follow-up data on patients in whom postoperative enhance-
tment tumor. We therefore provide clinical and radiographic
hancement at the operative site may simply represent recur-
removal can occur after brain tumor surgery; its appearance is
delayed and it disappears spontaneously; and (2) when en-
ancement at the operative site persists for longer than 3 to 4
months, there is increasing concern that recurrent tumor is a
contributing factor.

Frequent postoperative CT scans were the "methods of
procedure" by which we analyzed patterns of contrast en-
hancement in the postoperative brain. Dr Weisberg has con-
fused our method with our conclusion. We recommend a
single postoperative scan, but we also recommend that this
scan be performed on the third or fourth postoperative day.
Postoperative enhancement in brain tumor patients is more
than a radiological curiosity. It can mimic residual enhancing
tumor leading to inappropriate treatment decisions and mis-
interpretations of response to treatment [2].

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Episodic Tourette's Syndrome in a Patient with Citrullinemia

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We describe the case of a 13-year-old girl with citrullinemia
who in the course of episodic metabolic imbalances developed
symptoms of Tourette's syndrome. Citrullinemia was
diagnosed at ten weeks of age and she has been the subject of
previous communications in the literature [1]. Treatment
consisted of protein restriction and supplemental essential
amino acids. She did well on this regimen, although intercur-
rent infections triggered rare 1- to 2-day episodes of ataxia,
irritability, and lethargy associated with hyperammonemia.
At 11 years of age, she developed episodes of additional
symptoms of repetitive tics accompanied by involuntary
vocalizations.

She was referred to the Mayo Clinic for an attack follow-
ing a bout of gastroenteritis. Her parents described echolalia,
but by the time of her evaluation she showed only lethargy,
combativeness, and ataxia. Routine laboratory findings were
normal. Abnormal laboratory values consisted of: ammonia,
165 µg/dl (normal, less than 50); plasma glutamine, 207 µm/
dl (normal, 34 to 67); and citrulline, 203 µm/dl (normal 1.6
to 5.5). The patient was treated with intravenous fluids, pro-
tein restriction, supplemental arginine, and sodium benzoate,
followed by rapid improvement. On the next day examination
findings were normal. An electroencephalogram showed mild
generalized slowing. Computed tomographic scan showed
only a prominent sulcus in the left parietal cortex.

The impression was that she had an episodic form of
Tourette's syndrome precipitated by hyperammonemia and
elevated concentrations of certain amino acids. It was hoped
that the patient would return during future attacks to allow
further assessment, including video monitoring, but the par-
ten's home to obtain medical care closer to home.

While we observed this child on only one occasion and are
basing our impression of Tourette's syndrome on history
alone, we believe it worthwhile to note this possible associa-
tion. Citrullinemia produced by arginosuccinate synthetase
deficiency is a rare disorder of the urea cycle, and elevated
serum ammonia values is the hallmark. Cases are known [2]
in which citrullinemia was associated with normal ammonia
levels; therefore, ill effects on the nervous system may not
always be, or all be, due to the hyperammonemia. The brain
alters ammonia primarily by forming glutamine, with gluta-
maté—a known excitatory neurotransmitter—as an inter-
mediate metabolite [4]. It is of interest that Walser [3] cites
cases from the Japanese literature in which patients with the
late-onset type of citrullinemia show behavioral changes,
with manic episodes, frank psychosis, and echolalia.

We speculate that some transient disturbance of neuro-
transmission was responsible for the episodic Tourette's syn-
drome described in this patient.

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612 Annals of Neurology Vol 19 No 6 June 1986