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Čerenkov Light from ^{32}P as an Aid to Diagnosis of Eye Tumours

THE ^{32}P uptake ratio test[1] as an aid to tumour diagnosis has had limited success for tumours in the posterior half of the eye[2–4]. Phosphorus is incorporated preferentially in dividing rather than non-dividing cells[5] and so, in essence, all the test requires is the oral or intravenous administration of ^{32}P orthophosphate (typically 100–500 μCi) and the β radiation from the lesion is measured and compared with that over the surrounding normal tissue.

In practice the test is complicated mainly by two factors. Inflammatory and certain other actively metabolic lesions will also incorporate ^{32}P in preference to normal quiescent tissue; and because β radiation has a short mean path length (3 mm in tissue), the placement of the detector is critical.

The problem of inflammatory lesions is largely overcome by spacing two observations of uptake to 4 and 24 h after administration of the isotope[4,6]. The inflammatory and metabolic components of uptake at 4 h greatly decrease by 24 h because of the rapid turnover of phosphorus in these processes. Thus one criterion reported for a positive diagnosis of malignancy[6] is that the uptake at both 4 and 24 h with respect to surrounding normal tissue has to be greater than a ratio of 1.5 : 1.

The placement problem is particularly acute when the test is applied to lesions posterior to the equator of the eye, for which surgery of access is most often necessary. Most eye tumours arise in this region in the choroid.

However, it is possible to detect the presence of ^{32}P in the back of the eye by observing the Čerenkov light produced by the passage of the β radiation in the vitreous humour. This can be done quite simply through the pupil using a highly sensitive photomultiplier tube.

From the theory of the Čerenkov effect it may be shown that the average radiation from ^{32}P in water will produce about thirty visible photons[7] per emitted β particle, and this has been used with transparent solutions of ^{32}P-labelled compounds in liquid scintillation counters[8].

I have investigated the use of this effect as a simple diagnostic test for the presence of ^{32}P in the posterior half of the eye. A specially selected bi-alkali type of photomultiplier tube was obtained (Centronic type 4249). This has a background emission of only 3 electrons/s from the cathode. It was coupled to a short length of 'Perspex' light pipe which was positioned over the eye of an anaesthetized rabbit, in contact with the cornea. When 0.8 mCi of ^{32}P was injected intravenously into the medial ear vein a signal of 400 pulses/s was produced from the photomultiplier tube. After a few minutes observation, a 0.0005 inch layer of black tape was placed over the end of the light pipe. The count rate fell to 80 pulses/s, which represented the fraction of Čerenkov light created in the light pipe by primary β irradiation. Thus the bulk of the signal detected by the photomultiplier tube can be attributed to Čerenkov light emanating from the vitreous humour as a result of the circulation of ^{32}P through the highly vascular choroid.

This test for Čerenkov emission should, therefore, provide a sensitive means of readily detecting and quantitating ^{32}P incorporated by ocular lesions and its applicability to the diagnosis of human ocular melanomata is planned.

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Transfer of Blastocysts as Applied in Experimental Teratology

IT is well known that genetic constitution modifies the effect of teratogenic agents (Table 1). The most obvious example is the varying susceptibility of different strains of mice to teratogens such as cortisone, 6-amino-nicotinamide and salicylates[1–5]. In A/Jax mice, for example cortisone induces cleft palate in 100% of living embryos (if those with spontaneous cleft lip-palate—about 10% of births—are excluded). Isolated cleft palate can be induced with cortisone in only about 20% of embryos in other strains such as C57Bl and CBA[1,3].

Reciprocal cross differences demonstrated in the frequency of cleft palate induced by cortisone[3] or foetal death induced by salicylate[5] are matroclinous; the maternal influence could be chromosomal, cytoplasmic or uterine (Table 1). The maternal chromosomal effect would be transmitted with the *X* chromosome and the effect would thus be seen in the males, but cleft palate induced by cortisone is distributed equally among male and female embryos[3]. A cytoplasmic factor seems to be responsible for the differing susceptibility to 6-amino-nicotinamide in A/Jax and C57Bl mice[6], but Kalter could not demon-

Table 1 Genetic Factors modifying the Effect of Teratogenic Agents

Factors	Revealed by
1 Strain difference	Reciprocal crosses
2 Paternal influence	A × B
	B × A
3 Maternal influence	
(a) chromosomal	Increased risk in males
(b) cytoplasmic	Back crosses
	(A × B) × A
	(B × A) × A
(c) uterine	Egg transfer