Editorial: Ocular effects of ionising radiation: past and present problems

At the turn of the century the harmful effects of ionising radiation had been recognised (Birch-Hirschfeld, 1904). Information had been obtained from the use of x rays in the diagnosis and treatment of human disease, and this was soon supplemented by a study of the adult rabbit after exposure to ionising radiation of ill defined strength. Initially it was thought that the chief ocular effects of such radiation were confined to the skin of the lids, the cornea, and conjunctiva.

Since then a better understanding of the biological effects of radiation has been achieved by greatly improved methods of measuring the dosage and a realisation that, apart from the type, the age of the tissue is also very important. As a result a number of theories have been evolved to account for these effects and also to explain the usually considerable time lag between exposure and biological change. Briefly these theories are of 2 types and postulate either a direct or an indirect action on cells. The first group of theories, 'the direct hit theory', presumes that radiation has a direct action on the large protein macromolecules of the cell and alters them in some way. But the relative importance of the nucleus and the cytoplasm in these processes is debated. The second group of theories presumes that the change is initially in the cellular water, the radiation providing free radicles and their subsequent reaction with cellular enzymes producing harmful effects. Some credence to this idea is given by the fact that compounds such as cysteine which react readily with free radicles can protect animals to a limited extent from the ill effects of radiation. The delay is more difficult to explain, and any explanation is usually linked with the high sensitivity of rapidly growing tissue, selective harmful effects being only fully revealed when cells attempt to replicate. Thus if growth is slow the harm is long delayed.

This delayed action of ionising radiation is well illustrated by its effect on the conjunctiva and cornea. In the conjunctiva there is an initial acute reaction, with hyperaemia, oedema, and often a mucopurulent discharge. This settles down after a few weeks, but after months or years intense scarring is produced, often with symblepharon, while the blood vessels show telangiectasia. The cornea initially responds with a punctate keratopathy, which usually persists for months and is unresponsive to treatment. Eventually large ulcerated areas may suddenly appear, with a gross loss of corneal substance. After this repair is usually imperfect and slow.

The action of ionising radiation on the lens was thought to be negligible because experiments conducted on the adult rabbit at first failed to produce a cataract. It is now known that the lens of the mature animal is much more resistant to radiation. The production of cataract by this means was confined to animals in utero. However, since the epithelium of the lens, like epithelium elsewhere, continues to divide throughout life, it would be expected that the lens would also show changes which indicate that it is sensitive to ionising radiation.

Again, the effect was missed at first because it is delayed by months or years when exposure is with rays of moderate intensity—the only type available to early investigators. Since the part of the lens affected by cataractous change is the posterior pole, it could be argued that the effect is an indirect one and perhaps relates to the nutrition of the eye as a whole.

In contradiction to this, it has recently been shown (Fisher and Wakely, 1976) that the probable cause of this posterior cortical response to injury in general is the unusual growth properties of lens cells originating from the front of the lens. At the end of the second world war owing to the exposure of victims to intense gamma rays from atomic bombing a series of reports (Cogan et al., 1952) appeared on the acute effects of intense radiation of the lens. These reports indicated that the long known posterior cortical changes were intensified and occurred within 6 months to 2 years. The periphery of the cataractous area was particularly dense, and a so-called doughnut-looking opacity was produced. At this stage the cataract might remain quiescent or become opaque and mature within a few months.

In clinical practice cataract has been produced when it has been difficult to shield the eye from cobalt beam or x-ray therapy, these procedures being necessary in the treatment of carcinoma of tissues in or around the orbit (Lederman, 1964). These cataractous lenses, when they have been examined histologically, have usually been mature. Such changes as have been reported indicate
metaplasia of the lens epithelial cells, with the production of swollen bladder-like cells or cells which resemble fibroblasts.

From considerations of public health, however, the effect of low-dosage, possibly intermittent radiation is more important. This aspect of the problem appears to have been neglected because of the difficulty of having reliable evidence that the cataract, when present, has been caused by such exposure.

A paper in the present issue (page 457) attempts to provide a small amount of this necessary information. Again, exposure to low dosage radiation results in a very long latent period—some 25 to 30 years before a cataract of sufficient magnitude develops to impair vision seriously. It has been possible to examine such lenses by the slit-lamp and electron microscope, and 2 additional responses of the lens substance are reported in this paper.

Firstly, after the initial exposure of some years’ duration there was subsequent recovery and the laying down of clear and apparently normal lens fibres. Eventually, when the function of the lens epithelium began to fail, a posterior cataract opacity with unremarkable features developed. Secondly, failure of function of the lens epithelium was characterised by a vertical rather than horizontal grouping of cells in relation to the overlying capsule, while smaller disorganised cells were not found.

At present the importance of radiational injury of the lens as a hazard in certain occupations is not clearly understood. There is no doubt that intense radiation in the region of the eyeball usually produces cataract, but it does not follow—as is commonly supposed—that intermittent low-dosage exposure is ultimately as dangerous. Similar remarks could equally apply to the present state of our knowledge in regard to ultrasonic radiations.

To overcome these difficulties it is necessary to link 2 groups of workers matched for age, one group who are exposed to such intermittent risks, and the other group who have a similar environment in other aspects but who are not so exposed. In the case of ionising radiation, workers in nuclear power installations are probably the only choice. In microwave radiation such a study could be undertaken among armed Forces personnel who service radar equipment and those who do not. Such a study would enable an estimate of the true risk of low-dose, intermittent, and long-duration radiation to be made. Unfortunately at present, without such a study, the hazard remains undetermined.

References