Delayed radiation necrosis of the brain

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The cases of six patients are presented to delineate the clinical profile of delayed radiation necrosis of the brain. In five the diagnosis was verified histologically. Symptoms most often begin 9 months to 2 years after radiotherapy. Progressive visual impairment and dementia are common following perisellar irradiation, while hemispheric signs predominate following irradiation of the cerebrum. Cerebrospinal fluid protein may be elevated. Focal delta slowing is usually present on electroencephalography. The necrotic brain may appear on radionuclide brain scan as an area of abnormal uptake and also act as an avascular space-occupying lesion. With computerized tomography, radiation necrosis appears as an intracerebral area with diminished absorption coefficient that is often enhanced with intravenous contrast medium. The syndrome may be sufficiently characteristic to eliminate the need for surgical exploration and biopsy in some cases. Cumulative experience suggests that the risk-to-benefit ratio of radiotherapy becomes increasingly unfavorable for most patients with benign intracranial neoplasms when the standard brain tumor dose of 5000 to 7000 rads is fractionated at greater than 200 rads per day.

KEY WORDS • radiotherapy • fast neutrons • CT scan • brain tumor • radiation necrosis

IRRADIATION is an effective and relatively safe treatment for some intracranial neoplasms, but a small risk of disabling brain necrosis attends the use of even standard therapeutic doses of radiation. Delayed radiation necrosis of the brain is often mistaken for recurrent tumor. Exploratory craniotomy and biopsy have usually been required for identification. This has been our experience in five of six patients encountered at Walter Reed Army Medical Center in recent years. We describe and discuss the cases of these six patients in order to aid in delineating delayed radiation necrosis of the brain as a distinct nosological entity.

Case Reports

Case 1

This 44-year-old man received radiation therapy to the left temporal bone in February, 1974, for a biopsy-proven glomus jugulare tumor (Table 1). Ten months later he developed left-sided headaches, fluent dysphasia, and focal right-sided seizures. Radionuclide brain scan, electroencephalogram (EEG), and angiogram were consistent with an intrinsic left temporal lobe mass (Table 2).

At craniotomy on January 24, 1975, the left inferior temporal gyrus was yellow and gliotic. A small biopsy from this area was

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TABLE 1
Details of radiation therapy*

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Sex, Age</th>
<th>Primary Diagnosis</th>
<th>Method of Delivery of Radiotherapy</th>
<th>Field Size</th>
<th>No. of Treatments /Days</th>
<th>Calculated Tumor Dose (rads) Max/Min</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>M, 44</td>
<td>left glomus jugulare tumor</td>
<td>superior - inferior 60° wedge filtered fields</td>
<td>4 × 5 cm</td>
<td>25/34</td>
<td>5000/5000</td>
</tr>
<tr>
<td>2</td>
<td>F, 59</td>
<td>chromophobe adenoma</td>
<td>220° coronal arc rotation</td>
<td>5 × 6 cm at isocenter rotation</td>
<td>30/37</td>
<td>6738/6065</td>
</tr>
<tr>
<td>3</td>
<td>F, 43</td>
<td>chromophobe adenoma</td>
<td>220° coronal arc rotation</td>
<td>5 × 7 cm at isocenter rotation</td>
<td>30/46</td>
<td>6580/6000</td>
</tr>
<tr>
<td>4</td>
<td>F, 46</td>
<td>craniopharyngioma</td>
<td>65% via fixed lateral field &amp; 35% via 220° coronal arc rotation for each method</td>
<td>6 × 6 cm at isocenter rotation</td>
<td>33/45</td>
<td>6880/6200</td>
</tr>
<tr>
<td>5</td>
<td>M, 48</td>
<td>malignant melanoma of the ethmoid sinuses</td>
<td>single fixed anterior field</td>
<td>7 × 8 cm</td>
<td>14/50†</td>
<td>1960†</td>
</tr>
<tr>
<td>6</td>
<td>F, 54</td>
<td>craniopharyngioma</td>
<td>220° coronal arc rotation</td>
<td>7 × 7 cm at isocenter rotation</td>
<td>30/46</td>
<td>6885/6200</td>
</tr>
</tbody>
</table>

*4 MeV linear accelerator used for all but Case 5.
†Fast neutrons equivalent to 7000 rads given in 35 treatments in 53 days.

