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HEPATIC LESIONS IN SECONDARY SHOCK AND ACUTE CARDIAC FAILURE

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(Received August 19, 1959)

The literature contains numerous clinico-pathological and experimental data concerning functional and morphological changes induced in the kidney during secondary shock. The particular attention paid to renal changes of this kind is not surprising if we consider that damages of the kidney during secondary shock — designated by special terms, *e. g.* lower nephron nephrosis, crush syndrome, post-traumatic or post-operative anuria, shock kidney, etc. — frequently lead to death. Hepatic damage of a similar nature has far less been investigated. It was in the first place by German pathologists that hepatic lesions of aviators who had died of high altitude death, "akuter Höhentod", were studied during World War II these workers succeeded in reproducing these lesions in animals by exposing them to low atmospheric pressure and oxygen deficiency. As regards human patients, there are comparatively few pathological and still fewer clinical data concerning hepatic lesions due to similar causes.

Material and methods

Between 1956 and 1959, we studied the liver of all autopsied cases in which death had been preceded by symptoms of a longer or shorter shock, such as sharp diminution of blood pressure, impalpable pulse or — not infrequently — transitory or lasting loss of consciousness. In 32 cases were liver lesions encountered which are usually held characteristic. In 23 cases we examined also the kidneys. The specimens excised were fixed in formol, embedded in paraffin and stained with hematoxylin-eosin, Endes'thrichrome (12a) dye or — after freezing — with hemalum-Sudan III.

Summary review of the records

No. 1 (11) 1956. S. M., male, 62 years of age. Admitted with diarrhoea, bloody discharge. Gradual dehydration for 2 days, then death. Autopsy revealed grave acute and diffuse ulcerative enterocolitis.

No. 2 (20) 56. T. M., male, 56 years of age. Admitted with chronic cardiac decompensation and symptoms of acute intestinal obstruction. Intestinal suction and hemodialysis were instituted. Autopsy revealed scarring of the aortic valves with consecutive insuffiency, dysenteric enterocolitis, incipient diffusion peritonitis.

No. 3 (579) 56. B. F., female, 61 years of age. Clinical diagnosis: pancreatitis and intestinal obstruction. Death after 2 days. Autopsy: thrombosis of the descending branch of the left coronary artery with consecutive myocardial infarction.

No. 4 (16) 57. M. G., male, 38 years of age. Acute cardiac decompensation; death after 24 hours' unconsciousness. Autopsy revealed acute pancreatic necrosis, grave myocardial degeneration, acute dilatation of right ventricle.

No. 5 (22) 57. I. P., female, 59 years of age. Clinical diagnosis: myocardial infarction. Death after 3 days. Autopsy: thrombosis of the descending branch of the left coronary artery with myocardial infarction.

No. 6 (56) 57. J. H., male, 3 years of age. Death after a few weeks of measles bronchopneumonia. Autopsy, mostly organizing bronchopneumonia extending to all lobes; subacute cor pulmonale.

No. 7 (62) 57. A. L., female, 72 years of age. Palliative gastrostomy on account of inoperable gastric carcinoma. Autopsy, ulcerated cancer of the stomach with metastases; suppurative aspiration bronchopneumonia.

No. 8 (137) 57. P. B., male, 41 years of age. Died with symptoms of uremia after anuria of 11 days' duration following extensive contusions. Autopsy, contusion of both thighs and the gluteal region; confluent bronchopneumonia; the kidneys showed on both gross and microscopic examination grave myoglobinuric nephrosis.

No. 9 (411) 57. I. S., male, 53 years of age. Admitted with transverse myelitis, paralyzed lower limbs and cystitis; repeated collapses with hypotension. Autopsy, myelomalacia; immediate cause of death: embolism of pulmonary artery.

No. 10 (412) 57. J. K., female, 38 years of age. Had been suffering from disseminated lupus erythematosus for 4 years. Pathological picture dominated by renal lesion during the last 2 years. Hypertension. Sharp hypotension preceding death. Autopsy, disseminated arteriolar necrosis in the intestines, kidneys and the cerebellum, characteristic symptoms of malignant hypertension accompanied by inflammatory phenomena.

No. 11 (490) 57. M. S., male, 68 years of age. Admitted with acute abdomen failure due to gastric perforation. Aggravation of shock after explorative laparotomy, death on the next day. Autopsy, acute pancreatic necrosis with circumscribed peritonitis.

No. 12 (M. 220) 57. A. K., male, 57 years of age. Had had hepatitis three years ago. Admitted with hematemesis. Explorative laparotomy followed by gradual diminution of blood pressure for two days, then death. Autopsy, postnecrotic cirrhosis with gross nodules; fatal hemorrhage following rupture of oesophageal varix.

No. 13 (M. 24) 58. I. Sz., male, 57 years of age. Died with symptoms of grave cardiac decompensation. Autopsy, bronchiectasis, cor pulmonale, myocardial degeneration, pulmonary sclerosis.

