be generally expected of it, and that is a declaration that it is aware of the facts and condemns them.

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Asymmetrical Hepatic Fibrosis in Children

Disease in the adult liver may show a patchy distribution, and this is generally explained in terms of the fixed channels of portal blood flow from which different segments of the liver receive blood from different abdominal viscera. In infants, too, pathological lesions of the liver may vary in their distribution.

The liver can be divided into two lobes on a physiological basis. The physiological left and right lobes are delineated according to their blood supply,[1 2] and their junction differs from that of the anatomical left and right lobes in that the line of demarcation joins the inferior vena cava to the gall bladder. In the fetus a considerable amount of placental blood passes via the portal veins of the left lobe to the right side of the heart, so that the left of the liver receives highly oxygenated blood. At birth this supply is suddenly withdrawn, and the left lobe is henceforth perfused by the poorly oxygenated blood derived from the portal venous drainage of the abdominal viscera. It is thus to be expected that this particular part of the liver would be susceptible to the effects of noxious agents during the first few days of life[3] and that degenerative changes of the parenchyma terminating in fibrosis would be commoner than in the right lobe. The connective-tissue content of children's livers is higher in proportion to the parenchyma than it is in the adult organ, and it is higher in the left lobe than in the right.[4]

Recently M. L. Ghosh and J. L. Emery have investigated the distribution of connective tissue in the two lobes of the liver in 150 children who died between birth and 12 years of age in varying circumstances.[5] In 35 cases they could find no increase in connective tissue in the liver as a whole or separately in the right or left lobes. But in the other 115 cases the connective tissue content was above normal, and the left lobe was the more severely affected. In view of the distinct change in the blood supply that the left lobe undergoes shortly after birth, Ghosh and Emery tried to correlate the presence of fibrosis with a history of hypoxia in the perinatal period. It could be inferred that any degree of hypoxia sufficient to produce a lesion would produce cyanosis of a clinically noteworthy intensity. They found that in those babies with a normal amount of connective tissue in the liver 80% had no history of cyanosis round the period of birth, whereas only 27% of those with an increased amount of connective tissue were without perinatal cyanosis. The majority of babies with an increased amount of connective tissue in the left lobe suffered from prolonged perinatal cyanosis, while less than a tenth of those with a normal amount of connective tissue were similarly affected.

The causes of the hypoxia in these cases varied. The most frequent were cyanotic congenital heart disease, coarctation of the aorta, acute respiratory distress, abnormalities of the central nervous system, and surgical abdominal emergencies with postoperative shock. The incidence, degree, and duration of the hypoxia were correlated with the amount of connective tissue in the liver.

This study emphasizes the importance of taking sections from both lobes of the liver for routine postmortem histological examination, especially when there is hepatic fibrosis. It is possible that some cases of fibrosis of the liver in young children which were attributed to congenital dysplasias were in fact examples of fibrosis secondary to hypoxic destruction of liver parenchyma. It is certainly important to inquire about the perinatal state of any child whose liver shows an increased amount of fibrous tissue.

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[3] Emery, J. L., *Journal of Pathology and Bacteriology*, 1955, **69**, 219.
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Fall-out from Bomb Tests

The recent French atomic explosions at Mururoa have created a storm of public protest based mostly on the same considerations of radiation safety that were used to promote the 1963 treaty banning atmospheric tests of nuclear devices: namely that the radioactive nuclides of fall-out, like any other source of ionizing radiations, would cause malignant disease, fetal abnormalities, and hereditary genetic damage in those exposed to it. The risk of genetic damage used to attract most attention, but recent assessments suggest that radiation-induced cancer is more important.[1 2]

Fall-out after an explosion of a nuclear device may be deposited locally or globally. The force of a high-yield explosion takes fission products up into the stratosphere, from which they are distributed all over the world as global fall-out. The important components are longlived nuclides, especially caesium-137, in comparison with which strontium-90 is now recognized to be less damaging. Radioactive material from a low-power explosion remains in the troposphere below the stratosphere and is therefore deposited locally. Shortlived fission products, such as isotopes of iodine, may then be the main source of radiation dose to the tissues. The radiation doses from tropospheric fall-out received by the Marshall Islanders in 1954 were large enough to cause acute radiation effects and thyroid disease (both endocrine deficiency and cancer), but this was the result of special circumstances never likely to occur again. When, as in the recent French tests, there is a clamp-down on information, no one can know for certain what the radiation dose from local fall-out was to people near Mururoa or elsewhere in Polynesia, but it is likely to be well within the dose limits recommended by the International Commission on Radiological Protection for exposure of workers and the general public to controlled sources of radiation.

Each high-yield explosion produces far more radioactivity than a whole series of small explosions as well as distributing it stratospherically. The fission yield of the most recent French tests may not be known yet. But, taken together, all atmospheric tests add up to about 200 mega-tons fission yield, and so far the French are responsible for about 6 megatons total yield (and the Chinese 15 megatons).[3 4] The United Nations has sponsored comprehensive and detailed reports on fall-out[1] but has omitted any estimates of the individual contributions of particular countries. However, the data suggest that some 1-2% of the total radiation dose resulting from all stratospheric fall-out could be attributed