Consistent with radiation necrosis (Table 3). After surgery the patient's condition improved. Dexamethasone, 1 mg four times daily, was continued for 6 months after surgery.

On examination in January, 1976, he had a mild dysnomia and a minimal organic mental syndrome. Two computerized tomography (CT) scans done 20 and 23 months after irradiation showed areas with diminished absorption coefficient in the left temporal region and the left cerebellar hemisphere. Contrast medium injected intravenously failed to produce enhancement of the affected areas (Fig. 1). At 2½ years after irradiation, he still has a minimal organic mental syndrome.

Case 2

This 59-year-old woman received radiotherapy to the region of the sella turcica in April, 1973, following craniotomy and subtotal removal of a chromophobe adenoma of the pituitary (Table 1). Postoperatively she was neurologically intact except for a bitemporal superior quadrantanopsia. Nine months after irradiation she became forgetful and confused. Examination disclosed a moderately severe organic mental syndrome and frontal release signs. Visual fields were unchanged. There was increased uptake in the right frontal region on a radionuclide brain scan. The EEG showed intermittent bifrontal delta slowing. A pneumoencephalogram showed no evidence of a suprasellar tumor. A presumptive diagnosis of radiation necrosis of the brain was made (Table 2). She was started

![Fig. 1. Case 1. Computerized tomography scan. Areas with diminished absorption coefficient in left temporal lobe and cerebellar hemisphere were not enhanced after an intravenous injection of contrast medium.](image-url)
### TABLE 2

**Summary of findings in six patients with delayed radiation necrosis of the brain***

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Location of Radio-necrosis</th>
<th>Signs &amp; Symptoms</th>
<th>Onset† (mos)</th>
<th>CSF Protein</th>
<th>EEG</th>
<th>Angiogram</th>
<th>CT Scan</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>left temporal lobe</td>
<td>headache, dysphasia, focal fits, OMS</td>
<td>10</td>
<td>100 mg % temporal uptake</td>
<td>lt temporal slowing</td>
<td>lt temporal avascular mass</td>
<td>diminished AC lt temporal &amp; cerebellum</td>
</tr>
<tr>
<td>2</td>
<td>both frontal lobes, optic nerves &amp; chiasm</td>
<td>amblyopia, temporal field cut &amp; OMS</td>
<td>9</td>
<td>123 mg % bilateral frontal uptake &amp; 150 mg %</td>
<td>bifrontal delta slowing</td>
<td>lt frontal mass that resolved</td>
<td>diminished AC bilateral frontal</td>
</tr>
<tr>
<td>3</td>
<td>both frontal lobes, optic nerves &amp; chiasm</td>
<td>amblyopia &amp; OMS</td>
<td>13</td>
<td>53 mg % lt frontal uptake</td>
<td>bifrontal delta slowing</td>
<td>normal</td>
<td>diminished AC bilateral frontal</td>
</tr>
<tr>
<td>4</td>
<td>left frontal lobe</td>
<td>OMS</td>
<td>28</td>
<td>-</td>
<td>-</td>
<td>lt fronto-temporal delta slowing</td>
<td>lt frontal avascular mass</td>
</tr>
<tr>
<td>5</td>
<td>both frontal lobes, optic nerves &amp; chiasm</td>
<td>amblyopia &amp; fits</td>
<td>9</td>
<td>44 mg % bilateral frontal uptake</td>
<td>bifrontal delta slowing &amp; sharp waves</td>
<td>normal</td>
<td>diminished AC bilateral frontal</td>
</tr>
<tr>
<td>6</td>
<td>both frontal lobes, optic nerves &amp; chiasm</td>
<td>amblyopia, right hemiparesis &amp; pseudo-bulbar speech</td>
<td>13</td>
<td>76 mg % normal</td>
<td>-</td>
<td>-</td>
<td>normal</td>
</tr>
</tbody>
</table>

*All but Case 6 biopsy proven. Abbreviations: OMS = organic mental syndrome; AC = absorption coefficient.
†Time of onset of signs and symptoms after radiotherapy.

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**on dexamethasone 4 mg four times a day and improved.**

In May, 1974, 13 months after radiotherapy, she was readmitted to the hospital because her organic mental syndrome suddenly became worse. Carotid angiography disclosed a large avascular mass in the left frontal lobe (Fig. 2). Dexamethasone therapy was resumed and her condition improved.

