Vascular and Ductal Patterns of Pancreas by Microradiography and Their Relation to Lesions of Pancreatitis

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Histopathological findings of pancreas freshly obtained from 108 autopsy cases were analyzed and individual lesions were correlated with vascular and ductal patterns revealed by microradiography. As the intralobular artery was demonstrated as a terminal artery, its functional and/or organic occlusion was expected to produce focal atrophy, parenchymal necrosis and fibrosis of lobular unit. Atrophy of acinar cells leading to replacement by isolated fat cells developed presumably from disturbance of systemic arterial circulation. Venous stasis seemed to be responsible for the formation of pseudolobules.

Organic and/or functional obstructions in any part of the pancreatic duct caused various lesions of pancreatitis. Periductal location of focal necrosis and cell infiltration was regarded as a result of pancreatic juice leakage. Because the peripheral range of the pancreas was abundant in ductules, fat necrosis and fibrosis probably of ductal origin preferred the periphery.

It was presumed that the ductal and vascular factors could interact each other in producing pancreatitis and both might even precipitate serious damages of the pancreas.

Concerning the etiology of pancreatitis, various conditions have been taken into consideration, e.g., bacterial and viral infections, obstructions of the pancreatic duct, metabolic disorders, circulatory disturbances and allergy. However, there are few reports on the mechanism of the development of the disease and the distribution pattern of the lesions.

The aim of the present paper is to correlate histological findings of the pancreatic lesions with organic and functional changes in vascular and ductal systems. For this purpose, the basic pattern of blood vessels in the pancreas and of the pancreatic ducts was examined microradiographically by the use of contrast medium.

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MATERIALS AND METHODS

The materials of the present study were human pancreases obtained at autopsy within 3 postmortem hours during the period from October, 1965 to October, 1967 at the Department of Pathology, Tohoku University School of Medicine.

1) Radiography

Conceivably normal pancreases of young persons were screened up and radiographs were taken of their blood vessels and ducts in 10 cases each. For the blood vessel examination, each pancreas had been taken out together with the duodenum and the dorsally placed aorta including the celiac and superior mesenteric arteries. A cannula was then inserted into each of these two arteries and the pancreas was rinsed with isotonic salt solution. All the vessels in the peripheral range of the pancreas were ligated except for the cut end of the portal vein. The perfusion was followed by an injection of 50% 'micropaque' solution mixed with 5% gelatin, which could be gently infused to an amount of 70-90 ml until it met resistance. After a fixation for 24 hours, radiographs of the whole pancreas were taken by ultrasoft x-ray (Softex). Then, the pancreas was cut into slices by sagittal sections, and tissue pieces of 5 mm and 500 microns in thickness were excised from each region of the pancreas, i.e., the head, body and tail. With microangiographs of these slices, histological figures from the same specimens were compared in order to examine the distribution pattern of the small vessels. In this examination, the pancreases with the lesions of identified etiologies were excluded. For radiography of the pancreatic ductal system, the pancreas was first taken out together with the duodenum, and a cannula was inserted into the major pancreatic duct through the papilla duodeni, and 3-6 ml of the above micropaque solution were injected. All the other procedures were the same as in the case of microangiography.

2) Histopathology

The materials for histologic investigations were obtained from 108 autopsy cases within 3 postmortem hours. Cases of carcinoma involvement were excluded. The ages ranged from 9 to 81 years. After gross observations over multiple sections transverse to the longi-

<table>
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<th>Inflammation</th>
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<th>Gastric</th>
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tudinal axis, several tissue slices were excised from the head, body and tail to ensure a
systematical study of the entire surface of a transverse section, and fixed mostly by Zenker-
formol and partly by 10% formalin. The staining methods applied to paraffin sections
were Weigert's elastica-Masson and PAS in addition to the ordinary practice. For
microscopic observations, the adopted 108 cases were divided into 8 groups according to
basic diseases (Table 1). Histologic findings over the whole area of each transverse section
were described with considerations on the difference of lesions between central and
peripheral areas of the section.

