Original Article

Role of Stereotaxis in the Treatment of Cerebral Palsy

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ABSTRACT

There is a renewed interest in basal ganglia surgery for improvement of motor symptoms in cerebral palsy. Rigidity, choreoathetosis, and tremor can be improved or abolished by a well-placed radiofrequency lesion, either in the ventrolateral nucleus of the thalamus or ventroposterior pallidum. The target is chosen based on the predominance of the symptoms in a given patient. A review of the main reports on surgery of the basal ganglia for cerebral palsy, as well as the author's data, shows that the surgery can have a remarkable impact on patients' quality of life when motor dysfunction is improved. An update of the physiopathology of cerebral palsy motor symptoms related to anatomic findings on experimental work, magnetic resonance imaging, and autopsy is used to rationalize surgery of the basal ganglia. Modern stereotactic technique based on exquisite demonstration of the basal ganglia anatomy by magnetic resonance imaging is described and supported by intraoperative electricophysiologic studies. The author stresses the importance of a multidisciplinary approach to provide the cerebral palsy patient with a comprehensive treatment plan before stereotactic surgery. (*J Child Neurol* 1996;11(Suppl 1):S43–S50).

The term cerebral palsy encompasses a wide range of neurologic syndromes mingled with different levels of child development. It represents the variable degree of brain damage that leads to the defective development of specific neurologic functions. The definition that will be used here came from a group of neurologists discussing the term in 1964: "disorder of movement and posture due to a defect or lesion of the immature brain."[1] This definition lacks completion because mental status is not taken into consideration. However, the definition is adequate for the purpose of this discussion because operative procedures mentioned here affect only motor function, without any effect on mentation.

Surgery for this entity, usually performed by the neurosurgeon and/or orthopedic surgeon, must be directed toward the improvement of the compromised function. The surgery must always be corroborated by a multidisciplinary support program capable of complementing the surgical improvement and fostering functional recovery and development. This involves the close support of pediatricians, psychologists, physical therapists, and specialized schools. Having this support group, surgeons may work in concert to provide improvements in the patient's physical status or give the patient the opportunity to achieve a development that is closer to normal.

Nervous system surgery can be targeted to a specific group of symptoms when it is performed in the peripheral nervous system, or it can have a broader repercussion when it is directed to the central nervous system circuitry. The chosen central nervous system circuitry to be worked on is dictated by a general evaluation of the patient's neurologic signs. Because the operations are performed in a developing brain, they must be thought of as a strategy to provide freedom for areas of the central nervous system to develop. These areas would otherwise be hindered from development when an antagonistic system is overfunctional. It must be kept in mind that one is trying to achieve a balance of complex functions in development.[2]

The manipulation of the cerebral circuitry with surgical intervention is not a new concept. Stereotactic surgery emerged from the concept of provoking major functional changes in the brain with minimal disturbance of its structure.[3] The scope of these changes ranged from an abnormal motor function such as tremor to complex, high-level mental behaviors. Although it is intuitive to understand the motor repercussion of surgery in the nervous system for treatment of spasticity, rigidity, and alternating movements, which are common in cerebral palsy, the concept of changing the realm of psychological behavior is less acceptable. Families caring for children with cerebral palsy seek help, however, not only in the