No. 14 (37) 58. A. M., female, 13 years of age. Had been operated on five months before for ovarian tumour. Admitted with intestinal obstruction. Death after 5 hours' unconsciousness. Autopsy, intestinal obstruction caused by recurrent malignant teratoma.

No. 15 (66) 58. I. K., male, 45 years of age. Explorative laparotomy because of suspected gastric perforation, followed by marked hypotension. Death 2 days later with symptoms of shock. Autopsy, ichorous pleurisy and mediastinitis caused by perforated peptic ulcer in the lower portion of the oesophagus.

No. 16 (96) 58. L. F., male, 62 years of age. Laminectomy on account of transversal lesion of the spinal cord; removal of tumour. Died 3 days later of circulatory failure. Autopsy, multiple myeloma, foci in the vertebrae and many other bones.

No. 17 (114) 58. S. V., male, 47 years of age. Clinical diagnosis, circulatory failure and pulmonary fibrosis. Autopsy, organizing fibrinous endocarditis on the mitral and tricuspidal valves. Organizing emboli in many small branches of the pulmonary artery, probably originating from the tricuspid valve.

No. 18 (M. 194) 58. A. K., female, 42 years of age. Choledochoduodenostomy and removal of gallstones three days before death. Autopsy, diffuse fibrinous-suppurative bilious peritonitis due to sutural insuffiency.

No. 19 (207) 58. J. K., male, 29 years of age. Angina pains during the last month; death with symptoms of myocardial infarction. Autopsy, obturating thrombus in the descending branch of the left coronary artery with consecutive myocardial infarction.

No. 20 (230) 58. A. H., male, 61 years of age. Thoracolaparotomy on account of tumour of the cardia. Death after 3 days of shock. Autopsy, gastric carcinoma on the base of of chronic peptic ulcer; diffuse peritonitis; intestinal paralysis.

No. 21 (234) 58. S. F., male, 74 years of age. Resection of coecum and partial resection of colon for coecal carcinoma; death after two days. Autopsy, confluent bronchopneumonia; diffuse ichorous peritonitis consequential upon sutural insufficiency.

No. 22 (271) 58. M. F., male, 46 years of age. Resection of stomach and gastroenteroanastomosis for gastric ulcer. Collapse after a hot bath on the 8th day, drop of blood pressure

to 60/0; the next day death, apparently due to central and peripheral circulatory failure. Autopsy, grave myocardial degeneration; incipient acute intestinal paralysis.

No. 23 (392) 58. G. M., male, 19 years of age. Acute leukemia. Treatment with Degranol followed by sharp drop in leucocyte count; bronchopneumonia and death. Autopsy, extensive bronchopneumonia; leukemic infiltrations in the internal organs.

No. 24 (400) 58. K. D., male, 74 years of age. Operation for perforated duodenal ulcer followed after 3 days by death with cardiac decompensation. Autopsy, fibrinous inflammation in the small intestines, slight purulent peritonitis, degenerated myocardium.

No. 25 (16) 59. I. Sz., male, 68 years of age. Resection of large intestine because of sigmoid tumour, followed by emesis, peritonitis, cardiac failure, then death. Autopsy, diffuse peritonitis due to sutural insufficiency; aspiration bronchopneumonia.

No. 26 (15) 59. J. H., male, 20 years of age. Rheumatic fever during the last two years; precordial pains during the last two days; death after hypotension during the last day. Autopsy, partly cicatrizing and partly acute rheumatic endocarditis of the aortic and bicuspid valves; adhesive pericarditis.

No. 27 (42) 59. G. T., female, 22 years of age. High fever during the last week, pneumonia; tracheotomy on account of asphyxia. Death with symptoms of shock. Autopsy, partly interstitial, disseminated pneumonia of unknown origin; acute dilatation of right ventricle.

No. 28 (31) 59. E. B., female, 8 years of age. Influenza, high temperature, tonsillitis. Unconsciousness during 24 hours followed by death with cardiac failure. Autopsy, bronchitis, bronchiolitis, disseminated bronchopneumonia. Death seems to have been due to acute circulatory insuffiency induced by toxic influenza.

No. 29 (66) 59. M. B., male, 72 years of age. Gastrectomy and gastroenteroanastomosis for scarred peptic ulcer. Autopsy, beside old, scarred infarction fresh infarction in the wall of the left ventricle; circumscribed fibrinous-suppurative peritonitis in the operated area.

No. 30 (71) 59. L. P., male, 59 years of age. Thoracic pains and unconsciousness a few weeks prior to admission. Death with symptoms of cardiac failure and hypotension. Autopsy, thrombosis of the descending branch of the left coronary artery with consecutive myocardial infarction and rupture of interventricular septum.

No. 31 (K. 72) 54. A. K., male, 19 years of age. Unsuccessful operation of coarctation of the aorta, followed by shock. Transitory increase of blood pressure, thereafter death with symptoms of intestinal paralysis. Autopsy, disseminated necrotized foci varying in size in the brain, small intestine and kidneys, presumably due to grave postoperative hypotension.

