A mathematical analogue to calculate the counts from $^{32}$P in the choroidal blood during the $^{32}$P uptake test

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SUMMARY A mathematical model has been developed to assess the contribution of $^{32}$P in the blood in the choroidal vessels to the count obtained from the eye during the $^{32}$P uptake test. Applying the model at 48 hours after the administration of $^{32}$P it was found that, at this time, between 4% and 7% of the control count on the eye was due to $^{32}$P activity in the choroidal blood.

In a previous paper the authors described their experience of the $^{32}$P uptake test in the diagnosis of intraocular malignancies and discussed some of the factors affecting the uptake. The $^{32}$P test is based on the differential accumulation of radioactive phosphorus between neoplastic and non-neoplastic tissue following an intravenous injection of the isotope. Counts are recorded from a site on the surface of the eye, overlying the lesion, and from a control site on the eye. The $^{32}$P uptake is the difference between the test and control counts expressed as a percentage of the control count. In a recent review of the literature threshold values for a positive test were shown to have varied between 30 and 150% according to different investigators, but now a figure of around 60% is most frequently used. Several investigators have discussed the reliability of the test and stressed the need for accurate localisation of the probe. Various matters such as the rate of phosphorus uptake in uveal melanomas and the relation between $^{32}$P counts and histological features have been investigated.

An aspect of the $^{32}$P test which has received little attention relates to the source of the control counts. An uptake of 100% for example implies that there are half as many counts recorded over control tissue as from a site which overlies a malignant tumour. The counts from $^{32}$P in normal tissue are therefore not negligible in comparison with those from a malignant tumour. The activity recorded on the surface of the eye depends on the distribution of isotope in the tissue below the detector. A knowledge of this distribution is important, and one aspect which should be investigated is the extent to which the control counts are due to radioactivity in the blood. It is important to know to what extent blood borne $^{32}$P affects the count on the sclera so that due regard may be given to vascularity. It is, however, difficult to assess the contribution of the vascular component to the control counts and to facilitate this a mathematical model has been developed to estimate the effect of the $^{32}$P activity in the choroidal blood on the control counts.

Materials and methods

A brief outline of the method may help in the understanding of the use of the model. Blood is withdrawn from an antecubital vein from a patient who has received $^{32}$P, and the sample is counted by placing the same probe as used on the eye in contact with the blood in a suitable chamber. It is assumed that the concentration of $^{32}$P in the blood is uniform throughout the body, so that the concentration of isotope in the blood sample is the same as in the blood in the vessels of the choroid. It is further assumed that the attenuation of $\beta$ particles is the same in ocular tissue and in blood. The count recorded from the blood sample may be thought of as being derived from a series of bands at different levels within the sample. The contribution of each band to the total count recorded from the blood sample is obtained from the model. One of the bands in the blood sample is at the same distance from the probe as the choroid in the eye. The model determines the fraction of total blood sample count that is derived from the band which corresponds to the choroid. On the assumption that blood is the major component of the choroid, this...
fraction of the total blood sample count represents the count recorded on the surface of the eye due to the $^{32}$P in the blood of the choroid. This value is compared with the actual control count recorded on the surface of the eye.

The model is 2-dimensional, with elemental areas centered at source points 200 $\mu$m apart on a 35×110 point matrix (Fig. 1). The 200 $\mu$m spacing corresponds to the resolution required in the application of the model to the eye, described below. Each of the points is a source of radiation, and at this stage the model is not a representation of the eye. The detector used clinically is a solid-state probe 8 mm in diameter. The number of columns in the matrix is determined by the overall width of the detector plus a surround which represents the maximum range of $\beta$ emission in the tissue. Thus the detector in the model is represented by a row of detector points above the top row of the matrix from columns 36 to 75. Since the matrix points are 200 $\mu$m apart, the surface is 100 $\mu$m above the top row. The detector points are therefore 100 $\mu$m above the first row of radiation points and are in row 0·5. Each detector point is the center of an element of detection of radiation, and the total length of the detector is represented by the number of detector points used.

Consider an arbitrary unit strength source point in the matrix (a, b) detected by an arbitrary point in the detector (0·5, c) (Fig. 2). The activity which reaches the detecting element depends on 2 factors. The first is the attenuation of radiation as it passes through matter, dependent on the distance and the attenuation coefficient. In the model the attenuation coefficient of water was used. Secondly, since radiation is emitted in all directions, only that radiation which falls on the detector element is measured. Thus the activity which reaches the detector element depends on the degree of attenuation and the fractional angle of pick-up. The equation which describes this is given in Fig. 2. The activity at the detector from the point (a, b) is the sum of the detected activities at all the detector points, and from this an activity number is ascribed to the point (a, b).