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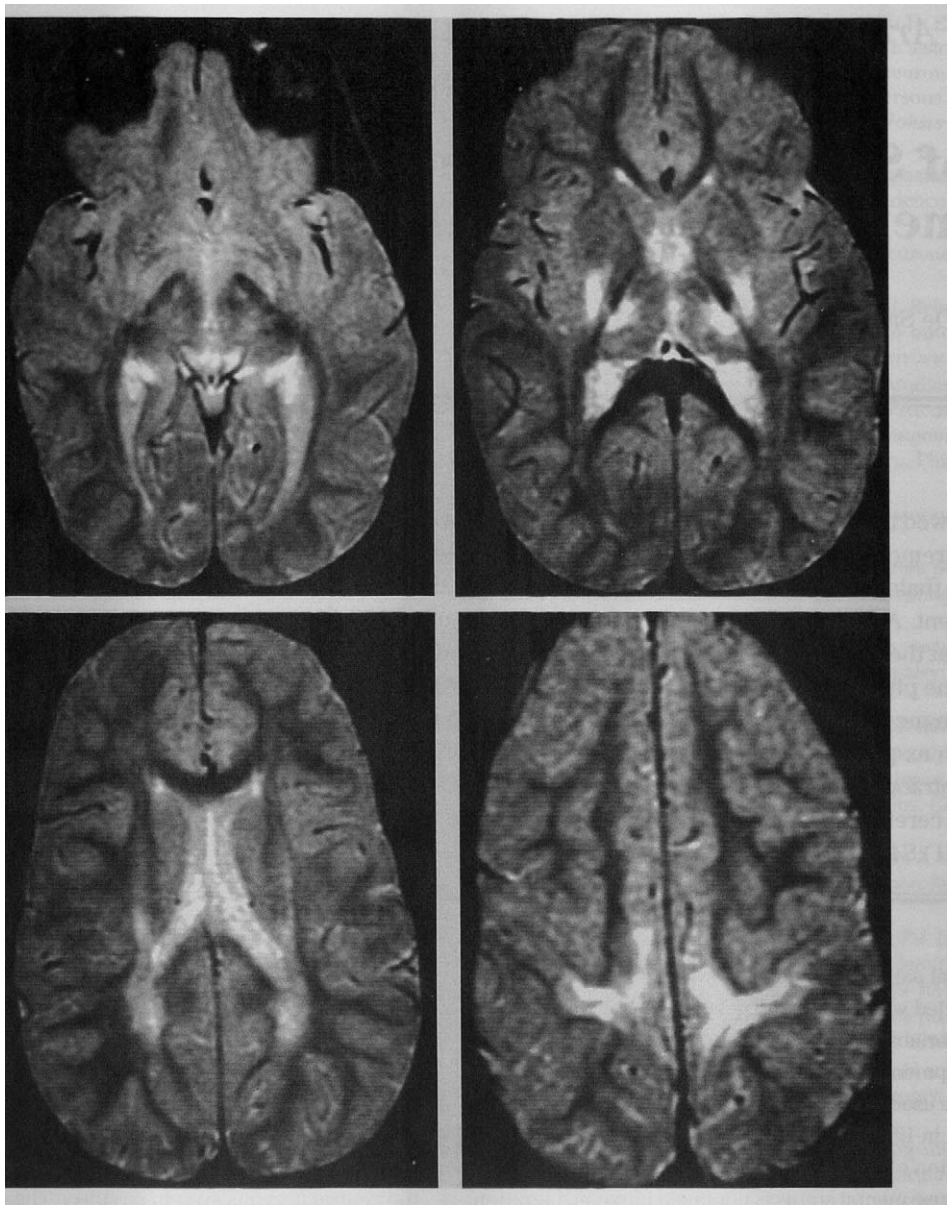

Figure 1. Two-year-old patient who developed choreoathetoid cerebral palsy secondary to a birth hypoxic event. This patient has severe motor abnormalities, including generalized hypotonia with inability to hold the head up and continuous choreoathetoid movements. The patient's mental status is developing close to normal for her age. *Upper left,* Bright signal of the subthalamic nuclei. *Upper right,* Bright signal at the level of the ventrolateral thalamus and posterior putamen. *Lower left,* Posterior periventricular bright signal corresponding to the bright signal at the sensory motor cortex (*lower right*). These findings are bilateral and symmetric.

motor but also in the behavioral sphere. It is not uncommon for parents to be extremely grateful after surgical procedures that did not clearly improve the child's motor skills, but enable the child to enjoy a better sleep pattern because of less painful spasms. The whole well-being of the patient is affected in this manner. Surgical procedures in the brain provide the opportunity for the family to work with a wide range of functional improvements that offer families and children hope and a better quality of life. This text will discuss the pathogenesis of cerebral palsy and relate it to selected central nervous system surgical procedures using modern stereotactic techniques.

PHYSIOPATHOLOGIC CONSIDERATIONS

The complex interplay between cerebral cortex, cerebellum, basal ganglia, brain stem, and spinal cord in the determination of smooth motor response and postural reflexes have been under scrutiny for several years.[4,5] Although the understanding of this interplay is far from complete, a review of the currently accepted concepts is important in the discussion of the physiopathology of cerebral palsy motor symptomatology.