No. 32 (K. 59) M. P., female, 52 years of age. Shock after cholecystectomy; unmeasurable blood pressure. A few days later oliguria, then death with uremia in spite of preceding renal decapsulation. Autopsy revealed signs of uremia with grave morphological changes, characteristic of "shock kidney".

Results

Generally speaking, in none of the cases did the liver reveal a characteristic picture on gross examination. It was only in some of the graver cases that the greyish-white or yellowish-white necrotic foci of the central part of the lobules in the surrounding brownish-red hepatic parenchyma resembled the picture of nutmeg livers. The characteristic nutmeg liver pattern was observed in cases where the changes had been preceded by chronic cardiac congestion.

Histological analysis revealed certain more or less characteristic lesions in each case. Among such lesions were the vacuolar degeneration of the cytoplasm of liver cells, frequently with protein-like homogeneous droplets in the vacuoles, further the fatty degeneration of the liver cells. Necrotic single cells or necrotic foci, mostly centrilobular, were invariably observable. Another characteristic phenomenon was the appearance of wide, blood-filled sinusoids and petechiae in the area of the necrotic foci.

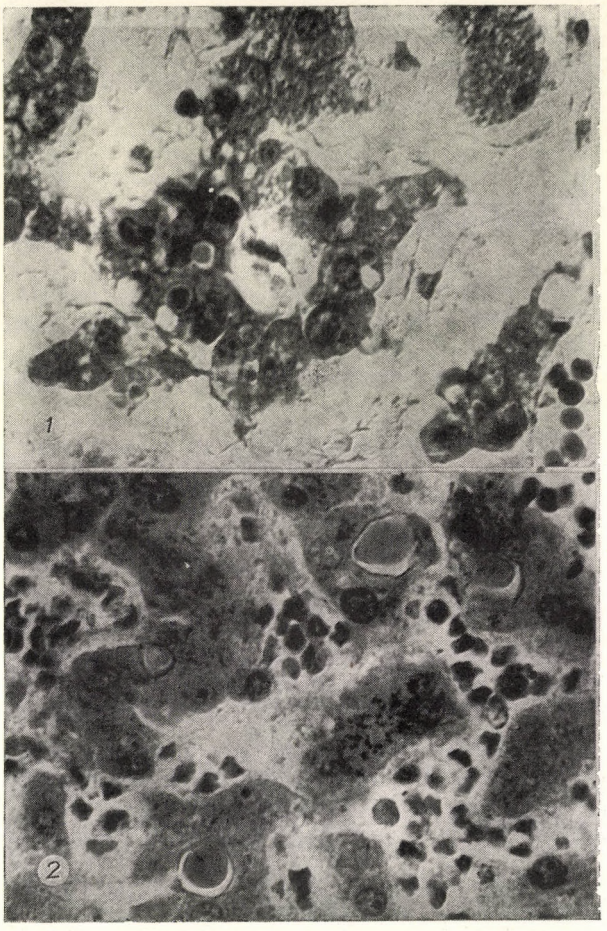

Fig. 1. (No. 4). Protein vacuoles surrounded by a clear zone in the cytoplasm of liver cells. Endes' trichrome. × 700

Fig. 2 (No. 29). Vacuoles in the cytoplasm of liver cells, containing bile clots and surrounded by a clear zone. H. E. × 580

Considering the gravity and extension of the said lesions and taking possible earlier hepatic lesions also into account, we have divided our cases into four categories.

Group I (Nos 3, 8, 11, 21, 22, 28) included 6 cases in which the hepatic lesions were slight, mostly incipient.

The cytoplasm of the liver cells in this group contained various kinds of vacuoles. Part of them, especially those in the vicinity of necroses of those situated in the necrotic cells, were empty and devoid of fat. Another part contained a drop of protein-like material surrounded by a clear zone, and stained light blue with trichrome *(Figs. 1, 2)*. In similar position, but some-