**Results**

*Arterial Patterns*

1) *Arteriography* (Fig. 1)

The pancreas was found to receive blood supply through eight tributaries from
the following three main trunks: the common hepatic artery, the splenic artery
and the superior mesenteric artery. They were all branches from the celiac artery.
The head was supplied with anterior superior and posterior superior pancreato-
duodenal arteries from the gastroduodenal artery and anterior inferior and
posterior inferior pancreato-duodenal arteries from the superior mesenteric
artery, while the blood to the body and tail was supplied by four tributaries from
the splenic artery. The latter four tributaries had been described by Pierson,3
Olsen and Woodburne4 as 'dorsal or superior pancreatic artery', 'inferior pancreatic
artery', 'pancreatic magna artery', and 'caudal pancreatic artery'. In the head, two
arterial arcades were formed, one by the anastomosis between the posterior superior
and posterior inferior pancreaticoduodenal arteries, and the other by that of
anterior superior and anterior inferior pancreaticoduodenal arteries. Between

<table>
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<th>Other intraperitoneal tumors</th>
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<th>Cardiac</th>
<th>Pulmonary</th>
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the head and body, posterior and inferior pancreatic arteries anastomosed with arteries in the head, and had a large caliber comparable in size with the pancreatico-duodenal artery.

2) Microangiography (Figs. 2-5)

One intralobular artery for each lobule divided from the interlobular artery. After it entered into the lobule, it sent off branches all running straight toward the islets of Langerhans without supplying the acinar parenchyma. Then, they were further subdivided into a large number of branches within the islets to form a close meshwork, which anastomosed with the capillaries of the acinus. This relation of the blood supply between the islets of Langerhans and acini in a lobule was similar to that between the glomerulus and tubules in the nephron. The tail containing many islets of Langerhans generally had abundant ramifications of intralobular arteries. In no region there appeared an interlobular vascular anastomosis. Interlobular pancreatic ducts received blood from interlobular arteries, through branches penetrating the ductal walls in a direction reverse to the flow of pancreatic juice. The arterial branches showed rather meager ramification.

Radiographs of the Ductal System (Figs. 6–9)

The pancreatic ductal system can be classified into the large ducts which run from the tail to head; secondary ducts that, running through the interlobular tissues, unite with the large duct; and peripheral ductules leading to intralobular acini. Unlike blood vessels, pancreatic ducts were in cul-de-sac at their peripheral frameworks, generally lacking in connective tissue in the walls. Therefore, the peripheral ductules were not filled with contrast medium. An attempt even with high pressure to make the medium reach peripheral ductules resulted in a leakage of fluid around the acini. The large duct in the head ran along the center of the pancreas, slightly dorsal to the middle. In other regions it ran nearly along the center and formed a sharp bent between the head and neck, where the behavior in the ramification of accessory ducts was different according to cases. Secondary pancreatic ducts were distributed in each region almost evenly in a radial form, the head, however, containing some large ones. It deserves special attention that the radiography demonstrated more abundant pancreatic ductules in the peripheral range than in the center of each transverse slice.

Histopathological Findings

1) Findings in basic disease relations (Table 1)

According to their basic diseases, total 108 cases examined were divided into eight groups of: biliary tract diseases, hepatic diseases, gastric diseases, other intraperitoneal tumors, renal diseases, cardiac diseases, pulmonary diseases, and others. Histopathological findings of the pancreas were summarized as follows:
Microradiography of the Pancreas

a) Biliary tract diseases (10 cases). Six cases among 10 of this group had severe diffuse lesions including interstitial inflammation, fibrosis, and fatty changes. Also notable in this group was a considerable share of various lesions such as slight edema, cellular infiltration, various types of fibrosis, hyperplasia of ductal epithelium and acinar dilatation. Localized parenchymal and fat necrosis were found in two cases each.

b) Hepatic diseases (16 cases). A high incidence of fat necrosis and cellular infiltration was remarkable. Diffuse interstitial inflammation appeared in three cases out of 16. A relatively high incidence of lesions was covered by extraneous fat invasion, papilliferous epithelial hyperplasia within secondary ducts, and intralobular fibrosis. Localized parenchymatous necrosis and the formation of pseudolobules were found in three and two cases, respectively.

c) Gastric diseases (12 cases). The findings here were rather inconspicuous compared with other groups except in 2 cases out of 12 of diffuse interstitial inflammation.

d) Other intraperitoneal tumors (10 cases). Noted in the cases in this bracket was a comparatively high incidence of cellular infiltration.

e) Renal diseases (8 cases). In this group there was a high incidence of cellular infiltration, acinar dilatation and scattered intralobular replacement by fat tissue. In addition, parenchymatous necrosis and fat necrosis were observed in two cases.

f) Cardiac diseases (11 cases). The incidence of scattered foci of replacement by fat tissue was highest in this group. The other noticeable finding was intensive fatty change in two cases. The formation of pseudolobules was found in four cases.

g) Pulmonary diseases (7 cases). Replacement by fat tissue dominated in this group. One case had diffuse hemorrhagic pancreatitis.

h) Others (34 cases). The findings were rather unremarkable in this group except for one case of hydrargyriasis with diffuse hemorrhagic pancreatitis.