Studies on spasticity suggest that periventricular leukomalacia, secondary to preferential decreased blood flow in the white

matter during the neonate ischemia,[6] promotes an overdevelopment of the anterior horn interneurons, with consequent hyperactive stretch reflex.[7] When sensory motor cortex is damaged in the neonatal period, an exuberant retention of neonatal projections into the thalamus and brain stem occurs, which in normal circumstances disappears during maturation.[8] These abnormal features may have a profound effect on fine motor function balance. Schneider and Crosby suggested that the balance of cortical output from the pyramidal system is obtained by the complex interaction of cerebellar output modulated at the level of basal ganglia.[2] Recent studies based on magnetic resonance imaging (MRI) findings in patients subjected to neonatal hypoxia showed that selective destruction of specific basal ganglia nuclei takes place.[9,10] Moreover, depending on the phase of central nervous system development in which the hypoxic event occurs (Figure 1), different areas of the basal ganglia or white matter are damaged.[6,11] This damage is accompanied by complex cerebral palsy motor abnormalities.

Kupsky et al showed that poor perfusion associated with hypothermia in toddlers undergoing cardiac surgery with circulatory arrest led to a peculiar distribution of lesions in the basal ganglia.[12] This was observed in postmortem pathology. The pallidum externum showed marked loss of myelin, and the lenticular fasciculus, pallidal thalamic fasciculus, and subthalamic fiber bundles crossing the internal capsule showed important vacuolation with myelin fragmentation. The caudate nucleus, putamen, subthalamic nucleus, thalamus, and hippocampus were well preserved in these patients. These toddlers presented in the postoperative period with severe choreoathetoid symptomatology, showing the importance of the level of development of the brain at the time of the insult to generate specific cerebral palsy symptomatology. These observations enhance the understanding of selective surgical approaches to the basal ganglia to control motor disturbances of cerebral palsy. To design interventions in the basal ganglia, it is necessary to understand the flux of motor-related information passing through the pallidum and thalamus.

OUTFLOW FROM PALLIDUM AND THALAMUS

A lesion well localized in the subthalamic nucleus consistently produced choreoathetoid and ballistic movements on the opposite side of the body of primates.[13] The subthalamic nucleus has an excitatory influence on the globus pallidus internum, which in turn inhibits the output from the ventrolateral thalamus toward the premotor cortex. This concept has been recently revived to support Parkinson's disease symptom improvement after posteroventral pallidotomy.[14] It is postulated that the positive modulation of globus pallidus internum via globus pallidus externum and subthalamic nucleus, known as the indirect pathway, is compromised in situations of striatal lack of dopamine. The net effect is a dyskinetic state. Also, the direct striatal-pallidal-thalamic pathway when compromised leads to a lack of outflow from the thalamus toward the premotor cortex. This lack of thalamic output to the premotor cortex causes the rigid-akinetic symptoms. Moreover, the dyskinesias observed in patients overmedicated in the advanced stages of Parkinson's disease, resembling the choreoathetoid movements observed in cerebral palsy, respond well to surgical

