THE PROBLEM OF THE GLIOBLASTOMAS*

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The years 1918 and 1926 are two important dates in the development of neurological surgery. Therefore, in studying the problem of the glioblastomas, we must first consider what conditions we faced prior to 1918 and then again what influenced this problem after 1926.

In 1918 ventriculography was discovered. Prior to that date, those few of us who were attempting to do neurological surgery were occupied primarily with detailed neurological studies to enable us to arrive at a diagnosis of brain tumor, and then we carried these examinations still further trying to localize the lesion. When we had finally exhausted all our methods of study, which at that time consisted solely of the history and neurological examination, we had the further problem, no mean one at that time, of convincing our neurological or medical colleagues that an operation should be undertaken; and then, finally, we had the problem of the operation itself. It is difficult for neurosurgeons today to realize what we were up against. For example, the first dozen patients with brain tumor I saw in 1911 were all blind. I wondered whether I would ever see one before he was blind. In those days, we never dared to make a positive diagnosis of tumor unless the patient had a full-blown choked disc. The only exception to this rule was if we were fortunate enough to have a patient with typical Jacksonian convulsions. I do not remember just when I operated upon the first patient with tumor who had no choked disc, but it was after 1918. Though we made most careful neurological examinations, many of the symptoms and signs elicited we were unable to interpret correctly. For example, paresis of one sixth nerve caused us no end of difficulty and mental juggling trying to fit it into a clinical picture. We did not appreciate that bilateral sixth nerve paralysis was a very different matter. While unilateral sixth nerve paresis was a general sign of increased intracranial pressure due, as Cushing pointed out in 1911, to pressure on the nerve by a branch of the basilar artery, bilateral sixth nerve paralysis was due to a pontine lesion where the two sixth nerve nuclei lie close together. It took some of us some time to realize what a great difference existed between paresis and paralysis of these nerves and how seriously this might affect the diagnosis and prognosis in a given case.

The significance of visual fields was by no means clear, and the sign of a partial or complete homonymous hemianopsia we didn’t learn to interpret until after 1911, when Adolph Meyer and Archambault described the visual

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pathway traversing the temporal lobe. It was only then that the importance of very 
careful study of the visual fields was appreciated. Even nystagmus and 
ataxia caused us difficulty, and I recall vividly exploring a patient’s 
cerebellum twice because of these signs, and only at a third attempt, 
because of some other signs that had developed, did I finally remove a large 
frontal meningioma. Frontal and cerebellar lesions were not infrequently 
confused and v. Monakow advanced the theory of diaschisis to explain such 
ocurrences. These are but a few of the diagnostic pitfalls we encountered. 

Then came the operative procedure. Though this is still a formidable 
affair, in those days it was far more so, for we lacked many of the aids we 
have today. Blood transfusion was just beginning to be used, but it was 
unusual to have more than one pint ready, and of course that was long 
before the days of blood banks. Matching of blood was not well developed 
and citrated blood had not been heard of. So we had to use either direct 
transfusion by the Crile method or Kimpton tubes, waxed cylinders in 
which the blood was collected and then introduced into the patient. The 
control of hemorrhage was an ever-present problem. Wax to control bleeding 
from the bone and silver clips to control bleeding from the brain vessels 
we did have. Bits of muscle were also used as a hemostatic, taken either 
from the patient himself or from another being operated upon at the time, 
or by using pigeon’s breast, as was practised by de Martel. The value of 
giving the patients fluid during the operation to counteract the fluid loss 
was not known or appreciated, and the control of increased pressure by 
tapping the ventricles had not become a routine procedure. In fact, I recall 
a meeting held here in Philadelphia in 1922 at which there was a lengthy 
discussion on the wisdom of doing a ventricular puncture in the course of an 
operation, and there were those who strongly opposed it.

Then it happened innumerable times that the patient was operated upon 
but the tumor was not found. No neurosurgeon today can fully appreciate 
the chagrin and mortification of being obliged to acknowledge that—time 
and time again.

When we did remove the tumor, the pathologist told us it was a glioma. 
In 1918 Dandy made his epoch-making discovery of ventriculography. 
This was not immediately accepted, but as soon as it was, it transformed the 
matter of localization completely. It soon became almost a disgrace not to 
locate a tumor at operation, and negative explorations were far less frequent. 
After ventriculography came into general use, the incidence of locating a 
tumor jumped from about 55 per cent to over 97 per cent, but there still 
were tumors that we did not expose because we did not have facilities to incise 
the cortex freely, and therefore hesitated to incise it with impunity as we do today.

The pathologist still reported gliomas, but from this time on we removed 
tumors much more frequently and came to realize that though the 
pathologist reported all the tumors as gliomas there were different types. 
Some were cystic, some were well encapsulated, some contained calcium and