In the model, activity numbers were obtained for each of the 35×110 source points, and the total of all the activity numbers was obtained. The equations were solved by means of an IBM 360/165 computer. The ratio of the activity number from any source point to the total of all the activity numbers in the matrix is equal to the fractional contribution from that source point to the counts at the detector from a uniform source distribution.

The model may be applied to the contribution of the $^{32}$P in the blood of the choroid to the count obtained on the surface of the eye as follows. If the sclera is taken as 400 $\mu$m thick and the choroid as 200

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**Fig. 1** Matrix in which points representing sources of radiation are distributed in a 35×110 point matrix with an interpoint separation of 200 $\mu$m. A row of points representing detection points are situated centrally above the source points and combine to represent the eye probe.

**Fig. 2** Equation used to determine the counts at an arbitrary detector point from an arbitrary source point.
μm, the choroidal points are represented by the third row of the matrix. The ratio of the counts derived from the choroid to counts obtained from a uniform source distribution is the ratio of the activity numbers of the third row to the total of all the activity numbers. A uniform source distribution may be achieved by using an arrangement similar to that of Fig. 1 to count blood from a patient who has received 32P, with the eye probe in contact with the blood sample. In 4 patients undergoing 32P tests, 48 hours after an intravenous dose of 500 μCi of 32P, test and control eye counts were measured, and at the same time venous blood was withdrawn from an antecubital vein. These blood samples were placed in a container, and radioactivity was determined by placing the detector in contact with this blood.

Results

The measured radioactivity from blood samples in 4 patients varied from 557 to 837 counts/100 s.

In the model the counts derived from the third row of the matrix, which corresponded to the choroidal layer, comprised 4% of those from a uniform distribution. The third and fourth rows of the matrix each contributed 4% to the total count, so that the exact location of the choroid did not appreciably affect the final conclusions. Thus, the predicted contribution to the control site count derived from blood in the choroid ranged between 24 and 36 counts/100 s (Table 1).

The counts recorded at the control site in the 4 patients ranged between 337 and 707 counts/100 s.

Table 1 Results from 4 patients showing the 32P activity in blood sample, the predicted choroidal contribution to counts recorded on the eye, and counts recorded from a control site in the unaffected eye

<table>
<thead>
<tr>
<th>Counts recorded from blood sample (c/100 s)</th>
<th>Predicted choroidal counts recorded on eye (c/100 s)</th>
<th>Counts recorded from ocular control site (c/100 s)</th>
</tr>
</thead>
<tbody>
<tr>
<td>557</td>
<td>25</td>
<td>707</td>
</tr>
<tr>
<td>557</td>
<td>24</td>
<td>337</td>
</tr>
<tr>
<td>837</td>
<td>36</td>
<td>672</td>
</tr>
<tr>
<td>720</td>
<td>31</td>
<td>634</td>
</tr>
</tbody>
</table>

Discussion

In assessing the normal choroidal thickness in the living eye regard was given to evidence from histology and ultrasound. Histological sections can be misleading in this respect because of blood loss from the tissue before fixation. A figure of around 200 μm for choroidal thickness has been obtained from histological studies. More recently ultrasound measurements in living eyes have shown that choroidal thickness is about 400 μm. If the choroidal blood is regarded as a uniform layer 200 μm thick, 400 μm below the surface of the eye, according to the model, about 6% of the control counts at 48 hours are due to 32P in the blood. The choroid, of course, contains a proportion of extravascular tissue and cannot be considered as being uniformly composed of blood. However, provided there is an accumulated depth of 200 μm of blood between 200 and 600 μm below the surface of the eye, the predicted contribution is not significantly affected. If the choroidal blood were treated as a uniform layer 400 μm thick, separated by 400 μm of tissue from the detector, its contribution would increase to around 11%. Thus, the model predicts that the control counts are not primarily vascular in origin, although an increased choroidal circulation may affect a borderline uptake. The results suggest that some tissue other than blood in the choroid is retaining phosphate, thus accounting for the relatively high uptake levels at control sites.

The mathematical analogue described in this paper is useful because of the paucity of experimental information. It is particularly difficult to obtain a direct measurement of the contribution of blood-borne 32P to the counts recorded on the sclera during an uptake measurement. The model may also be applied to distributions of 32P other than those detailed. The interpoint spacing may be reduced if greater resolution is required provided the number of points is increased to maintain the overall dimensions of the matrix. The model is applicable to other postinjection times provided blood is withdrawn and counted by the eye probe. The application of the model to nonvascular 32P may be made only if the concentration and distribution of isotope in the nonvascular component are known.

References

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