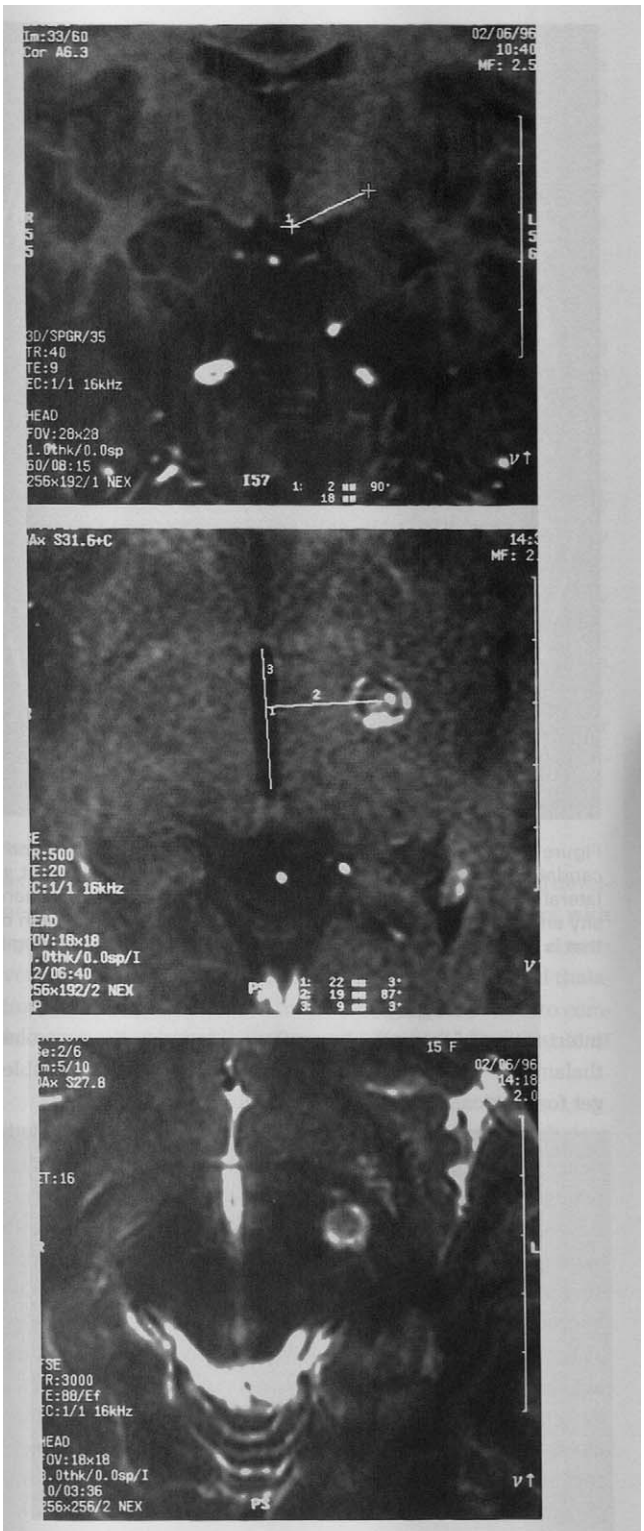

Figure 2. Ventroposterior pallidal lesion to control unilateral dystonia in a 14-year-old patient who developed symptoms after childhood encephalitis. This lesion was successful in improving her torticollis and dyskinetic movements in her extremities. Target determination was based on mammillary body depicted on a coronal image perpendicular to the anterior-posterior commissure line (*top*). A gadolinium-enhancing lesion on a T_1-weighted MRI scan at the level of the anterior commissure–posterior commissure plane is shown. Notice the measurements of the lesion location based on anterior commissure–posterior commissure landmarks (*middle*). Lesion seen on a T_2-weighted image at its most ventral level (*bottom*).