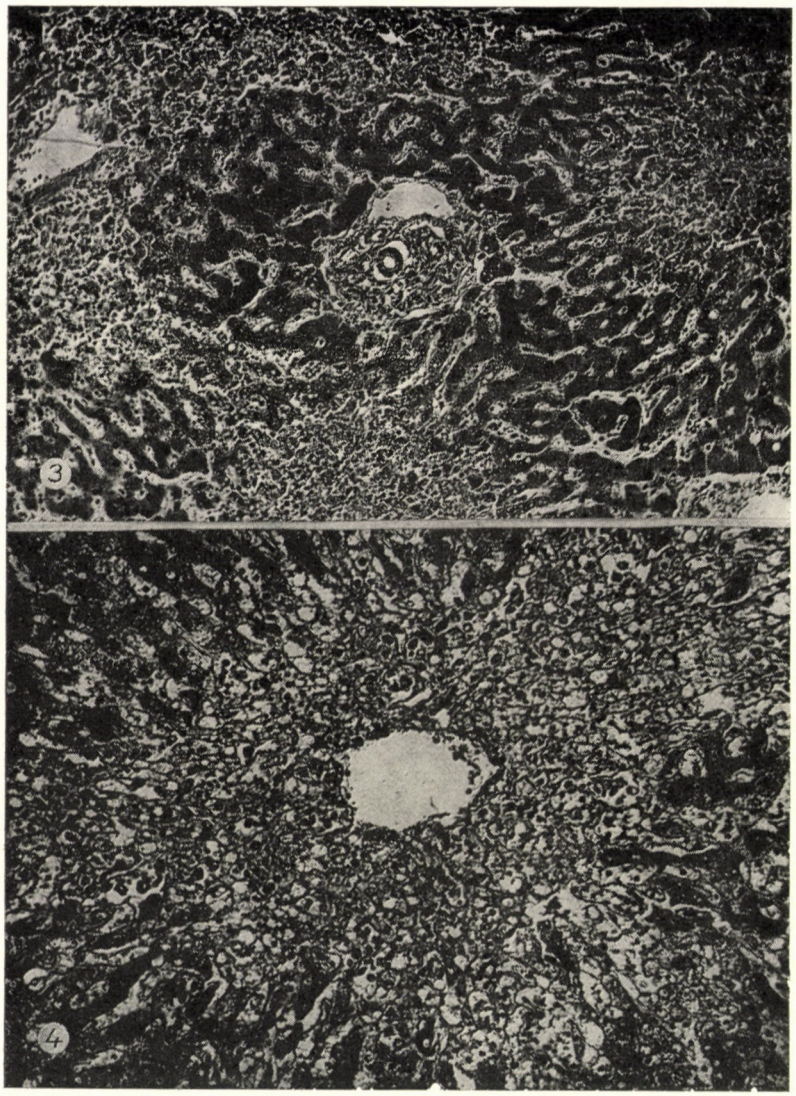

Fig. 3 (No. 24). Partly confluent, centrilobular necrotic foci. Hepatic parenchyma containing minute vacuoles can be seen in the centre of the picture: it surrounds a portal area. Endes' trichrome. × 110

Fig. 4 (No. 27). Parenchymal cells destroyed, reticulin framework intact in the necrotized hepatic tissue. Gömöri's silver impregnation. × 130

times independent of necrotic foci, minute or larger fatty vacuoles were frequently encountered in the liver cells. Minute vacuoles were always present, whereas the number of the larger ones varied from case to case. Necrotic single cells were always present, while the necrotic foci were few and small, they were usually situated in the central area of the lobules. Some of the

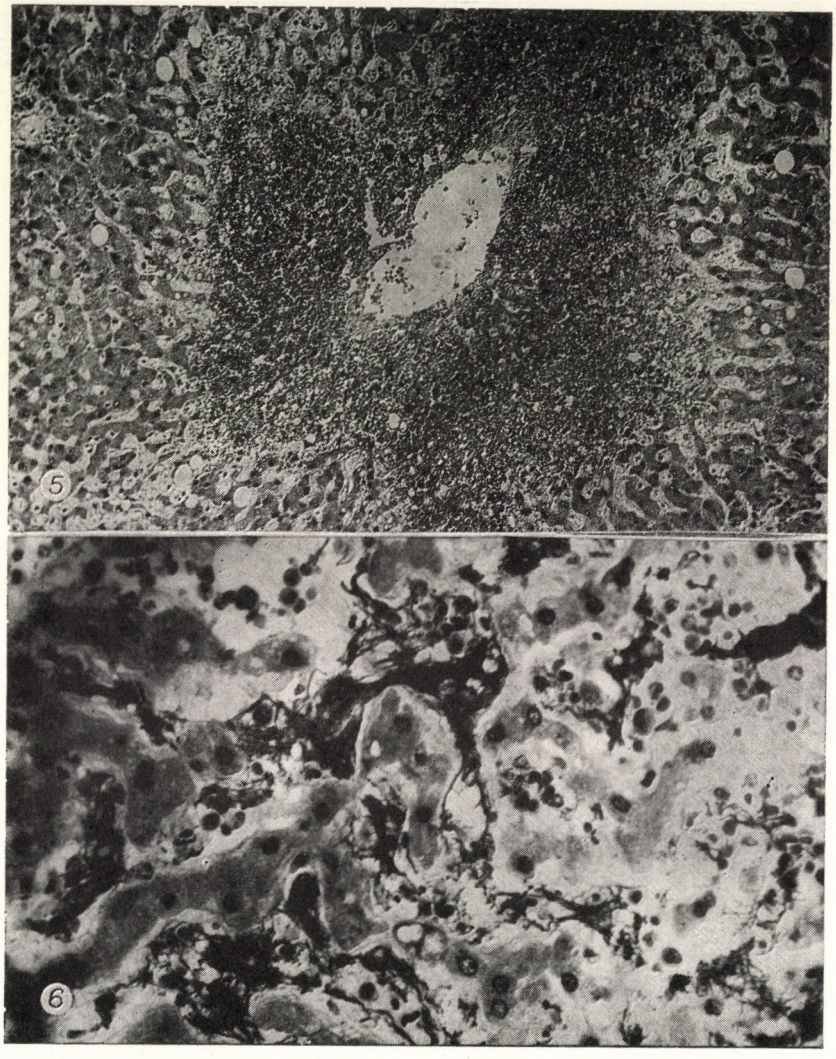

Fig. 5 (No. 19). Necrotized hepatic parenchyma with blood pool, around dilated central vein. Adjacent to focus, large and small fatty vacuoles. H. E. × 100