### TABLE 3

**Pathological findings in five cases of delayed radiation necrosis of the brain**

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Blood Vessel Abnormalities*</th>
<th>Myelin Necrosis</th>
<th>Gliosis</th>
<th>Perivascular Round Cells &amp; Hemorrhage</th>
<th>Edema</th>
</tr>
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<tbody>
<tr>
<td>1</td>
<td>+</td>
<td>+</td>
<td>++++†</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>2</td>
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<tr>
<td>4</td>
<td>+</td>
<td>+</td>
<td>++++†</td>
<td>+</td>
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<tr>
<td>5</td>
<td>+</td>
<td>+</td>
<td>++++†</td>
<td>+</td>
<td>+</td>
</tr>
</tbody>
</table>

*Thickening, hyalinization, fibrinoid necrosis, plasmatic transudates, recanalization, and endothelial hyperplasia.
†Atypical cells present.
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In January, 1976, 2 years and 9 months after irradiation, she was readmitted because of progressive loss of vision in the right eye. Examination disclosed a severe organic mental syndrome, only light perception in the right eye with a Marcus-Gunn pupil, a dense temporal field defect in the left eye, and bilateral Gegenhalten and Babinski signs. Carotid angiograms at this time were normal. On pneumoencephalography, the ventricles were moderately enlarged and the suprasellar cisterns were free of filling defects. A CT scan revealed areas with diminished absorption coefficient in both frontal lobes, more marked on the right. The affected areas were not enhanced with an intravenous injection of contrast medium. A craniotomy was performed on January 23, 1976, in hopes of preserving the remaining vision. No recurrent tumor was found. The orbital gyri were gliotic and discolored brownish-yellow. The right optic nerve was abnormally thin and translucent. Biopsies of brain and right optic nerve were consistent with radiation necrosis (Table 3). Despite dexamethasone therapy, the patient’s dementia has progressed.

Case 3

This 43-year-old woman presented in April, 1974, with markedly decreased visual acuity in the right eye and a temporal field defect in the left eye. A chromophobe adenoma of the pituitary was subtotally removed at craniotomy and vision improved. The sella was irradiated postoperatively (Table 1).

In June, 1975, 13 months after radiotherapy, she developed bifrontal headaches and lethargy. Examination revealed a mild organic mental syndrome. Visual acuity had deteriorated. The right eye was blind. Acuity was 20/30 in the left eye with a dense temporal field defect. Carotid angiograms and pneumoencephalogram showed no definite evidence of recurrent tumor. A radionuclide brain scan showed a large area of increased uptake in the left frontal area. A CT scan performed in June, 1975, showed irregular areas with diminished absorption coefficient in both frontal lobes and rims of enhancement around the frontal horns after an intravenous injection of contrast medium. A second CT scan in August, 1975, showed slight compression of the left frontal horn. Areas of enhancement were again noted in both frontal lobes, the one on the left appearing as a distinct “ring-blush” (Fig. 3). To rule out recurrent tumor as the cause of visual loss, right frontotemporal craniotomy was performed on September 5, 1975. The leptomeninges were adherent to the dura. The orbital gyri were yellow and gliotic, and the right optic nerve was atrophic. No tumor was...
CASE 5

This 48-year-old man had a malignant melanoma of the ethmoid sinuses and nasal cavities subtotally resected via a combined subfrontal-epidural and transnasal approach in November, 1973. Shortly thereafter he was referred to another center for fast neutron therapy to the residual tumor (Table 1). Four months later he had a seizure. An EEG showed right frontal delta slowing and epileptiform activity. He was given Dilantin and did well until August, 1974, 9 months after radiotherapy, when he began to lose vision in the frontal region that showed “ring-blush” enhancement after an intravenous injection of contrast medium (Table 2).

Craniotomy performed on January 22, 1976, showed the gyri of the left frontal lobe to be flattened and discolored brownish-yellow. A firm avascular mass, suggesting an infiltrating glioma, involved the deeper tissue. Some areas were soft, cystic, and necrotic. Histopathology of the resected portion of the left frontal lobe was consistent with radiation necrosis (Table 3).

The patient was treated with dexamethasone postoperatively and her organic mental syndrome improved. Follow-up examination 6 months after craniotomy showed further improvement in her mental status, and CT scan at this time demonstrated interval dilatation of the ventricular system, without shift or contrast enhancement.