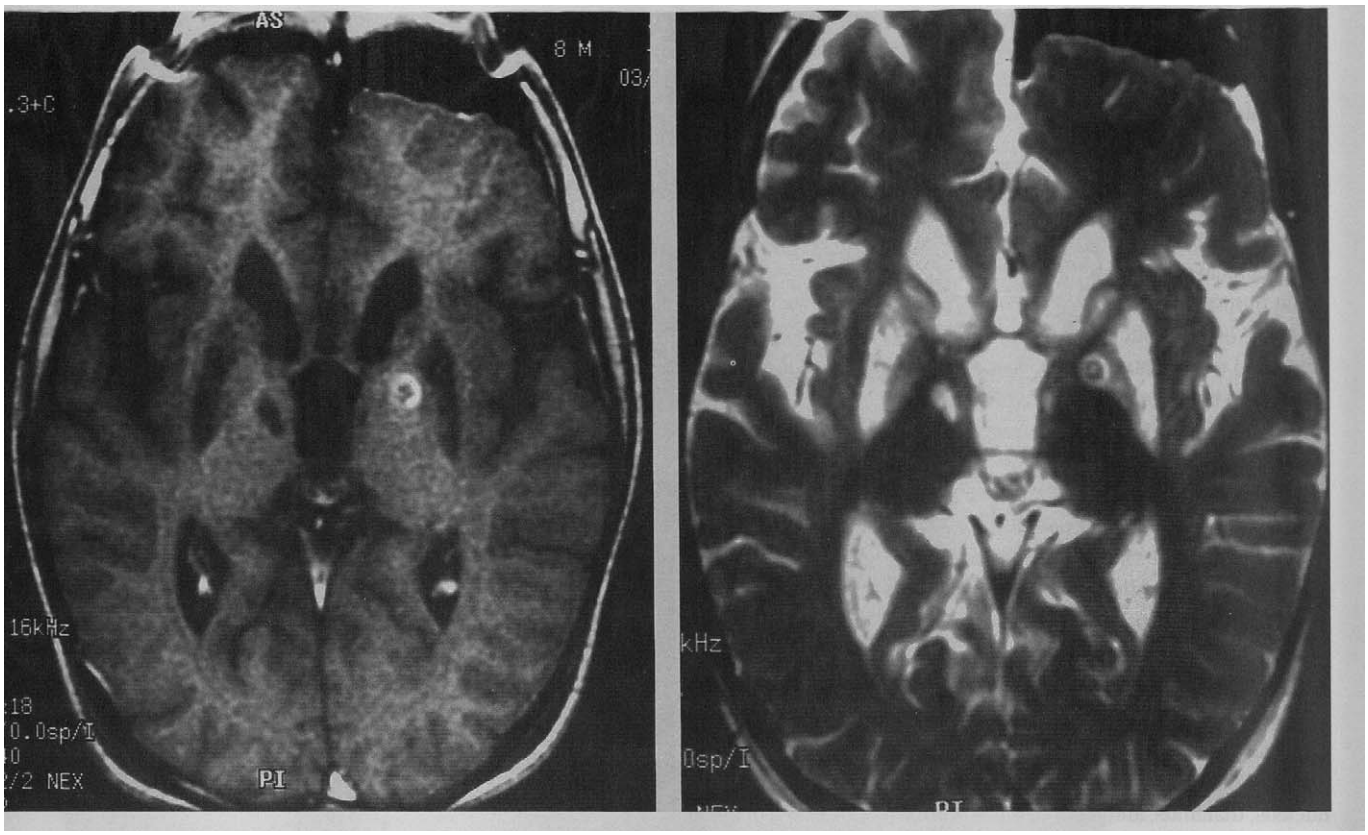

Figure 3. T_1-weighted MRI after gadolinium infusion (*left*) and T_2-weighted MRI (*right*). Notice the complete destruction of the putamen and caudate seen as dark signal on T_1-weighted image and bright signal on T_2-weighted image. Also notice a large chronic lesion on the ventrolateral nucleus of the right thalamus and an acute radiofrequency lesion in the left globus pallidus internum showing a center of hypointensity and a rim of gadolinium enhancement. This lesion is seen on T_2-weighted image as a center of bright signal, surrounded by a dark signal that is encompassed by an area of bright signal corresponding to edema. The aspect of these lesions has been previously described.[24]

interruption of the pallidum outflow directed to the ventrolateral thalamus.[15] This site must therefore be regarded as a possible target for surgical intervention for cerebral palsy symptoms.

Recently, microelectrode studies in humans provided evidence of neuronal hyperactivity in the globus pallidus internum of patients with Parkinson's disease.[16–18] This suggests that globus pallidus internum lesion would have a positive effect on the dystonic symptoms of cerebral palsy (Figure 2). Early and recent data on surgery of the basal ganglia support a remarkable improvement in choreoathetotic, dystonic, and tremor types of cerebral palsy.[19–22] There are two inputs to the globus pallidus internum. These inputs, also known as direct and indirect striatal-pallidal pathways, have an opposite effect on thalamocortical outflow. When inputs to globus pallidus internum are not balanced, ie, when direct and indirect pathways are out of balance, either bradykinesia and rigidity caused by increased globus pallidus internum outflow or hyperkinesias and dyskinesias caused by decreased globus pallidus internum outflow ensues. A lesion blocking this imbalance, either at the globus pallidus internum or ventrolateral thalamus, brings back the balanced motor function to be controlled only at cortical levels. The repercussion of such lesions beyond motor improvement is poorly described. The ability to learn new motor skills based on visual cues may be affected, as demonstrated by experimental work in primates.[23] Exuberant retention of neonatal projections into the thalamus and brain stem when the sensorimotor cortex is ablated in the neonatal period, as mentioned above,[8] may lead to a complex motor syndrome with spastic and rigid components. When this is the case, specific lesions in the thalamus, usually in the ventrolateral nucleus, would in theory improve the motor abnormality.[21] On the other hand, increased outflow from the pallidum internum, leading to choreoathetoid movements with a large rigid and dystonic component, secondary to ischemic lesion of the pallidum externum, as described by Kupsky et al,[12] may be improved by a lesion in the pallidum internum (Figure 3). The mechanism of improvement is similar to that observed in the parkinsonian patient undergoing pallidotomy. A lesion of the cerebellar dentate nucleus for treatment of spasticity[25] has also been suggested. Despite the proven functional relationship between cerebral cortex, thalamus, and cerebellar regions,[26] the reported results of dentatotomy have not been reproduced. Proponents of dentatotomy for treatment of spasticity in cerebral palsy believe Schneider and Crosby's theory of a predominance of the cerebellar output over the cortical output.[2,27] This theory has as such been abandoned because of the uncertain results obtained by dentatotomy.

