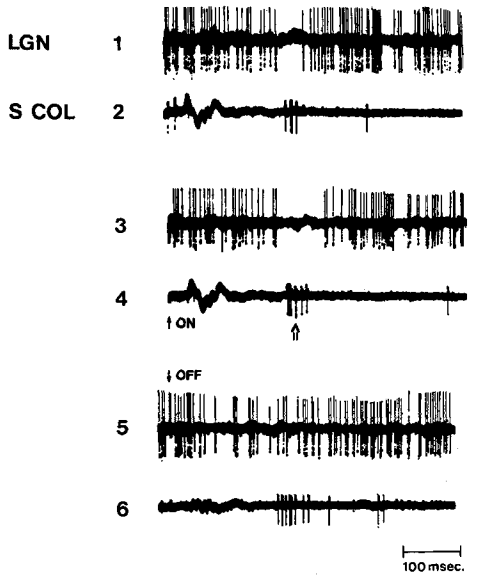

Fig. 2. Simultaneous recordings from L. G. N., P cell (tracings *1*, *3*, *5*) and S. C. (tracings *2*, *4*, 6). Following an 'ON' step, the geniculate neuron presented a transient interruption of its activity. The burst of the collicular cell occurred while the geniculate cell was prevented from firing. All tracings were obtained from 4 to 5 superimposed sweeps. The polarity of the collicular evoked potentials to the 'ON' stimulus (tracings *2*, *4*) and the absence of a distinguishable 'OFF' response (tracing *6*) confirm that the collicular unit is located very superficially in the S.C.

The example in Figure 2 provides additional insight into the mechanism underlying this alternating type of activity between the L.G.N. and S.C. The geniculate unit shown in Figure 2 (tracings *1*, *3*, *5*) responded rather weakly to 'ON' and 'OFF' stimuli but was capable of firing at any moment for 1 sec following the 'ON' stimulus, except for a short period of time between 195 and 240 msec. In comparison, the collicular cell (Figure 2, tracings *2–4*), recorded simultaneously, exhibited a very slow rate of firing. Its highest period of excitation occurred with a long latency (195 msec) and thus, precisely during the silent period of the geniculate neuron. No such alternating effects were observed from the opposite (OFF) stimulus (tracings *5* and *6*).

Discussion. Application of an 'OFF' stimulus shows that the cessation of activity of the geniculate cell is not associated with an increase of excitability of a collicular cell and vice versa. Thus, it seems that direct mutually inhibitory relationships between the L.G.N. and S.C. cannot be involved, although such connections may exist[18]. Furthermore, electrical stimulation through the recording electrodes fails to elicited a response in the complementary unit. The 2 remaining most likely sites to which these alternating effects may be attributed are: the retina and the visual cortex. Although the retina cannot be disregarded, it is interesting to point out in this respect that the response in Figure 1 (tracings *6* and *8*) as well as Figure 2 are not typical of retinal ganglion cells, and that abolition of cortical function with 3 *M* KCl application results in a disruption of the normal firing pattern of geniculate neurons[19]. These results suggest a possible involvement of the superior colliculus in scanning processes of the eye[20] which mediate pattern perception.

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Vestibular units during decompensation[1]

G. B. Azzena, O. Mameli and E. Tolu

Institute of Human Physiology, University of Sassari, via Muroni 23, I-07100 Sassari (Italy), 14 July 1976

Summary. Typical modifications of the unitary discharge of vestibular units have been recorded following the transection of the spinal cord of hemilabyrinthectomized and compensated guinea-pigs. These results support the concept that the spinal cord is essential in the compensation of the symptoms resulting from a lesion of one labyrinth.