CASE 4

This 46-year-old woman received radiotherapy to the suprasellar region in February, 1973, following craniotomy and subtotal removal of a craniopharyngioma (Table 1). She remained neurologically normal until June, 1975, 28 months after radiotherapy, when she developed a slowly progressive memory loss and became lethargic.

Examination in December, 1975, revealed a moderate organic mental syndrome with particular impairment of recent memory. Visual field testing showed only enlargement of the blind spot on the right. Angiography and EEG were abnormal and consistent with a left frontal mass lesion. A CT scan showed a shift of the anterior ventricular system toward the right, and an irregular area with diminished absorption coefficient in the left frontal lobe to be flattened and discolored brownish-yellow. A firm avascular mass, suggesting an infiltrating glioma, involved the deeper tissue. Some areas were soft, cystic, and necrotic. Histopathology of the resected portion of the left frontal lobe was consistent with radiation necrosis (Table 3).

The patient was given dexamethasone postoperatively, and her organic mental syndrome temporarily improved for 2 months. Then both mental status and visual acuity deteriorated further. A repeat CT scan in June, 1976, demonstrated interval dilatation but no shift of the ventricles, and areas with diminished absorption coefficient in both frontal lobes that enhanced irregularly. Currently (27 months after radiotherapy) she is severely demented, bilaterally amaurotic, and bedridden.
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left eye. Radionuclide brain scan at the time showed increased uptake in the frontal area. The EEG was unchanged. During the next 4 months, his left eye became blind. It was assumed that the loss of vision was due to recurrent malignant melanoma and he was started on a chemotherapy and immuno-therapy protocol. His condition remained stable until January, 1976, when he began to lose vision in the right eye.

Examination in April, 1976, revealed that vision in the right eye was reduced to 20/100 with correction and there was a temporal hemianopsia. Carotid angiograms were normal. Pneumoencephalogram revealed normal suprasellar cisterns, mild ventricular enlargement, and cortical atrophy. A CT scan showed areas with diminished absorption coefficient in both frontal lobes. There was distinct ring-like enhancement of the area in the right frontal lobe after an intravenous injection of contrast medium (Fig. 4 and Table 2).

In hopes of preserving the vision that remained, a craniotomy was performed 30 months after radiation therapy. The right frontal lobe was soft, necrotic, and discolored yellow. Both optic nerves were atrophic and translucent. No tumor was seen. The resected portion of the right frontal lobe showed features typical of radiation necrosis (Table 3). Vision has continued to deteriorate despite dexamethasone therapy. The patient remains clinically free of tumor.

Case 6

A 54-year-old woman underwent radiation therapy to the suprasellar region in April, 1974, following craniotomy and subtotal excision of a craniopharyngioma (Table 1). On discharge, visual acuity had improved to 20/20 bilaterally and she was normal neurologically. Thirteen months later she developed slurred speech and mild unsteadiness of gait. Radionuclide brain scan, carotid and vertebral angiograms, and pneumoencephalogram were normal. A CT scan disclosed areas with diminished absorption coefficient in the frontal lobes that did not enhance with contrast medium. A diagnosis of radiation necrosis was made. Symptoms improved with dexamethasone.

Three months later, as the dose of dexamethasone was reduced, the patient's gait and speech deteriorated *pari passu*, and urinary incontinence developed. Examination at this time revealed a moderately severe right hemiparesis, marked dysarthria, mild optic atrophy, and visual acuity decreased to 20/200 bilaterally (Table 2). Perimetry disclosed for the first time superior bitemporal quadrantanopsia to red. Repeat CT scan revealed the previously noted areas with diminished absorption coefficient and interval dilatation of the ventricular system. The clinical diagnosis of radiation necrosis remained unchanged. On reexamination 21 months after radiotherapy, the patient was neurologically the same and required 1 mg of dexamethasone four times a day.

Pathological Findings

All specimens were studied with the standard hematoxylin and eosin stain. In addition, luxol fast blue stain for myelin, Bodian stain for axons, Alician blue for acid mucopolysaccharides, Congo red for amyloid, and Masson stain for connective tissue were performed on selected sections.