CLASSIFICATION OF CEREBRAL PALSY

There are no defined boundaries to classify cerebral palsy. The basic components causing the motor disturbances in cerebral palsy are spasticity, rigidity, dystonia, tremor, and ataxia. They appear in

various combinations to determine the clinical presentation in a particular patient. Based on the predominance of these basic components, cerebral palsy can be divided into major groups. The spastic group can be helped by selective rhizotomy and infusion of baclofen intrathecally.[28-30] The rigid, athetodystonic, and tremor types are candidates for stereotactic surgical procedures directed to the central nervous system circuitry and will be addressed here, based on the previous physiopathologic considerations. Because a combination of all these types is common or almost the rule, careful analysis of symptoms with interventions in mind must be conducted by a team of specialists. The indication of a specific procedure must be discussed with future expectations of the child's development and possible adjuvant physical therapy and complementary orthopedic approaches in mind.

PATIENT SELECTION FOR STEREOTACTIC SURGERY

Rigidity

Rigidity is a common component of cerebral palsy. When spasticity is more pronounced than rigidity, the patient is classified under spastic type; however, when the inverse is the case, the patient is classified as a rigid type. Patients with rigid cerebral palsy usually have no hyperreflexia, or it is mild, depending on the degree of associated spasticity. Patients with exaggeration of the phasic stretch reflex with consequent clonus are included in the spastic group. Patients with pronounced rigidity as a consequence of increased tonic stretch reflexes are included in the rigidity group.

When rigidity is more pronounced than spasticity, even though the patient is in the mixed state called rigidospasticity, a characteristic posture is observed. The arm is often extended and externally rotated at the shoulder, with flexed fingers. The leg is semiflexed at the knee, adducted, and internally rotated at the hip. The foot is usually inverted. When spasticity dominates the picture, the arm is flexed at the elbow, the leg is extended, and the foot is everted.[21] When there is a mixture of features, stereotactic surgery can improve the rigidity and allow for treatment of the spasticity. When these patients have preserved intellectual abilities, they obtain remarkable improvement from stereotactic thalamotomy or pallidotomy. Signs of extrapyramidal predominance of symptoms are pronounced in these patients.

Athetodystonia

This is a complex picture associated with slow, alternate movements and tonicity of selected muscle groups, leading to a deformed patient who is plagued with pain and the inability to use the extremities appropriately. When the truncal muscles are affected by hypertonus, the patient has a lack of axial control. Choreic movements, torticollis, tortipelvis, opisthotonus, scoliosis, and secondary changes such as joint dislocation appear. The underlying rigidity predicates much of the surgical result. Some of these patients cannot acquire the sitting, and much less the standing, position. They are restricted to the lying position in a deformed and uncomfortable manner. Painful spasms and torsions prevent them from proper sleep and make care difficult. Surgery for these patients, usually directed to the pallidum internum, aims to achieve better bed position, to release the painful spasms and torsions and to facilitate care.

Tremor

Patients with mild perinatal hypoxia may have a near-normal development but have a cerebellar type of tremor that appears with action. Infantile head trauma can also lead to mild to severe coarse movements that impair fine motor functions. Ventrolateral thalamotomy can completely control tremor, bringing the patient to complete or almost complete normal function.[21] The result of