2) Regional distributions of various lesions (Table 2)

Various lesions checked in the present examination were tabulated according to their regional location of head, body and tail. Observations were accordingly conducted for the following findings.

a) Edema. In the current tabulation, edema appeared in the tail with a frequency slightly lower than that in the head or body. This trend was remarkable in four cases in the biliary tract group with conspicuous findings, but the regional difference was not so remarkable in all the other disease groups. The lesion extended diffusely in a sagittal section and no difference was confirmed between the central and peripheral ranges.
b) **Cell infiltration.** The lesion appeared with a lower frequency in the tail than in the head or body. The trend is likewise remarkable in the group of biliary tract diseases just as in the case of edema. In sagittal sections the lesion preferred the anterior and posterior periphery. The trend was also notable in the hepatic disease group.

c) **Fat necrosis.** No remarkable regional difference was confirmed in its incidence. In sagittal sections the lesion usually appeared most conspicuously in the anterior periphery, while in the tail it also preferred the posterior periphery.

d) **Parenchymal necrosis.** No definite tendency in its incidence was confirmed according to regions. Necrosis was generally irregular in shape and occasionally localized in the periphery or center of the lobule and around pancreatic ducts.

e) **Fibrosis.** Fibrosis of all kinds appeared more frequently in the head and body than in the tail. Intralobular fibrosis showed two different patterns: in one of them fibrosis was diffusely distributed around the acini, and in the other it was localized or accentuated around pancreatic ductules, small vessels, and the islets of Langerhans. The lesions of localized or accentuated pattern were predominant in the peripheral zone along the anterior and posterior surface.

f) **Fat invasion.** Fat invasion was prevalent in the head and body than in the tail. The lesion also preferred the anterior and posterior periphery.

g) **Replacement by isolated fat cells.** No distinct regional difference was observed in its incidence. Dominant appearance was found in the anterior periphery of the head and scattered lesions were found diffusely distributed in the body and tail. This trend was observed in all disease groups.

h) **Formation of pseudolobules.** The lesion was more prevalent in the body and tail than in the head.
DISCUSSION

1) Relation of Pancreatic Lesions to Vascular Systems

Circulatory disturbance has been regarded as one of the major pathogenetic factors of pancreatitis. In sustaining normal pancreatic circulation, it is important that the blood flow in each region of the pancreas is kept constant. It is further interesting that the vascular pattern of the pancreas characterized by abundant anastomoses among major arterial branches seems to be effective in preventing pancreatic infarction: this condition is rare and could not be found in the present study.

Two arterial arcades in the head connect with the splenic artery in the head and body (posterior and inferior pancreatic arteries) and work at the same time as collateral channels to the hepatic artery and the superior mesenteric artery. This anastomosis with branches of the superior mesenteric artery or of the common hepatic artery varies considerably from case to case, and may be even absent in rare instances. Hentschel injected two kinds of dyes into the pancreas at autopsy, each through the celiac artery and the superior mesenteric artery and observed that the blood supply to the body and tail as well as to the anterio-superior portion of the head came from the celiac artery, while the posterio-inferior portion of the head alone received the blood from the superior mesenteric artery. He explained these findings by the embryological difference in the pancreatic primordia, viz., the former is of dorsal origin, while the latter originates from ventral sprout. Certainly his explanation seems plausible so far as the anatomy is concerned, but it is probable that the actual blood flow in the living organ may not follow his experimental finding. According to certain reports on pancreatic circulation, dyes injected through one of the three major pancreatic arteries in the anesthetized dog were distributed all over the pancreas, though not absolutely complete. Based on this finding, interregional blood flow through anastomoses was assumed in the pancreas covering different ranges irrigated by different arterial trunks. On the other hand, the anastomoses are still short of covering completely the function of the vascular trunks. An experimental ligation of one of the arterial trunks produces in some cases localized small necrotic foci, although gross infarction does not develop. However, there is a clinical report that the occlusion of the superior and inferior pancreatico-duodenal arteries brought about duodenal necrosis but no change whatever in the head of the pancreas. In view of the results available as above, it is considered that the collateral arteries in the pancreas work in substitution for the blood flow of certain arteries and the obstruction in one of the three trunks — the common hepatic artery, the splenic artery, and the superior mesenteric artery — will not induce such a serious change as infarction in the pancreas. However, in the pancreas lacking vascular anastomosis between the head and body, the tail receives blood supply neither from the common hepatic artery nor from the
superior mesenteric artery, and consequently occlusion of the splenic artery as in thrombosis may induce pancreatic infarction.