A continuum of histological changes characteristic of radiation necrosis was noted, reflecting variation in the intensity of reaction to injury (Table 3). The cerebral white matter was selectively damaged, although small isolated areas of cortex were also necrotic. Damage within white matter was primarily directed toward small arteries and arterioles, which showed typical thickening and hyalinization of the media, loss of smooth-muscle detail, and reduction of lumen diameter (Fig. 5 upper left). Small pial vessels were often similarly involved. The adventitia of the involved vessels was thickened and fibrotic, while endothelial cells showed characteristic proliferation and nuclear pleomorphism. Many of the thickened and hyalinized small vessels contained PAS-positive material as well as foci of acid mucopolysaccharides. Multiple channels were seen within the primary lumen of some of the smaller vessels, suggesting previous thrombosis and subsequent recanalization. White-matter changes ranged from mild rarefaction of myelin with minimal reactive gliosis to areas of focal necrosis with atypical gliosis. In most cases, extensive areas of parenchymal necrosis were seen. Fibrinoid necrosis of the small muscular arteries was seen within the necrotic fields (Fig. 5 upper right). Many of the necrotic and fragmented vessels were sur-
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Discussion

Radiation necrosis of the brain should be included in the differential diagnosis of any neurological deficit that develops after therapeutic irradiation of the brain. However, it often cannot be differentiated from recurrent neoplasm without biopsy especially if the original tumor was a glioma, carcinoma, or other aggressive neoplasm that usually recurs. Both delayed radiation necrosis of the brain and intracranial neoplasia may present with papilledema,7,8 progressive loss of vision,1 hemiparesis,8,4 dysphasia,12,18 focal seizures,12,17,18 dementia,6 or hypothalamic insufficiency.1,4 Diagnostic studies rarely prove decisive because both radiation necrosis and recurrent neoplasm may 1) elevate cerebrospinal fluid protein, 2)
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**Fig. 6. Pathogenesis of radiation necrosis. The postulated sequence of events incited by radiation.**

appear on radionuclide brain scan as an area of increased uptake, 3) produce focal delta slowing of the EEG, 4) present angiographically as an avascular mass, and 5) appear on CT scan as an area with diminished absorption coefficient that may or may not be enhanced by intravenous injection of contrast medium (Table 2).

In contrast to the diagnostic dilemma often encountered among patients who have been irradiated for an aggressive neoplasm of the brain or skull, our experience suggests that a working diagnosis of delayed radiation necrosis of the brain is sometimes possible without biopsy following irradiation for the usual benign pituitary adenoma or craniopharyngioma (Cases 2 and 6). Both necrosis and these neoplasms are capable of producing progressive loss of vision, dementia, hypothalamic insufficiency, and lateralizing hemispheric signs. Therefore, one cannot differentiate radiation necrosis from recurrent pituitary adenoma or craniopharyngioma with confidence on clinical grounds alone. But in these cases the pneumoencephalogram and CT scan may provide an answer. An analysis of our cases suggests that if 1) symptoms develop less than 18 months after radiotherapy, 2) the pneumoencephalogram shows that the suprasellar cisterns are normal, and 3) the CT scan reveals an intracerebral area with diminished absorption coefficient, with or without a mass effect, that is not in anatomic continuity with the contents of the sella turcica, a diagnosis of radiation necrosis would be favored over recurrent pituitary adenoma or craniopharyngioma. However, these criteria cannot distinguish between radiation necrosis and a mass lesion developing de novo, such as an intracerebral glioma, carcinoma, or abscess. Moreover, a normal pneumoencephalogram does not rule out completely the possibility that a small, surgically treatable lesion is the cause of progressive visual loss. For this reason, whenever there is doubt, we favor surgical exploration in the hope of preventing blindness, as illustrated by our Cases 2, 3, and 5.

The pathogenesis of delayed radiation necrosis of the brain remains speculative. Some believe that the basic mechanism is a direct effect of radiation on parenchyma, whereas others contend that vascular mechanisms play the principal role. Crompton and Layton suggest that radiation may alter the antigenic structure of the brain leading to an autoimmune vasculitis. We favor the view that the primary site of radiation injury is the endothelial cell. The postulated sequence of events incited by this injury is depicted in Fig. 6.

Therapy for delayed radiation necrosis of the brain often depends on whether or not a mass is responsible for the patient's symptoms. Dexamethasone therapy often improves symptoms dramatically, as has been noted by others and illustrated by some of our cases. But surgical excision of the necrotic swollen brain, which removes both the mass and the source of edema, is necessary when there is intracranial spatial decompensation and impending tentorial herniation; in these cases the patient may subsequently improve permanently. A large mass can resolve without surgery (Case 2), while on the other hand some patients may continue to deteriorate despite surgery and dexamethasone (Case 3).