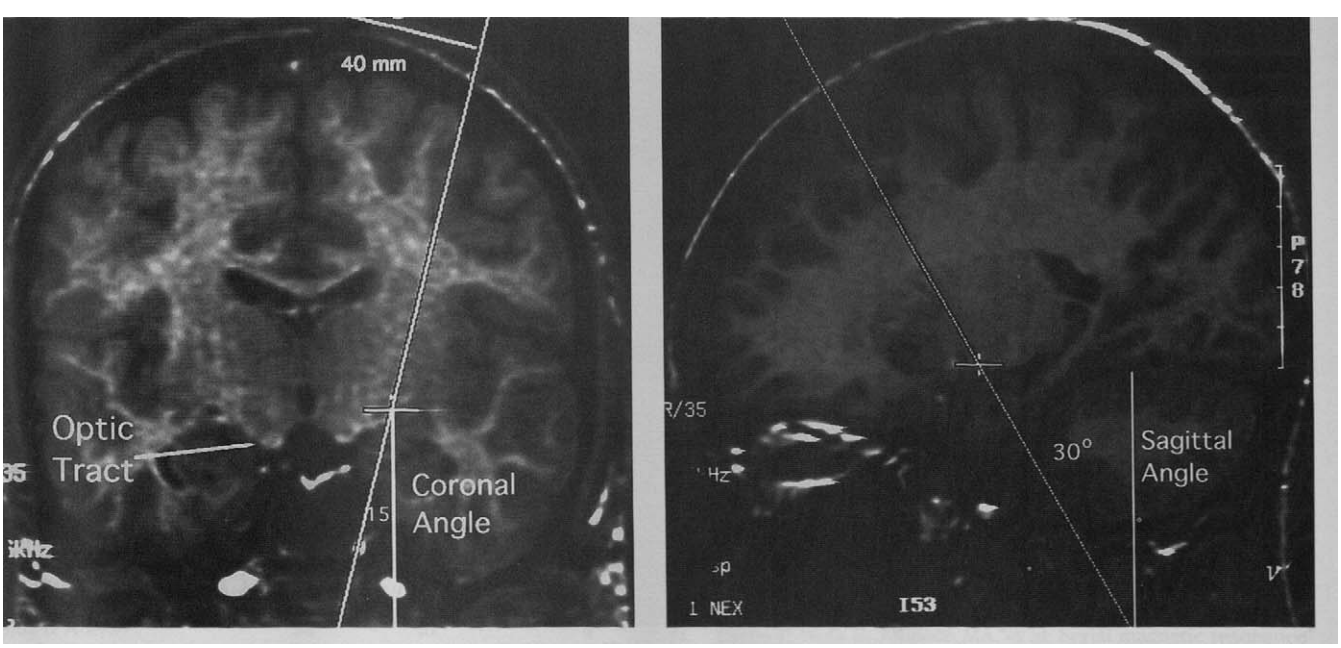

Figure 4. The angles of electrode entrance are determined based on the coronal and sagittal planes. The burr hole is placed 4 cm from the midline and 1 cm anterior to the coronal suture. Notice the target just above the lateral cerebral fissure (*left*) and above the amygdala (*right*).