Microangiography revealed no anastomosis between the lobules. Schönbach et al. had also regarded the intralobular artery as a ‘terminal artery’. Because a simple ligation of an arterial trunk does not cause a serious change in the pancreas, various investigators attempted to induce hemorrhagic infarction and other severe damages by injecting foreign materials such as oil, wax, air and mercury into the pancreatic arteries. Pfeffer et al. injected particles, different in size from 8 to 400 microns in diameter, into the pancreatico-duodenal artery and confirmed that pancreatic necrosis was produced exclusively by fine particles (8 to 20 microns in diameter), while larger ones had not such an effect. As a main agent causing such necrosis, they suggested the blockade of the minimum unit of the vascular system without collateral channel. The lobule is regarded as representing this minimum unit. Therefore, if the arterial region originating intralobular arterioles is subjected to obstruction, ischemic lesions covering the lobular area would eventually be induced. This type of arterial circulatory disturbance will lead to localized necrosis and other lesions, and may be caused by atheromatous embolization, malignant hypertension and periarteritis nodosa. The present authors found localized necrosis in 10 cases. Among them only one case with malignant nephrosclerosis had organic changes in the arterial wall and necrosis was induced in the center of a lobule in the tail of pancreas. It is of interest to note that necrosis of this case was nearly the same as central necrosis which Smyth produced in an animal experiment by means of mercury injection.

In addition to arterial factors, venous factors such as passive congestion and thrombosis have been regarded as being responsible for inflammatory diseases of the pancreas. It has also been pointed out that coronary heart diseases are frequently associated with local pancreatic inflammation. Even in the absence of organic changes, a decreased blood flow, e.g., in shock, will result in anoxia or venous congestion. Some of those localized slight changes as often seen in the autopsy pancreas may be induced by venous factors. In this regard, the term of ‘terminal pancreatitis’ has been advocated by some authors. In the present study, inflammatory lesions in the pancreas were not prevalent in the group of cardiac disease and only one case had localized necrosis. Cases with venous dilatation, on the other hand, showed a high incidence of inflammatory changes though pathogenetic analysis failed to give any definite answer. In the pathology of the pancreas, portal venous congestion deserves attention along with the systemic circulatory disorder. This will not particularly significant in cases of inflammatory lesions frequently associated with hepatic diseases.

In cases with congestive heart failure, the formation of pseudolobules was occasionally found as a specific picture. This lesion slight in degree was observed in the present study in 4 cases of cardiac disease, 2 cases of hepatic disease and 1 case of gastric ulcer associated with the Banti syndrome. The development
of the lesions of this kind is attributed to the condition that the acini distant from
the islets of Langerhans are more susceptible to atrophy due to passive congestion
than those near the islets, because of their location distal to the arterial trunk
as revealed by microangiography. Pseudolobules slight in degree were also
observed in the group of liver diseases and this would be ascribed to the effect
of portal venous congestion. The lesion was found more frequently in the body
and tail than in the head. This difference according to anatomical areas will
depend on different density of the islets of Langerhans as well as on different
anatomical positions in the portal system.

It was noticed that the cardiac group registered the largest share of scattered
fat necrosis and included two cases of severe fatty change. Aging process,
pancreatic ductal occlusion, circulatory disturbances and metabolic disorders are
regarded as the causes of pancreatic fatty change.22 The present authors would
like to emphasize the importance of circulatory disturbances in the pathogenesis
of fat replacement, i.e., appearance of isolated fat cells intralobularly scattered.
The lesion was found predominantly in cases of cardiac diseases and of arterio-
sclerosis in the present study and was considered to be different in source from
that of interlobular one, which was seemingly an extension from the surrounding
tissue.