Fig. 6 (No. 26). Delicately patterned, in some part aggregated fibrin network in the sinusoids of the necrotic foci. Endes' trichrome. × 370

necrotic liver cells had no nuclei: they had dissolved and disappeared. Others contained an intensely staining pyknotic nucleus in the homogeneous eosinophil cytoplasm. Extravasation of blood, so-called »Blutseen« (blood pools), in the necrotic area were observed in a single case.

Group II included 17 typical cases (Nos 1, 4, 5, 6, 7, 9, 13, 14, 15, 16, 18, 20, 23, 24, 25, 31, 32) in which the hepatic structure was gravely damaged.

Vacuoles with protein droplets were invariably present; their number was small and they were mostly situated in or around the necrotic areas. Minute fatty vacuoles were observed in all cases, while larger ones only in four. Characteristic of the picture were the frequently extensive necroses in the centre of the lobule or occupying a sector of this. Adjacent necroses were often coalescing, forming a ringlike figure surrounding the preserved parenchymal islets around the portal areas *(Fig. 3)*. Apart from centrilobular necroses focal ones and necrotic single cells were present. Only the parenchymal cells

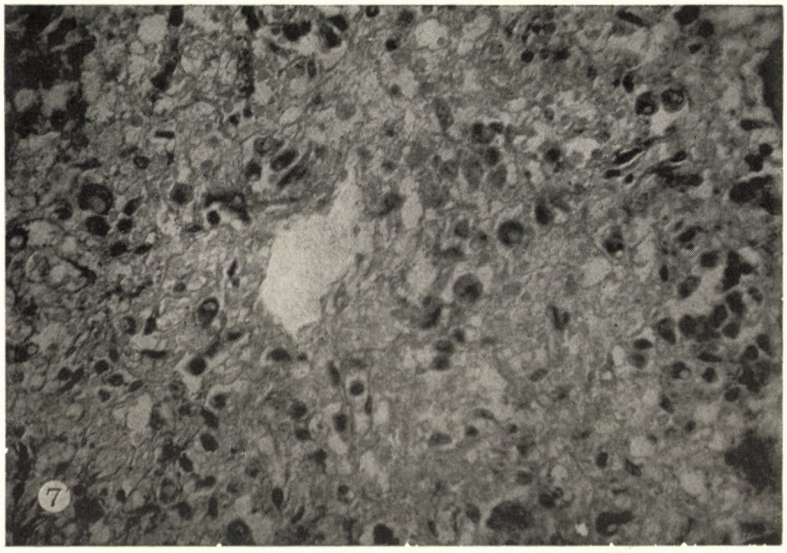

Fig. 7 (No. 27). Slight increase of fibers at the site of a necrotized area. Endes' trichrome. × 340

were affected in these areas, the reticulin framework survived *(Fig. 4)*. In the area of large central necroses blood pools were encountered in 6 cases *(Fig. 5)*, and, independently, some sinusoids were greatly dilated and engorged with erythrocytes.

Group III included 8 cases (Nos. 2, 10, 17, 19, 26, 27, 29, 30) where the said acute lesions appeared in a congested hepatic tissue. Atrophied hepatocellular trabecules and, between them, dilated blood-filled sinusoids were observable around the dilated central veins. A characteristic feature of this category were the extensive necroses, usually with blood pools. There were few protein-containing vacuoles; some of the vacuoles were filled with yellowish-green bile clots. Small fatty vacuoles were invariably present, while large ones less frequently. In 4 cases a delicate fibrinous network was seen in the necrotic foci and, independently, in the sinusoids: this network was conglutinated in

some places *(Fig. 6)*. In some protracted cases a slight increase of the fibres was observed at the site of the central necrosis *(Fig. 7)*.

Group IV consisted of a single case (No. 12) where it was in a cirrhotic liver that the alteration occurred. Necrotic foci were seen in the irregular regenerative parenchymal islets, some of them with blood pools. There were a few protein and fatty vacuoles in the liver cells adjacent to the necrotic areas.