Previous research[2,3] has demonstrated that the spinal cord is involved in the mechanism of compensation of the motor deficits resulting from a previous hemilabyrinthectomy. In fact, it has been observed that spinal cord transection at the mid-thoracic level, after compensation of the symptoms produced by lesion of one labyrinth, will be followed by the reappearance of some of these same symptoms. This new stage has been identified as the decompensation period. Furthermore, cutting the spinal afferent pathways in a hemilabyrinthectomized and compensated animal provokes clear-cut modifications of the field potentials generated by electrical stimulation of the ampullar receptors of the intact side and recorded from the vestibular nuclear complexes of both sides. The effects induced by the transection of the cord consist of facilitation of N_1 or N_1–N_2 waves[4] recorded from the vestibular nuclei of the intact side and inhibition of the evoked potentials in the vestibular nuclei of the deafferented side. Therefore, it is evident that the compensation of the vestibular deficits is controlled by the output of the vestibular nuclei, which in turn is dependent upon the influence of the spinal cord.

The purpose here is to describe the results of series of experiments carried out with the aim of analyzing the behavior of the vestibular units during the compensated and decompensated stages. Ether anesthesia of 75

guinea-pigs, weighing 450–600 g, was followed by hemilabyrinthectomy at the left side using the method of Simonelli[5] which consists of injecting of a 50% solution of oil and chloroform into the middle ear. After recovery from anesthesia, all the animals displayed the typical symptoms resulting from the lesion of one labyrinth[2,6]. Within 10–40 days following the vestibular deafferentation, the animals were anesthetized by i.m. administration of 10 mg/kg of ketamine (Ketalar, Park-Davis) and 10 mg/kg of diazepam (Valium, Roche). Following tracheotomy, each animal was fixed in the stereotaxic apparatus (AB Transvertex), paralyzed with Flaxedil and artificially ventilated. Subsequent craniotomy and a laminectomy between C_3–C_4 and T_6–T_7 were performed in order to expose the cerebellum and the spinal cord, which were thereafter protected with warm mineral oil. The unitary discharges of vestibular units of both sides were recorded by tungsten microelectrodes (tip diameter 3–5 μm; 700–900 kΩ) introduced through the cerebellum into the vestibular nuclei by means of an electronic microdrive. Signals from conventional preamplifiers were recorded on magnetic tape for later analysis of the discharge frequency with a Schmitt trigger and a 4672 Ortec instantaneous frequency/timer meter. Only negative or negative-positive spikes of units, identified by antidromic stimulation at the C_3–C_4 level, were recorded. Recordings were performed before and after transverse section of spinal cord at the mid-thoracic level. The recorded site was marked for the subsequent histological control by passing current to produce a lesion (figure 1). The present report is based upon the analysis of deitersian units of both sides; 36 were recorded from the right and 30 from the left nucleus. In the compensated stage, the units showed a tonic discharge ranging from 5 to 35 impulses/sec. In only one case was the pattern phasic. The transection of the spinal cord induced a modification

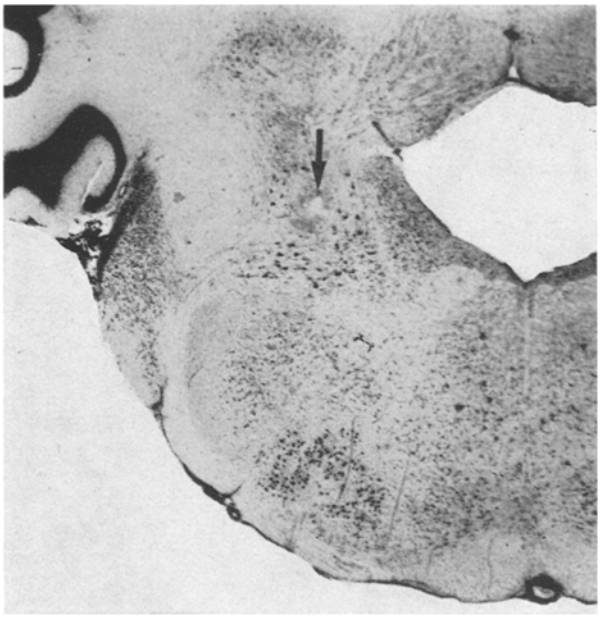

Fig. 1. Transverse section of the brain stem of guinea-pig No. 17; the arrow points to an electrolytic lesion on the left Deiters nucleus.