The lesion preferred the anterior periphery of the head and the whole area
in the body and tail. This distribution pattern agrees with the territory of the
celiac artery.5 Fat replacement was also predominant in cases of renal diseases,
and this was suggestive of some connection of the lesion with acinar ectasia. In
the cardiac group, however, the development of acinar ectasia was not remarkable.
This indicated that the major cause here was rather atrophy of acinar cells. In
cases of pseudolobule formation, the development of fat tissue was also predomi-
nant in the area of atrophied cells located apart from the islets of Langerhans.
Without doubt circulatory disturbances were responsible for a series of these
changes so far as this disease group was concerned. In this connection it is to
be pointed out that extensive fatty changes are predominantly caused by
pancreatic ductal occlusion, but they may be induced rarely in the course of
circulatory disturbances.

As to acute pancreatitis mainly consisting in hemorrhagic necrosis, it is not
determined whether circulatory disturbances play a primary or secondary role in
its pathogenesis.23,24 In the present study, hemorrhagic pancreatitis was observed
in two cases, one with pulmonary tuberculosis and the other with hydrargyriasis.
In this case of tuberculosis the patient had fallen into dyspnea with transient
heart failure two days before death. Cardio-massage relieved the patient from
immediate death but left him in a state of continued hypotension. This case
seems to suggest an important role of circulatory disturbance in the etiology of
pancreatitis and to cast doubt upon the recent theories, in which circulatory
disturbance is regarded only as a secondary agent.1,2,24
2) Association of Pancreatic Lesions with Ductal Systems

On radiographs of the duct system, a sharp curve of the large pancreatic duct between the head and neck is regarded as the joint of two major ducts from two embryologically different primordia, one being the large duct in the head (the duct of Wirsung) of the ventral origin and the other of the dorsal genesis running from the tail to neck. As the ventral primordium unites with the dorsal one on the posterio-inferior of the right pole, the large pancreatic duct is thought to run a little posterior in the head. The pancreatic duct in the head from the dorsal primordium is observed as either an evident accessory pancreatic duct (the duct of Santorini) or an obscure one. The accessory duct has been regarded as an important collateral channel when the major pancreatic duct is obstructed at the papilla.

As to the development of pancreatitis, stasis of pancreatic juice and supervening juice leakage into the interstitial tissue are predominant in its etiology. Different from the blood flow complicated with anastomoses, the flow of pancreatic juice is very simple, taking clear courses from the tail to head, and from the periphery to the center. Therefore, any occlusion of a pancreatic duct due to organic and/or functional changes will injure the exocrine glands peripheral to the point of ductal occlusion.

As causal factors for pancreatic ductal occlusion are counted edema, stones, tumors, spasm of the sphincter of Oddi and fibrosis at the papilla; squamous metaplasia of pancreatic ductal epithelium; calculus and round worm in pancreatic ducts; and intrapancreatic tumors. Birnstingl and Berman et al. observed inspissated ductal secretion, squamous metaplasia of the ductular epithelium and localized fibrosis in the portion where pancreatic ductules were indiscernible on the ductal radiogram or the vinyl acetate cast of postmortal pancreases. They explained that localized lesions on periacinar and perilobular fibrosis was due to blockade of small ducts by intraductal epithelial proliferation or by ductal inspissation. The present authors also confirmed these findings as well as proliferation of pancreatic ductules in the area of fibrosis. But the question whether or not localized fibrosis precedes any of these changes has remained unsolved. Nevertheless, the peripheral range of the pancreas was abundant in localized fibrosis, mild and intensive, and also in radiographically demonstrable ductules. These findings suggest a relationship between the development of fibrosis and pancreatic ductal occlusion. Fibrosis of lobular unit was also observed in the peripheral range of the pancreas and this seems to be induced by circulatory disturbances as was described above. On the basis of pancreatography, Birnstingl confirmed a close relationship of localized parenchymal and fat necrosis to ducts with intraductal epithelial proliferation. He considered that the thin-walled spaces accompanying pancreatocyst and other cystic changes in the ducts and glands would make the leakage of pancreatic juice easy. Besides, functional disturbance such as spasm of pancreatic ducts is also considered to bring about congestion and leakage of pancreatic juice. Periductal cell infiltration...
and small foci of necrosis in the present materials are presumed to have an effect of pancreatic juice leakage. The same effect may be assumed in the case of small localized foci of perilobular necrosis, according to the experimental results obtained by Menguy et al.9