Discussion

MARTIN *et al.* [32] were the first to describe vacoular degeneration of the liver cells on the effect of oxygen deficiency. On the basis of decompression experiments PICHOTKA [39] was the first to regard vacuolar degeneration as a specific consequence of oxygen deficiency. ROSIN [41] found various animals (mouse, guinea pig, rabbit, chicken) to differ in the sensitivity to oxygen deficiency. ATERMAN [2], too, regards vacuolation as the most frequent symptom of hypoxia. Alterations occurring in the liver of aviators who had crashed from great altitudes because of »akuter Höhentod« due to hypoxia, were described by MÜLLER and ROTTER [35]. Similar liver injuries were described by the American authors LEWIS and HAYMAKER [27] and the French GRANDPIERRE and GROGNOT [17]. HESSE [20] found no vacuolation in the liver of airmen who had crashed from low altitudes, *i. e.* from atmospheric layers with adequate oxygen tension. Hepatic lesion was observed by MOON [33] after battle casualties; by HESSE [20] and KETTLER [23, 23*a*, 24] in autopsy material.

TERBRÜGGEN [47] and TROWELL [48] refute the theory that vacuolation is characteristic of oxygen deficiency. FARKAS [14] regards vacuolation as the consequence of a physical process. TÖRŐ [48] writes of aqueous vacuoles of hepatocellular origin and attributes them to a release of the water bound by the cytoplasm. SHERLOCK [44] applies the term "hydropic degeneration" to the process in question. ALTMANN [1] found the lesion to regress in surviving animals and stated that the process was reversible. HANZON [19], studying the liver of rats under the fluorescence microscope, observed a rapid appearance and disappearance of the vacuoles. MÖLBERT and GUERRITORE [34] demonstrated by electron microscope the decomposition of the cristae mitochondriales and the reversibility of the process.

MALLORY [30] and ULRICH [50] observed inclusions in the vacuoles which were regarded by MALLORY as a symptom of incipient necrosis. Many authors qualify this substance as fibrin. Treated with trichrome, the droplets stain blue or red. ALTMANN [1] suggests that the colour depends on the water content: if concentrated, the substance stains red; if rich in water, it stains blue.

HORTEGA [22] considers the droplets of hepatocellular origin; BÜCHNER [5, 6], that they consist of cellular protein. Most authors, however, attribute

their existence to blood plasma entering the liver cells, a theory which HAITINGER and GEISER [18] as also NAIRN *et al.* [36] tried to prove by means of the fluorescence microscope.

TROWELL [49] suggests that vacuolar degeneration is due to a rise of capillary pressure, while KETTLER [23, 23*a*, 24] attributes it to increased permeability of the capillary walls. SHERLOCK [44, 45] ascribes an important role to VDM (vasodepressor material). It has been emphasized by BÜCHNER [5] that the parenchymal cells are more sensitive than the capillary endothelium and thus more easily damaged by a lack of oxygen; he regards the alteration as the result of disturbed cell metabolism.

We encountered empty vacuoles or such containing protein in all of the examined cases. The number of vacuoles was, however, small in most of our cases (in 20 out of a total of 32), and they were confined to the area adjacent to the necrotic foci; therefore, we do not regard their occurrence as the most significant, in any case not as significant as it appears to be in animal experiments. It is quite possible that our patients did not die of the hypoxia suddenly developing in the acute phase of shock, *i. e.* their death did not occur in that phase which coincides with the development of experimentally-induced vacuolar degeneration.

As long ago as 1907 did RÖSSLE [42] suggest that the central hepatocellular fatty change was of hypoxic origin. This kind of degeneration was observed by LEWINSTEIN [26] and BALÓ [3] in animals exposed to low atmospheric pressures. Recent investigations support the view that acute hypoxia causes centrilobular fatty degeneration (usually with large fatty vacuoles), a lesion attributed by BÜCHNER [5] to the inhibition of ATP-formation.

In 12 of our cases could we observe centrally-situated large fatty vacuoles, but they were widespread in 5 cases only. In the rest there were minute fatty vacuoles in the liver cells, and their number was not proportional with the gravity of liver injury. The possibility cannot be excluded that fatty degeneration may in these cases have been due to hypoxia that was in no connection with the disease immediately preceding death.

It was in cases of protracted oxygen deficiency that MARTIN *et al.* [32], ZALKA [51], CAMPBELL [9] and LOEWY [29], and in oxygen deficiency of short duration that PICHOTKA [39] observed hepatocellular necrosis. On the evidence of autopsy material, HESSE [20] and MOON [33] also ascribed the necroses to the lack of oxygen.

The necroses vary in extent. According to ALTMANN [1], necrosis of single cells is characteristic of moderate hypoxia. Necrosis extending to many cells is usually centrolobular, a phenomenon due to the special circulatory features of the liver, the area around the central vein being the least supplied with oxygen. Zonal necrosis has been interpreted by RÖSSLE [42] and CAMERON [8] as due to the fact that not all capillaries are simultaneously

open and further that the portal vein and the hepatic artery drain into the sinusoids separately (OLDS and STAFFORD, 37). ELIAS [11] even supposes that the arterial capillaries may reach the centre directly.