1 Supported by a grant of CNR.

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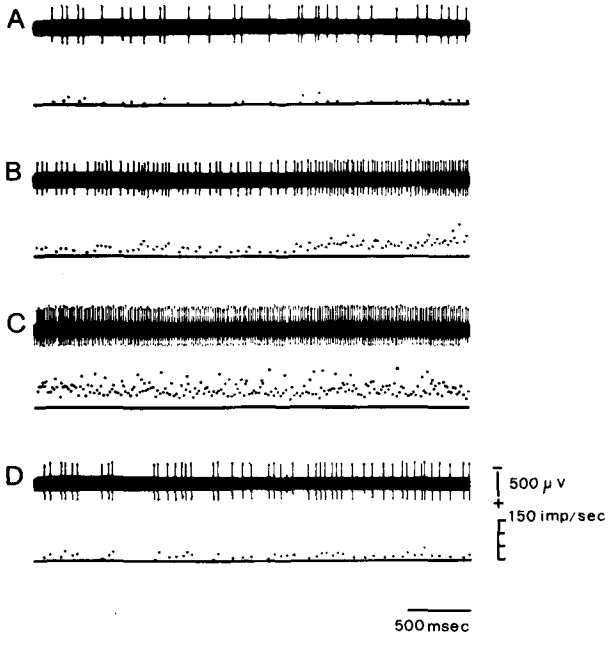

Fig. 2. Electrical activity in the right lateral vestibular nucleus; top trace: unitary discharge; bottom trace: instantaneous discharge frequency. A: in the compensated stage; B, C and D: 10 sec, 7 and 12 min after the transection of the spinal cord.

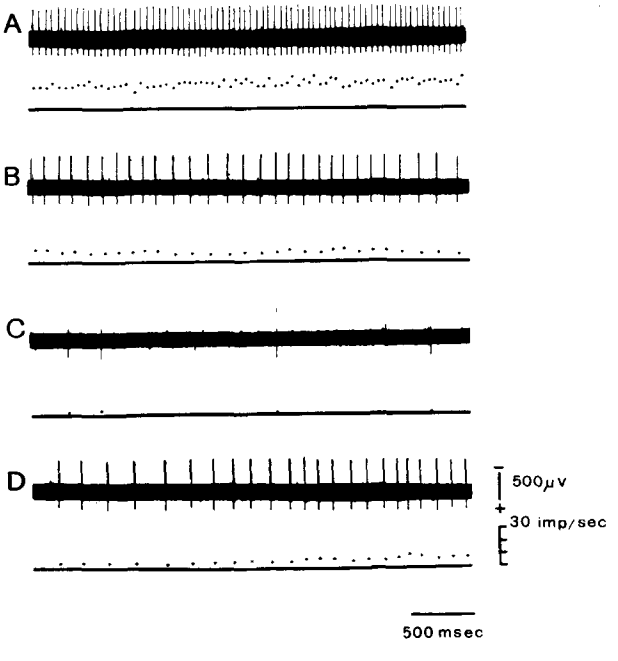

Fig. 3. Electrical activity in the left lateral vestibular nucleus; top trace: unitary discharge; bottom trace: instantaneous frequency. A: in the compensated stage. B, C and D: respectively, 10 sec, 20 and 40 min after the transverse section of the spinal cord.

of the unitary discharge in 61.1% (22 units) of the cells recorded from the right lateral vestibular nucleus and in the 56.7% (17 units) of those of the opposite side. Figures 2 and 3 show the 2 patterns of activity which were prevalently recorded from the right or left Deiters nuclei. Figure 2 A–D illustrate the facilitation of a right deitersian unit following the surgical interruption of the spinal cord in a hemilabyrinthectomized and compensated animal, while figure 3 A–D represent the inhibition induced by spinal cord section on a left deitersian unit of another hemilabyrinthectomized and compensated animal. In fact, the transection of the spinal cord acts in a different manner upon the cells of both Deiters nuclei. Figure 4 displays the fraction of the units of both sides responding to the cord transection with activation or inhibition. Regarding the 22 cells of the right side responding during decompensation, it can be seen that activation represents 64.7% of these cells and inhibition 35.3%. An opposite pattern was observed for the 17 left deitersian units which showed activation in 31.2% and inhibition in 68.8% of the cases. As far as the activation-inhibition pattern is concerned, it must be pointed out that these 2 types of responses show different characteristics if their time course is taken into account. As a matter of fact, the inhibition is a long-lasting phenomenon having a duration of 30–40 min or more. Activation, on the other hand, usually disappears within 10–15 min.

