

Average count of 9 pituitary adenomas were 169.4% of normal hemispheric areas, 192.5% in 3 solid craniopharyngiomas, 192.3% in 6 meningiomas and 193.3% in 5 ectopic pinealomas.

Difference of average ratio of lesion count to normal hemispheric count was significant statistically between cystic craniopharyngioma and adenoma, ectopic pinealoma, meningioma, glioma and solid craniopharyngioma, and between adenoma and acromegaly with $p < 0.005$, and between solid craniopharyngioma and acromegaly and between glioma and acromegaly with $p < 0.025$. In the ratio of lesion count to sagittal sinus count, on the other hand, diffe-

rence of average ratio was significant with $p < 0.005$, only between cystic craniopharyngioma and ectopic pinealoma, and between cystic and solid craniopharyngioma.

These facts suggested that sagittal sinus count is unsuitable to be a standard count of an anterior scintigram to compare with basal midline lesion count. Semiaxial anterior view of pertechnetate brain scintigrams proved their clinical diagnostic value for various basal midline lesions which size required craniotomies. Differential diagnosis between solid and cystic lesions of scintigrams are found very helpful in the decision of surgical indications.

A Model Subtracting of Scalp and Skull Isotope Contents from Brain Scans

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In routine brain scans, brain must be viewed through scalp and skull. These superficial tissues interfere with the precise analysis of brain isotope content.

Usually one collimator sees geometrically a demarcated space to determine radioactivity. This space can be splitted into three compartments, that is, scalp, skull and brain.

By using a mathematical expression as below;

$$\begin{pmatrix} \text{Scalp } \gamma_2 \\ \text{Skull } \gamma_3 \\ \text{Brain } \gamma_3 \end{pmatrix} = \begin{pmatrix} A1 & B1 \cdot V1 & C1 \cdot V1 \cdot U1 \\ A2 & B2 \cdot V2 & C2 \cdot V2 \cdot U2 \\ 1 & V3 & V3 \cdot U3 \end{pmatrix}^{-1} \begin{pmatrix} \text{Total } \gamma_1 \\ \text{Total } \gamma_2 \\ \text{Total } \gamma_3 \end{pmatrix}$$

We can calculate three isotope contents from

the compartments.

In an energy spectrogram, 99m Tc pertechnetate demonstrates total γ_1 count (18 kev X-ray) and γ_3 (140 kev gamma ray), but does not present γ_2 . This isotope limits us to use the items which have suffix "2" in the matrix, so that we can determine only scalp and brain γ_3 counts.

Under an experimental situation, 13 human external head counts were obtained. The results were that $36 \pm 3\%$ of total count originated from scalp and when brain has same a unit volume as blood in the calculation then $20 \pm 2\%$

of blood count came from brain. This figure was larger than the expected value which should be less than 10% because of our previous animal experiments.

Half value thickness of ^{99m}Tc 140 keV gamma ray is 4.6cm in water and is 3.8 cm in bone. A breadth of a human head is maximally 17.5cm (scalp, 1.0 skull, 1.2 brain, 15.3cm) with a lateral view. We found that

the brain count was contaminated with the scalp count in opposite side.

A mathematical model which can discriminate less than 3 tissues from an external count, is demonstrated.

But a three compartmental model which is consisted of superficial tissue, brain and again superficial tissue, could be more profitable.

The Study of the Abnormal Brain Scan (^{99m}Tc -pertechnetate) in the Cases of Vasospasm after Ruptured Intracranial Aneurysm

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ECKER and RIMENSCHNEIDER (1951) supplied angiographic evidence of some important cerebral arteries in ruptured aneurysms localized in the circle of Willis.

Since then numerous workers have considered that vasospasm is the main cause of mortality in patients with ruptured intracranial aneurysms. Many workers have described, in fatal ruptured intracranial aneurysms, there are many cases with massive cerebral infarction, and mechanical obstruction by thrombosis, embolism, or atherosclerosis is not considered to be significant in producing in producing in infarcts. They have suggested the vasospasm contributes significantly to the production of the cerebral infarction.

We presented 5 cases to show that the cerebral vasospasm accompanying subarachnoid hae-

morrhage can be associated with abnormal brain scans, just as cerebral infarction.

These abnormal R.I. (^{99m}Tc -pertechnetate) accumulation occurred in the distribution of one or two of the major cerebral arteries approximately 2 to 25 days after the onset of the neurological deficits. In one of 5 cases, gross and microscopic examination appeared ischemic softening, without mechanical stenosis of major cerebral arteries, suspected to result from cerebral vasospasm. In other 4 cases, cerebral like findings without haematoma in the area of abnormal R.I. accumulation were found in the gross examination under macroscopic operation.

This study was carried out in these cases to confirm the relationship between the areas, or density of abnormal R.I. accumulation, the