CHANGES IN THE PANCREAS FOLLOWING REPEATED EXPOSURE TO TRANSVERSE RADIAL ACCELERATION

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The histological and morphometric study of the pancreas in dogs exposed repeatedly to transverse radial acceleration revealed changes indicative of the development of pancreatic atrophy.

KEY WORDS: *pancreas; atrophy; overloading.*

An important role in the complex processes of adaptation to external environmental factors such as overloading is played by the endocrine-glandular apparatus of the digestive glands and, in particular, of the pancreas [1, 2, 10, 11]. The available information on this problem is mainly concerned with the physiology of the pancreas under these conditions [2, 8, 11]. A few morphological studies [4, 5] have been made of the character of the structural changes in the gland after a single exposure to overloading.

No data could be found in the literature on the character of the morphological changes in the pancreas during repeated exposure to transverse acceleration. Such an investigation is important because the pancreas plays a definite role in the maintenance of homeostasis of carbohydrate and lipid metabolism [15].

EXPERIMENTAL METHOD

The pancreas of male dogs exposed to acceleration with an intensity of 8 units, produced by spinning the animals on a fixed-speed centrifuge with a radius of 4.2 m, was investigated. The duration of exposure was 3 min and the overloading acted in the chest-spine direction. The animals (four dogs) of group 1 were exposed to acceleration once, those of group 2 (nine dogs) repeatedly (12-14 times) at intervals of 30 days. Material was taken under superficial ether anesthesia 30 days after the final exposure. Two dogs from group 1 were killed on the day of the experiment to analyze the acute changes in the pancreas and two dogs from group 2 were killed 1 year after the final exposure. The pancreas of intact animals served as the control. A series of survey and histochemical methods was used: staining with hematoxylin-eosin, by Van Gieson's method with counterstaining for elastic tissue, by Mallory's method in Heidenhain's modification, and silver impregnation by the method of Gordon and Sweets. Lipids were demonstrated by Goldman's method and with Sudan black by the method of Lison and Danielli. Fuchsinophilic granules in the β-cells of the islets of Langerhans were revealed with aldehyde-fuchsin by Gomori's method in Maiorova's modification. For the morphometric investigations the sections were drawn by the RA-6 apparatus and the parameters to be studied determined by planimetry. The ratio between the exocrine and endocrine parts of the gland and the dimensions of the islets were determined and the number of islets per unit area calculated. The numerical results were subjected to statistical analysis.

EXPERIMENTAL RESULTS

After a single exposure to acceleration two types of changes were observed in the pancreas on the day of the experiment. The first type consisted of the almost total disappear-

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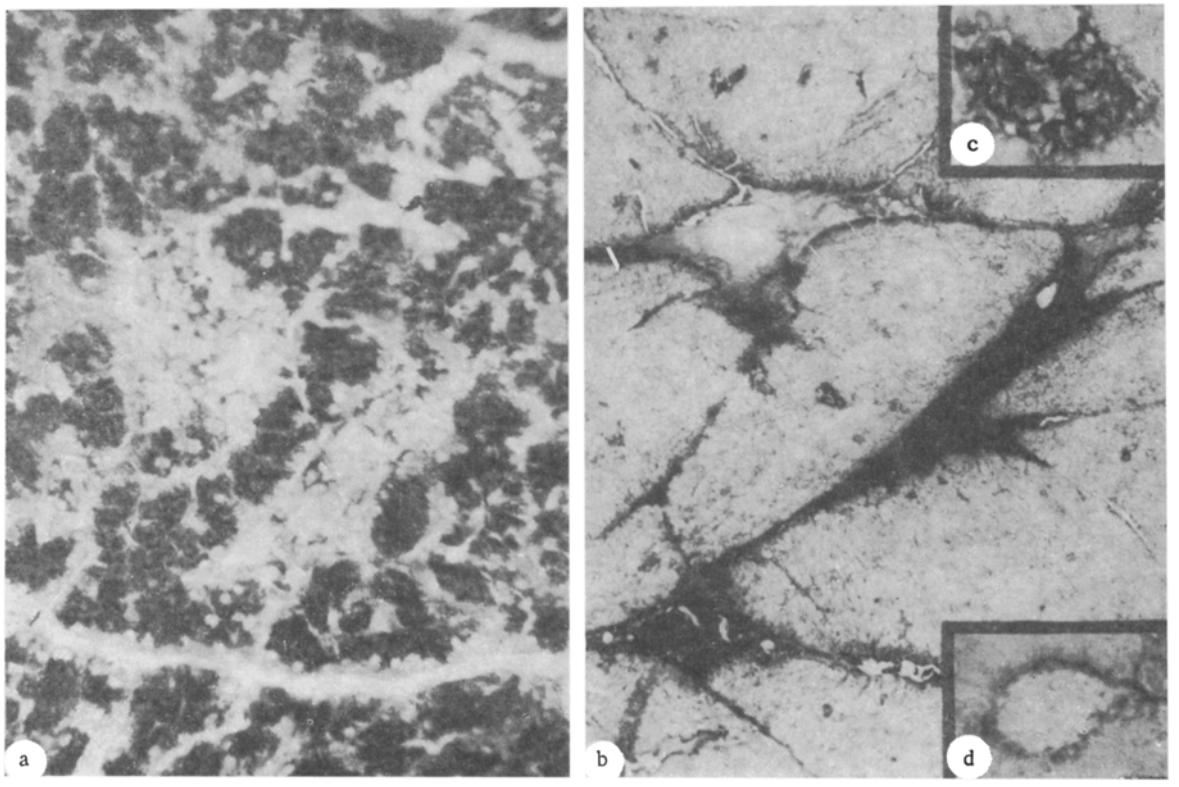