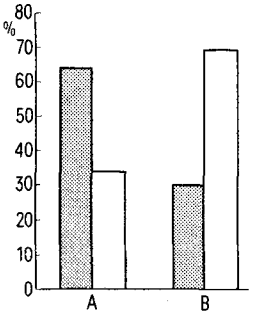

Fig. 4. Fraction of the units of the right (A) and left (B) lateral vestibular nucleus with activation (dotted column) or inhibition (open column), following the spinalization.

Data of this present investigation show for the first time that the transection of the spinal cord in a hemilabyrinthectomized and compensated animal induces characteristic modifications of the unitary discharge of vestibular units. Thus, the concept that the inputs from the spinal cord are essential for the correct balance of the vestibular output of the 2 sides is strengthened[2,3]. Furthermore, in the present research it has been observed that the interruption of spinal afferents acts in a different manner upon the vestibular cells of the 2 sides: the predominant effect upon the vestibular cells of the intact side is activation while cells of the differented side are inhibited. The data of the present experiments, compared with the results of previous research on the effects of an acute lesion of one labyrinth on the unitary discharge of vestibular units[7,8], demonstrate conclusively that the transection of the spinal cord reestablishes the same pattern of activity of the vestibular units as during the acute period following the lesion of the labyrinth.

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Stimulation of glucagon and inhibition of insulin secretion evoked from carotid baroreceptors

J. Järhult and J. J. Holst

Department of Physiology and Biophysics, Sölvegatan 19, 223 62 Lund (Sweden), and Department of Clinical Chemistry, Bispebjerg Hospital, Copenhagen (Denmark), 12 July 1976

Summary. The influence from carotid baroreceptors on portal immuno-reactive glucagon and insulin levels and on arterial plasma glucose concentration was studied in vagotomized cats by sectioning of the sinus nerves. Such a complete elimination of the afferent baroreceptor discharge caused a prompt and pronounced increase in the glucose and glucagon levels, whereas the insulin concentration significantly decreased. The role of vascular baroreceptors in the hyperglycemic response to hemorrhage is discussed.

Marked changes of the insulin and glucagon secretion occur in a variety of physiological and pathophysiological stress situations such as exercise, starvation, trauma and hemorrhage[1–6]. Several studies have shown that the altered pancreatic hormone release in these situations is caused mainly by adrenergic stimuli acting on the α- and β-cells[7,8], but hitherto there is little information available about which type of receptor that initiates these adrenergic adjustments of the endocrine pancreas. However, in a recent study we found that a decreased activity of the arterial baroreceptors caused a clearcut hyperglycemia, whereas the influence from arterial chemoreceptors on the plasma glucose concentration was much less pronounced (Järhult, Holmberg and Lundvall, in press). The present experiments were therefore performed to investigate whether arterial baroreceptors can influence also the endocrine pancreas and, if so, if the pattern of secretory changes is similar to that seen in hemorrhage.

Material and methods. 3 cats were anesthetized i.v. with chloralose (50 mg/kg) and urethane (50 mg/kg) after induction with ether. A tracheal cannula was inserted and the vagus and sinus nerves were freed bilaterally in the neck. The abdomen was opened with a midline incision. After heparinization (1000 IU/kg b.wt.), a polyethylene catheter was inserted into a small jejunal vein and advanced until its tip was placed in the portal vein. From this catheter, blood samples were withdrawn for determination of plasma immuno-reactive glucagon and insulin concentration. Arterial blood samples were taken from the cannulated right axillary artery. Mean arterial blood pressure was recorded with a Statham P23 AC transducer connected to the axillary artery. Heart rate was measured with a Grass tachograph triggered by the systolic pressure wave.

Arterial plasma glucose concentration was measured with the conventional glucose-oxidase method. Portal plasma glucagon concentration was determined with a radioimmunoassay-technique described recently[9], using an antiserum which is highly specific for pancreatic glucagon. Portal plasma insulin concentration was mea-