It was after administration of thyroxin that GERLEI [16], and after that of tannic acid that KORPÁSSY [25] observed centrilobular necrosis. Toxic agents elicit vasospasm according to HIMSWORTH [21]. Poisons responsible for hepatocellular necrosis induce, according to LICHTMAN [28], first a temporary constriction and then a dilation of the hepatic vessels. Other authors suggest that centripetal circulation were impeded by the swelling of marginal and midzonal cells and this would lead to necrosis.

SHERLOCK [45] in biopsy and autopsy specimens of livers from cases of acute heart failure observed a peripheral spreading of necroses. She suggests that circulatory disturbances responsible for liver injury depend on the following factors: (i) cardiac output; (ii) arterial oxygen saturation; (iii) portal pressure; (iv) duration of the process.

*

We observed necrosed foci in all of our cases. Minute and mostly central necroses were found in Group I. Necroses in Group II were likewise central, but more widespread, often sectoral; the adjacent necrotic foci were sometimes coalescent. The necrotized area contained blood pools in 6 cases. In Group III it was combined with chronic congestion that widespread necroses developed, almost invariably with central blood pools. Necrosis was the most characteristic hepatic lesion in our material, presumably because the patients had died after a longer phase of hypoxia: death was always preceded by a state of shock that lasted a day at least. We, too, attribute the necroses to oxygen deficiency induced by circulatory disturbance.

The question arises as to what happens to the described changes after transitory shock. As regards the vacuoles, TROWELL [49], ALTMANN [1] and others think that the cells are capable of so to say isolating the invading fluid in the form of droplets. The protein may later be ejected from the vacuoles and is then secreted with the bile (albuminocholia), a phenomenon observed by FRANKE and SYLLA [15] under the fluorescence microscope. At autopsy of patients who died of myocardial infarction CLARKE [10] observed disappearance of the necrotic foci a month after infarction. The observation made by BYWATERS [7] and SHERLOCK [44] after traumatic shock is indicative of parenchymal regeneration. In ALTMANN's animal experiments [1] the number of mitoses following shock was, however, not sufficient to indicate cellular replacement, wherefore he explained the regeneration process by endoamitoses. We observed a great number of dividing cells in one of our cases only, and are inclined to agree with ALTMANN as regards amitoses. In favourable cases, the preservation of the reticulin framework offers a possibility of complete

parenchymal regeneration. Studying the reticulin in convalescing cases, SHERLOCK [45] observed condensation and proliferation. We saw proliferation of the reticular fibres only in a few cases belonging to Group III; they might have rather been due to the prolonged congestion.

We analyzed the kidneys in 23 cases, and discovered morphological changes in 11 of them. Taking into account the clinical and laboratory data, renal dysfunction had been diagnosed in 9 cases: it had manifested itself with oliguria, a rise of N. P. N. and, in cases 8 and 32, with uremia. In these last two cases, the clinical symptoms were well justified by the grave injury similar to that observed by one of the present authors in connection with shock [12]. This would mean that acute circulatory failure often affects the kidneys as well. The occurrence of lesions is not so frequent in the kidneys as in the liver, but if they do occur, they mean an extremely grave, often fatal, complication.

Many authors are of the view that the disturbed permeability of the sinusoid wall plays a decisive role in the origin of pathological changes. EPPINGER [13] speaks of "Albuminurie ins Gewebe" and RÖSSLE [42] of serous hepatitis. Dyshoria has been the term applied by SCHÜRMANN and MACMAHON [43] to the pathological alternation of the subendothelial basement membrane: they attributed parenchymal damage to the histotoxic action of the leaking plasma. The primary role of these changes has been questioned.

ALTMANN [1] and POPPER [40] regard the appearance of protein-rich fluid in the dilated spaces of Disse a terminal-agonal phenomenon. We, too, had occasion to observe this phenomenon, especially when the terminal phase was protracted; we do not think, however, that the lesion of the vessel walls precedes, and is more important than, the damage of the sensitive parenchymal cells.

BÜCHNER *et al.* [5, 6] and BECKER [4] were chiefly responsible for introducing hypoxydosis, a new pathogenic principle: it is by a reduction of the cells' biological oxidative capacity that the theory of hypoxydosis tries to explain those morphological and functional changes which are observed in shock. Hypoxydosis may be of three kinds: 1. hypoxic hypoxydosis when, for various reasons, there is not enough oxygen available to the cells; 2. hypoxydosis caused by a want of oxidable substances ("Substratmangel"); 3. dysenzymatic hypoxydosis due to a damage of the cells' enzyme system. There exists, according to the said authors, a correlation between histological changes and the duration of hypoxia. While the suddenly arising, milder form of hypoxia causes vacuolar degeneration of the reversible type, gradually developing hypoxia is followed by fatty degeneration, whereas grave hypoxia of long duration induces cellular necrosis.