Fig. 1. Structure of the dog pancreas after a single exposure to overloading: a) normal dog pancreas: moderate number of cells containing fuchsinophilic granules in islets of Langerhans; acinar cells filled with zymogen granules (stained with aldehyde–fuchsin by Gomori's method; 400×); b) marked edema of interlobular connective tissue; absence of acinar cells, islet cells contain many fuchsinophilic granules (stained with aldehyde–fuchsin by Gomori's method; 100×); c) detail of same preparation: accumulation of fuchsinophilic granules in islet cells; emptying of acinar cells (stained with aldehyde–fuchsin by Gomori's method; 400×); d) detail of same preparation: destructive changes in endocrine part of pancreas; islet of Langerhans with signs of cell destruction and almost total disappearance of fuchsinophilic granules from cells (stained with aldehyde–fuchsin by Gomori's method; 400×).

ance of zymogen granules from the acinar cells and a considerable increase in specific fuchsinophilic granules in the β cells of the islets of Langerhans (Fig. 1a-c). These changes were diffuse in character and evidently represent the stereotyped response of the gland to stressors [1]. Focal changes also occurred. In these areas the normal structure of the acini in the parenchyma of the gland was lost and dystrophic and necrobiotic changes were present; the cytoplasm of the cells showed evidence of cloudy swelling and vacuolar degeneration. The lumen of the acini and of the intralobular ducts was dilated and contained no secretion. The interlobular ducts showed features of dystonia and invagination of the walls. Together with degenerative changes, degranulation of the cytoplasm of the endocrine cells was observed, sometimes amounting to total disappearance of the granules (Fig. 1d). The blood vessels were grossly dilated and marked edema of the interlobular connective tissue was present (Fig. 1b). The interlobular arteries were surrounded by zones of extravasation. Similar changes have also been described in other organs following exposure to overloading under the name of plasmorrhagia of the interlobular arteries [7].

After 30 days (toward the time of the second exposure to acceleration) the structure of the pancreas was not fully restored. Evidence of perivascular sclerosis was present in areas where extravasation had been present previously.

Repeated exposure to overloading led to an increase in the density of the gland tissue and a decrease in its size. Signs of well marked annular sclerosis and of atrophy of the parenchyma were found in the gland (Fig. 2a). One factor in the development of sclerosis

