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 in some cases 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 At birth this supply is suddenly withdrawn, and the left lobe is henceforth perfused by the poorly oxygenated blood derived from the portal venous drainage of the abdominal viscera.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 life³ 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 liver of 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.

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.⁶ ⁷

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 long-lived 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. Short-lived 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).⁸ ⁹

The United Nations has sponsored comprehensive and detailed reports on fall-out¹⁰ 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
to the French tests. In round terms this would be 3 milli-rads, or 3% of one year's unavoidable exposure to natural background.

For the purposes of radiation protection it is assumed, but not proved, that any dose of radiation, however small, has some finite probability of doing harm. We cannot even say how much injury natural background radiation causes, and some might claim it causes none. The only way to reach some estimate of casualties is to pick a numerical coefficient for the risk of cancer (or other effect) per unit radiation dose, and calculate from that. The evidence from Japanese bomb survivors, who received 50-500 rads, and from antenatal diagnostic exposures at a mean dose of 2-3 rads, may suggest a cancer risk of 1 in 10,000 during a lifetime from a dose of 1 rad continuing, as it would with fall-out, over a lifetime. We may assume, but certainly cannot prove, that the risk is exactly 1,000 times less for a dose of 1 millirad. If we do assume this, a world population of 4,000 million people each exposed to 3 millirads would experience 1,200 additional radiation-induced cancers. But this excess could never be demonstrated in the face of the 3 million cases which the same calculation would show to be the result of a lifetime's exposure to natural background radiation, or of the 1,000 million cancers occurring "spontaneously." Moreover, if the assumption that cancer risk is simply proportional to dose is not true, the numbers of radiation-induced cancers could be overestimated by a 100-fold or 10,000-fold or more.

Tropospheric fall-out of shortlived radionuclides in Australia after the French tests at Mururoa in 1970, 1971, and 1972 gave external doses of about 1, 1, and less than 0-1 millirad respectively. 1-7 A similar risk calculation based on simple proportionality for the Australian population of 12-13 million would suggest that this fall-out from the three successive series of tests would cause 1, 1, and zero cases of cancer respectively. The stratospheric fall-out from the same series of tests would be likely to cause a similar number of cancer cases in Australians.

All attempts to calculate risk or numbers of casualties from small doses of radiation, such as fall-out, or from the individually larger doses received in occupational exposure or radiological diagnosis, suffer from the same lack of a firm scientific foundation for ideas about the relationship between dose and risk and the same inability to make an observational check on the accuracy of conclusions. These uncertainties make it fruitless to argue too heatedly about the significance of quite sizeable differences in fall-out dose. Indeed quantitative assessments of numbers of casualties expected from radioactive fall-out, taken in isolation, seem to provide an inadequate argument against bomb tests. The important arguments relate to the benefits which justify radiation exposures, including medical and industrial. All radiation exposures should be justified by the expected benefit. 8


Screening for Glaucoma

Between 1964 and 1966 screening for glaucoma was undertaken in Bedford, when 5,941 people over the age of 40 were examined. 1 People with a tension of 21 mm Hg or greater and those with ophthalmoscopically abnormal optic discs were referred for more detailed study. Fifty-five cases of primary glaucoma were detected, a total incidence of 0.93%, of which 0.71% represented the incidence of chronic simple glaucoma and low-tension glaucoma combined. A further 212 persons with equivocal or suspicious signs were advised to have an annual re-examination.

E. S. Perkins 2 has recently reported on this last group, followed up over a period of five to seven years. A quarter failed to attend regularly and half have been discharged as showing on further study no evidence of glaucoma. Five cases of primary glaucoma (2.63%) were discovered, and there remain 30 persons who are still under surveillance, two of whom may prove to suffer from chronic simple glaucoma.

The question posed on designing this survey was to what extent ocular hypertension as an isolated finding is a precursor of chronic simple glaucoma in view of the fact that 8% of the population have tensions of 21 mm Hg or more. Of the 124 people with ocular hypertension followed up over seven years only four have developed chronic simple glaucoma, an incidence of 3%.

The results of this follow-up study suggest that the risk of field defects developing in persons with moderate ocular hypertension over a seven year period is small, and treatment may legitimately be withheld until glaucomatous field changes declare themselves. But two further points should be kept in mind. If ocular hypertension is associated with suspicious signs at the optic disc, there is more likelihood of the development of glaucoma. Furthermore, the higher the tension on screening, the greater the probability of its development. 3

Perkins 4 has also reported on a sample follow-up study, planned with a bias towards the older age group, of those persons who were dismissed as normal during the period 1964 to 1966; 770 individuals were re-examined. The most striking finding was the constancy of the tonometric readings after an interval of seven years in the great majority. Twelve of this group were found on their second examination to have tensions of 21 mm Hg or above, two of whom were thought to be cases of chronic simple glaucoma. The Globuck screener disclosed 23 cases with field defects from various causes, two of which proved to be glaucomatous. There was a fifth case of probable chronic simple glaucoma in the 770 rescreened, making a total incidence of 0.52%.

From these findings it is reasonable to question the widely held belief that raised tension precedes glaucomatous field defects probably by a decade. 5 They can certainly develop in a much shorter time, and evidence is accumulating to suggest that they arise suddenly and increase by steps. 6 The practical conclusion is that little weight should be given to a negative screening test carried out by present techniques. Nevertheless a change in tension to a higher level over a period of years, just as a change in extent of cupping of the optic disc with time, 7 must always be considered to be a likely indication of the early development of chronic simple glaucoma.

Another point emerges from this study. Two out of 15 persons found to have suspicious discs and normal tensions...