As regards our own material, we attribute the observed hepatic damages to acute deficiency of oxygen caused by acute circulatory disturbance, no

matter by what it is released. It is the special blood supply of the liver which we regard the decisive factor in pathogenesis. This is why alterations appear first — and in the gravest form — in the centre of the lobules, the area with the weakest blood supply. The observed alterations bear a certain resemblance to the picture of nutmeg liver concomitant of cardiac failure. While the nutmeg liver is characterized by a process of gradual congestion and a slow development of parenchymal atrophy, our material was dominated by suddenly arising and grave cellular damage. Although we examined the liver in all cases where death had been preceded by a state of shock, only about 60 per cent showed a characteristic histological picture. That some 40 per cent of the said cases were negative from this point of view may, in our opinion, have been due to the fact that — apart from the duration of the shock and the gravity of hypoxia — some regulative mechanisms played a major or minor role in the origin of the pathological changes. None of our numerous other cases showed a similar picture; this argues for the specificity of the alterations and justifies the use of the term "shock liver". It deserves comment that hepatic lesions, however serious and widespread they were, never manifested themselves with clinical symptoms. This may partly be due to the great reserve power of the liver and partly to the fact that the patients died before the hepatic damage had time to influence the clinical picture. It was obviously due to the great regenerative power of the liver that no permanent trace or chronic form of the discussed alterations was observed in any case of our material.

Summary

1. Pathological changes of the hepatic tissue, to which the collective term "shock liver" may be applied, were observed in 32 subjects who had died with secondary shock, acute abdomen or cardiac failure.

2. The cases were divided into four groups: I. slight lesions with few necroses of small size; II. typical cases with extensive necroses; III. alterations accompanied by chronic congestion; IV. alterations accompained by cirrhosis.

3. Protein vacuoles and fatty degeneration were not general, probably because death was usually preceded by hypoxia of longer duration.

4. Centrilobular necrosis, often with hemorrhages in the necrotized area, was the most characteristic lesion observed.

5. We attribute the observed pathological changes to hypoxia caused by acute circulatory disturbance, and suggest that their centrolobular position is determined by the special blood supply of the liver.

6. The absence of clinical symptoms is obviously due to the liver's great reserve power, and complete regeneration may be expected whenever the patient is able to survive the state of shock.

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ИЗМЕНЕНИЯ ПЕЧЕНИ В СВЯЗИ С ВТОРИЧНЫМ ШОКОМ И ОСТРОЙ НЕДОСТАТОЧНОСТЬЮ СЕРДЦА

А. ХАРАСТИ и П. ЭНДЕШ

1. На умерших вследствие вторичного шока, острой брюшной катастрофы и острой недостаточности сердца авторы наблюдали в 32 случаях тканевые изменения печени, которые можно объединить под термином «шоковая печень».

2. Эти случаи можно распределить на четыре группы: I) случаи со слабыми изменениями, с некрозами небольшого распространения, II) типичные случаи с обширными некрозами, III) изменения, развивавшиеся при хроническом застое и IV) изменения при циррозе печени.

3. Белково-капельное вакуольное перерождение и жировое перерождение на данном материале оттесняются на задний план. Это можно объяснить тем обстоятельством, что вскрытие, как правило, состоялось после длительной гипоксии.

4. Самым характерным изменением в материале оказался некроз в центральной дольке. В этой области часто наблюдались также кровотечения.

5. Изменения объясняются недостатком кислорода (гипоксией), сопровожденном острым расстройством кровообращения, а их локализация — своеобразными условиями кровоснабжения печени.

6. Отсутствие клинических симптомов предположительно связано с значительной резервной силой печени и в случае выживания после вызывающего изменения шокового состоянии можно считаться с полной регенерацией.

LEBERVERÄNDERUNGEN BEI SEKUNDÄREM SCHOCK UND AKUTER HERZINSUFFIZIENZ

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1. Bei mit sekundärem Schock, akutem Abdomen- und chronischer Herzinsuffizienz Verstorbenen wurden in 32 Fällen Gewebsveränderungen in der Leber wahrgenommen, die unter dem Namen Schock-Leber zusammengefasst werden können.

2. Diese Fälle wurden in vier Gruppen unterteilt: I. Fälle mit geringen Veränderungen, mit wenig Nekrosen geringer Ausdehnung, II. typische Fälle mit ausgedehnten Nekrosen, III. Fälle in denen die Veränderungen mit chronischer Stauung und IV. mit Zirrhose vergesellschaftet waren.

3. Vakuoläre Entartung infolge Entstehung von Eiweisstropfen, sowie fettige Degeneration war im Material nicht ausgeprägt, was damit erklärt werden kann, dass die untersuchten Fälle meist nach längere Zeit bestehender Hypoxie zur Sektion gelangten.

4. Als typischste Veränderung erwies sich im Material die zentrolobuläre Nekrose, häufig mit Blutungen.

5. Die Veränderungen werden auf die mit der akuten Zirkulationsstörung einhergehende Hypoxie zurückgeführt, und ihre Lokalisation mit den eigenartigen Blutversorgungsverhältnissen der Leber erklärt.

6. Das Fehlen von klinischen Symptomen hängt offenbar mit der grossen Reservekraft der Leber zusammen, und wenn der Schockzustand überlebt wird, kann mit einer vollkommenen Regeneration gerechnet werden.

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