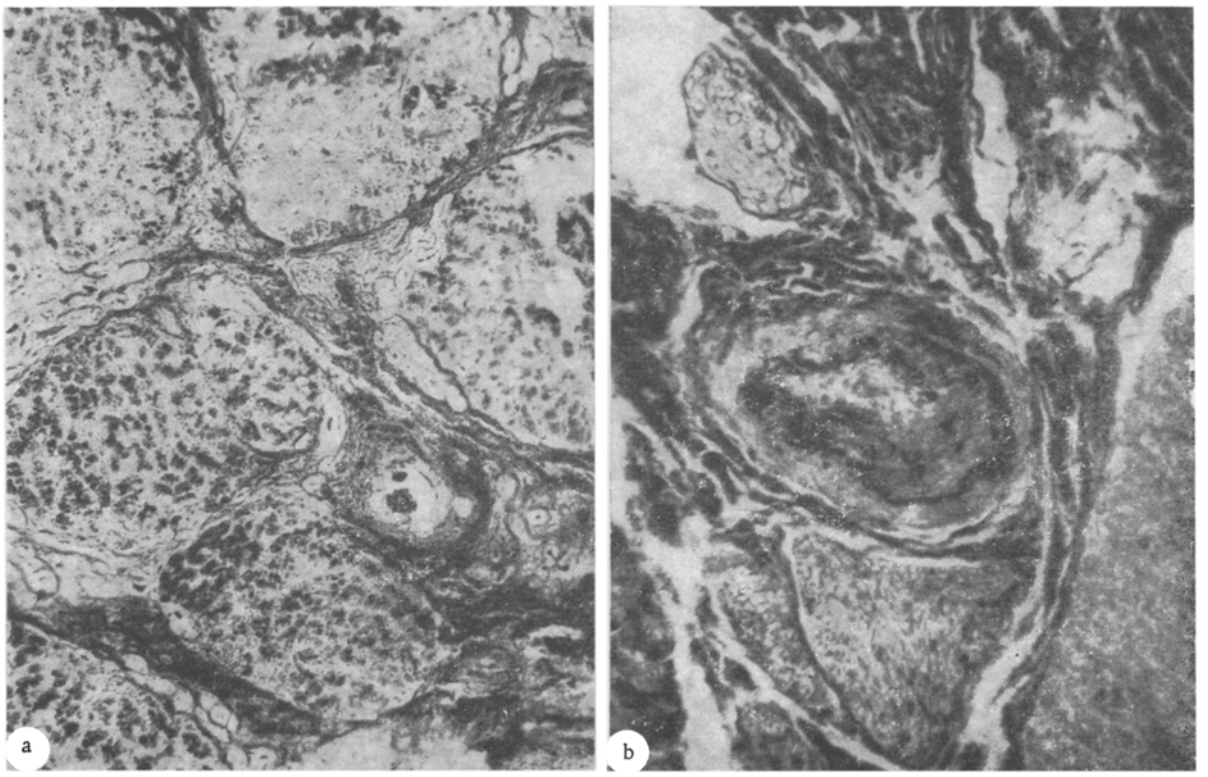

Fig. 2. Structure of dog pancreas during repeated exposure to overloading: a) annular sclerosis of pancreas (stained by Mallory's method in Heidenhain's modification; 100×); b) recalibration of artery in pancreas (stained by Van Gieson's method; 200×).

Table 1. Results of Morphometric Study of Dog Pancreas after Exposure to Radial Acceleration

Conditions	Number of animals	Number of islets in 1 mm^2 tissue	Area of islets, μ^2	Ratio of endocrine and exocrine part of pancreas
Control	6	97,8±5,70	1133,28±110,41	1 : 8,02
Exposure to acceleration:				
once on day of experiment	2	100,1±3,47 $P>0,5$	1105,20±141,78 $P>0,5$	1 : 8,04
30 days later	2	175,3±6,66 $P<0,001$	1527,36±116,17 $P<0,05$	1 : 2,73
repeatedly:				
30 days after last exposure	7	154,8±6,30 $P<0,001$	1482,08±59,50 $P<0,05$	1 : 3,35
1 year after last exposure	2	126,7±4,59 $P<0,01$	1439,78±62,86 $P<0,05$	1 : 4,48

could be hypoxia, which is present in the tissues during exposure to acceleration [7, 12, 13], and another could be the direct harmful action of this stimulus on connective tissue [6]. The foci of sclerosis were most extensive around areas of lymphostasis. The reason was evidently that during lymphostasis the main function of the lymphatic system, namely absorption of products of protein metabolism and of acid glycosaminoglycans, which accumulate to excess in hypoxia, is interrupted. Retention of these products contributes to the development of sclerosis, more especially around the lymphatics [3, 9].

The development of sclerotic changes in the pancreas was accompanied by reorganization of its lymphatic and blood circulation. Clear signs of lymphostasis and reduction of the blood circulation were found, in the form of recalibration of the arterial trunks (Fig. 2b); this is evidently a universal mechanism of adaptation of the blood stream to changes in the blood inflow required by the organ during atrophy [3, 14].

Less marked atrophic changes were found in the endocrine part of the pancreas. They were observed mainly in the large islets and consisted of coarsening of their stroma, thickening of the capsule, and the almost complete disapparance of fuchsinophilic granules from the β cells. The smaller islets were unchanged. Changes also were observed in the ratio between the endocrine and exocrine parts of the pancreas (Table 1).

Similar structural changes were found in the pancreas of animals during the after-period (1 year after the final exposure); the severity of the atrophic changes in this case was greater.

The changes found are evidently the structural basis of the disturbance of pancreatic function resulting from exposure to transverse acceleration. It seems likely that the principal role in the structural reorganization of the pancreas is played by repeated hemodynamic and dystrophic changes which are cumulative during repeated exposure to the unfavorable factors.

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