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.

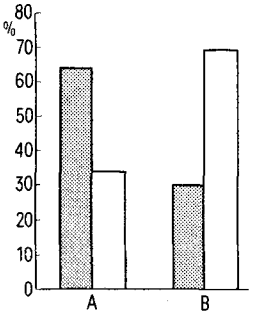

Fig. 4. Fraction of the units of the right (A) and left (B) lateral vestibular nucleus with activation (dotted column) or inhibition (open column), following the spinalization.

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.

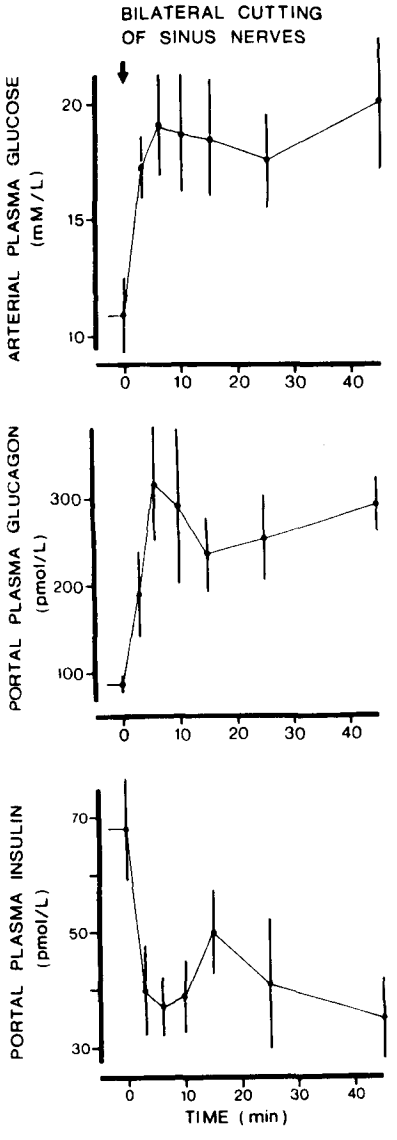
Marked changes of the insulin and glucagon secretion occur in a variety of physiological and pathophysiological stress situations such as exercise, starvation, trauma and hemorrhage[1–6]. Several studies have shown that the altered pancreatic hormone release in these situations is caused mainly by adrenergic 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-

sured according to Albano et al.[10]. Our results with this technique are similar to those obtained with a different technique previously reported from this laboratory[11].

Results. The figure shows the arterial plasma glucose concentration as well as the portal plasma immunoreactive glucagon (IRG) and insulin (IRI) concentration before and after bilateral sectioning of the sinus nerves in cats, which had been vagotomized about 30 min earlier. Such a sinus nerve section leads to a complete elimination of the normal inhibitory discharge from the carotid baroreceptors, thereby stimulating a blood pressure drop in the carotid arteries down to 40–60 mm Hg. It is clearly seen from the figure that such a baroreceptor interference caused marked alterations in the plasma levels of glucose, IRG and IRI. Thus, the glucose concentration was almost doubled, IRG increased 3–4 times, and IRI decreased to about half the control value. All these changes developed very rapidly and were maintained throughout the 45 min of observation. Concomitantly, there occurred large increases in heart rate and in mean arterial blood pressure, indicating a rise in the activity of the sympatho-adrenal system.



Changes of arterial plasma glucose concentration and of portal plasma glucagon and insulin concentrations evoked by bilateral cutting of the sinus nerves in vagotomized cats. Mean values ± S. E. M. from 3 experiments are given. Such nerve sections cause complete elimination of the normal inhibitory discharge from the carotid baroreceptors, thereby mimicking the effects of carotid artery hypotension.

Discussion. The present experimental data clearly demonstrate that drastic adjustments of the pancreatic glucagon and insulin release can be initiated from alterations in the carotid baroreceptor discharge. It is evident that the evoked hormonal changes significantly must have contributed to the concomitantly observed hyperglycemia. It is well known that the effects of adrenergic stimuli on endocrine pancreas consist of a reduction in the insulin release and an increase in the glucagon release[7,12]. The presence of a similar secretory pattern in the described experiments therefore strongly suggests that the baroreceptor influence on the α- and β-cells is mediated by adrenergic mechanisms. It remains to be shown whether these adrenergic stimuli reach the pancreas via adrenal catecholamines, or whether they are linked to the recently described direct neurogenic pathways[13,14].

Numerous studies have shown that hemorrhage leads to a hyperglycemic state both in animals and man[15]. The benefit of this hemorrhagic hyperglycemia is not only to supply the ischemic tissues with energy substrate, but also to serve as an important expander of the plasma volume by causing a transcapillary osmotic absorption of fluid from the extravascular to the intravascular compartment[16]. The hyperglycemia is mainly due to an increased release of glucose from the liver[17], in turn caused by a multifactorial neuro-hormonal influence on the hepatic glycogenolysis and gluconeogenesis[15]. The pancreatic hormones no dout play essential roles here, since several investigators have observed a hyperglucagonemia and hypoinsulinemia to develop during bleeding[4,5,18,19], i.e. the same hormonal adjustments as reported in the present study. Since, in addition, the activity of the arterial baroreceptors is decreased (for abolished) during hemorrhage[20], it might be concluded that the carotid baroreceptors help to initiate the adjustments of the glucagon and insulin secretion during hemorrhagic hypotension.

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