Extra-pancreatic occlusive factors, e.g., various lesions in the papilla of Vater and the sphincter of Oddi are thought to play an influential role in causing pancreatitis frequently associated with biliary tract diseases. Actually, the present authors observed severe and mild lesions of interstitial pancreatitis, fibrosis and fatty changes. As to the regional distribution, edema, cellular infiltration and fibrosis were most prevalent in the head and this trend was most conspicuous with edema. Considering occlusive factors as the causes, the tail should be subject to deleterious effects more easily than the head. But the fact that these lesions contrarily occurred predominantly in the head suggests the additional effect of inflammation extending from the biliary tract.

**CONCLUSION**

Microangiographically, the intralobular arteries of the pancreas are demonstrated as terminal arteries, and their obstruction induces localized atrophy, necrosis and fibrosis of the parenchyma of lobular dimension. Circulatory disturbance generally causes acinar cell-atrophy with sequent diffuse fatty replacement. However, such changes result rarely in severe and extensive lesions. The formation of pseudolobule in many cases is caused by circulatory disturbances due to venous stasis.

Organic and/or functional occlusion in any unit of the pancreatic ducts will lead to various kinds of lesions of the pancreas. The peripheral range of the pancreas, embracing a mass of ductules, is associated with a high incidence of fat necrosis, localized fibrosis or locally intensive fibrosis. Localized periductal necrosis and cellular infiltration and also peripheral lesions of intralobular necrosis are presumed as the outcomes of the reactions to pancreatic juice leakage.

Pancreatic lesions of either vascular or ductal origin are considered to interplay each other, even resulting in serious changes, e.g., hemorrhagic necrosis.

**References**

6) Andoh, S. Studies on the vascular supply of pancreas. Report 1. The arterial
[Illustrations follow]
Fig. 1. Arteriogram of the whole pancreas.
A. Common hepatic artery.
B. Splenic artery.
C. Superior mesenteric artery.
1. Anterior-superior pancreatico-duodenal artery.
2. Posterior-superior pancreatico-duodenal artery.
3. Anterior-inferior pancreatico-duodenal artery.
5. Posterior pancreatic artery.
6. Inferior pancreatic artery.
7. Pancreatic magna artery.
Arrows indicate anastomoses between the head and body.

Fig. 2. Radiogram of an interlobular artery with intralobular arterial branches directly leading to the islets of Langerhans. ×100.

Fig. 3. Histologic picture of Fig. 2 under the same magnification. (Weigert's elastica-Masson stain, ×100)

Fig. 4. Radiographically demonstrable anastomoses of the arterioles in the islets of Langerhans with those of exocrine portion. ×100.

Fig. 5. Arteriogram in a part of exocrine parenchyma adjacent to a duct (upper part). ×100.
Fig. 6. Ductogram of the whole pancreas showing large secondary ducts in the head.

Fig. 7. Cross-cut sections of head, body and tail (left to right). Each shows abundant small ducts at peripheral range.

Fig. 8. Peripheral range of a cross-cut slice showing abundant branches of radiographically demonstrable small ducts. ×10.

Fig. 9. Histologic picture of Fig. 8 under the same magnification. (Weigert's elastica-Masson stain, ×10)
Microradiography of the Pancreas
Fig. 10. Formation of pseudolobule. Acinar cells distant from the islets of Langerhans show atrophy. (Hematoxylin and eosin, ×200)

Fig. 11. Replacement of atrophied acinar cells by fat cells associated with the formation of pseudolobule. (Hematoxylin and eosin, ×200)

Fig. 12. Fibrosis as seen at the peripheral range of a cross-cut section. (Weigert's elastica-Masson stain)
Fig. 13. Fibrosis localized in a lobule associated with fat tissue invasion. (Weigert’s elastica-Masson stain, ×10)

Fig. 14. Lesions of parenchymal necrosis mainly involving the periductal area. (Weigert’s elastica-Masson stain, ×40)

Fig. 15. Inflammatory cell infiltration localized in the periductal area. (Hematoxylin and eosin, ×100)