The efficacy of dexamethasone in treating...
radiation necrosis presumably derives from its anti-edema activity. However, evidence has been presented indicating that the therapeutic activity of dexamethasone may also depend upon its maintaining the integrity of excitable membranes and preventing the loss of intracellular potassium. In our experience, dexamethasone was most effective when edema seemed to be the major part of the problem, and was ineffective for the discrete lesions of the visual pathways; visual deficits usually became worse despite dexamethasone therapy. In treating radiation necrosis of the brain medically, we employ an empiric regimen based upon the experience of using dexamethasone for the treatment of brain swelling from a variety of causes. We usually give 10 mg intravenously as a loading dose and follow this with a maintenance dose of 4 mg every 6 hours intramuscularly, or orally when possible. If no improvement ensues within 36 hours, we double the maintenance dose before concluding that dexamethasone is ineffective and should be discontinued. We have found dexamethasone to be well tolerated by most patients for many months even in doses of 16 mg per day. Nevertheless, once neurological improvement has stabilized at a satisfactory level for 1 or 2 weeks, dexamethasone administration is reduced gradually to the smallest effective dose. Most patients can eventually be weaned from the drug.

Experimental data suggest that the critical dose of radiation for the monkey brain is between 5000 and 6000 rads fractionated at 200 rads per day. Nevertheless, radiation in excess of 6000 rads has been commonly employed to treat craniopharyngiomas and other relatively resistant intracranial neoplasms, but clinically apparent radiation necrosis of the brain has been reported only infrequently. Preparatory to an attempt to explain the unusually high incidence of radiation necrosis of the brain encountered at our institution, we thoroughly reviewed all radiotherapy records, affirmed that the dosimetry was accurate, and recomputed the dose received by each patient (Table 1). As listed, Case 1 received orthodox irradiation for a glomus jugulare tumor. We have concluded that he was unusually and unpredictably sensitive to the effects of radiation. The larger doses of radiation given to the four patients with either pituitary adenoma or craniopharyngioma (Cases 2, 3, 4, and 6) have been commonly used by others to treat craniopharyngiomas with what has been regarded heretofore as acceptably low morbidity. Recently, Harris and Levene reported that five of 55 patients with either pituitary adenoma or craniopharyngioma whom they irradiated developed clinical evidence of radiation damage to the optic pathways. They observed that only patients who received 250 rads per day or more suffered visual impairment. Harris and Levene concluded that "... the fraction size should not exceed 200 rads per day." In view of the experience reported here and elsewhere, we endorse this conclusion.

Reports of delayed radiation necrosis of the brain following fast neutron therapy are rare. There was nothing unique about the clinical or pathological features of our patient (Case 5) who received fast neutron therapy. When the relative biological effectiveness of fast neutron radiation is taken into account, he received approximately 200 rads per day to a total dose of 7000 rads.

**Summary and Conclusions**

The diagnosis of delayed radiation necrosis of the brain without biopsy is most readily made in patients who have been irradiated for pituitary adenoma or craniopharyngioma. In these patients, the syndrome is commonly characterized clinically by progressive amblyopia or intellectual deterioration, or both. The diagnosis is strengthened if, on pneumoencephalogram, the suprasellar cisterns are free of tumor, and the CT scan shows intracerebral areas with diminished absorption coefficient.

The diagnosis of delayed radiation necrosis of the brain is difficult to make without biopsy when it follows irradiation of a malignant neoplasm of the brain or skull. In these patients, a diagnosis of radiation necrosis is favored if hemispheric dysfunction is not associated with a mass, papilledema, or other signs of intracranial hypertension, and the CT scan demonstrates intracerebral areas with diminished absorption coefficient that are anatomically separate from the original tumor site.

Surgical excision of the necrotic, swollen part of the brain may be necessary to save a life. However, when evidence favors a
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diagnosis of radiation necrosis and the risk of intracranial spatial decompensation is small, a trial of dexamethasone may carry the patient over the period of maximum swelling and make surgery unnecessary.

A growing body of clinical and experimental evidence suggests that the risk-to-benefit ratio of radiotherapy becomes increasingly unfavorable for most patients with benign intracranial neoplasms when the standard brain-tumor dose of 5000 to 7000 rads is fractioned at greater than 200 rads per day.

References

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