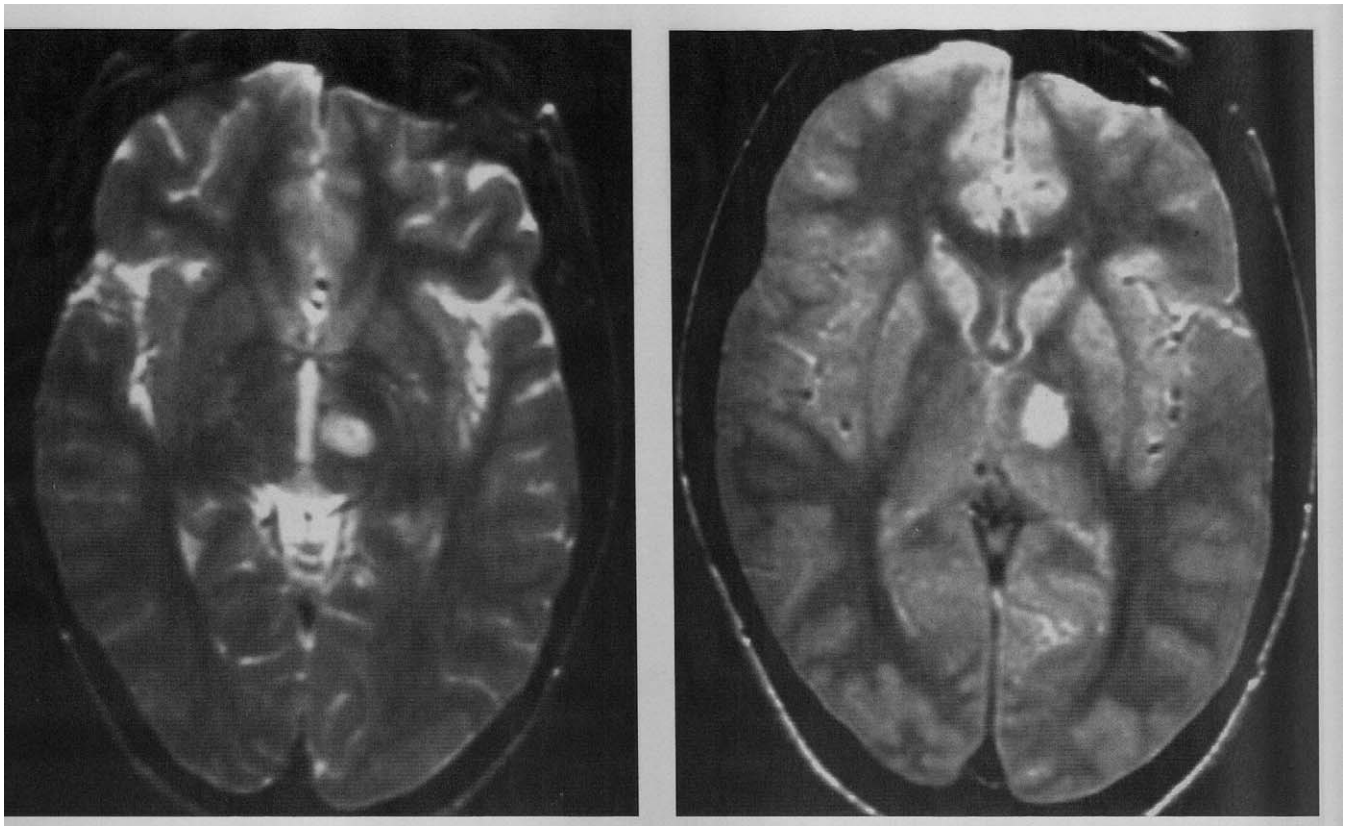

Figure 5. T_2-weighted image showing a left ventrolateral thalamotomy at the level of the anterior commissure–posterior commissure line (*left*) extending to the level of the foramen of Monro (*right*). Notice the proximity of the lesion to the posterior limb of the internal capsule. Scan was obtained within 1 hour of surgery.

thalamotomy for treatment of tremor type cerebral palsy has been confirmed.[31] Ventrolateral thalamotomy affects the thalamic input from the globus pallidus and cerebellum, as discussed previously.

BASAL GANGLIA SURGICAL TECHNIQUE

MRI allows for detailed visualization of the basal ganglia and its relationship to eloquent structures to be avoided (Figure 1). The submillimetric precision of modern stereotactic frames provides the surgeon the necessary confidence that a specific target can be reached. Several possible sources of error must, however, be taken into consideration. The precision of the vertical coordinate is limited to half the thickness of the image slice.[32] Also, distortions of the magnetic field can cause significant targeting errors.[33] Therefore one can not rely only on imaging studies to perform functional stereotactic procedures. Electrophysiologic studies are necessary.[34]

Targeting

The target is determined using the sagittal, coronal, and axial MRI planes of 1-mm thickness obtained with volumetric sequences. The sagittal plane is used for determination of the anterior commissure and posterior commissure. A line between these two structures is used to determine the direction of the coronal planes. Coronal planes are obtained perpendicular to the anterior commissure–posterior commissure line. The pallidal target is determined based on the coronal plane. The mammillary bodies are identified, as well as the internal capsule. The target is located in this coronal plane, just lateral to the internal capsule and above the amygdala, before reaching the lateral cerebral fissure, and 2 mm above and lateral to the optic tract (Figure 2, top). This site corresponds to the target described by Laitinen et al.[15] The measurements given by these authors for Parkinson's disease patients are not applicable to children with cerebral palsy because of the variability of the size of their brain (Figure 2, middle). The angles of electrode entrance are defined based on the coronal and sagittal planes (Figure 4).

The ventrolateral nucleus of the thalamus target is determined based on the proportional atlas of Talairach and Tournoux.[35] The target lies at a point situated just medial to the internal capsule, as visualized on the anterior commissure–posterior commissure axial plane, and at the anteroposterior site situated at the junction of the posterior third with the medial third of the anterior commissure–posterior commissure line (Figure 5).

Electrophysiologic Guidance

The cerebral palsy patient is generally unable to cooperate during the surgical procedure. Lack of understanding because of early age and inability to communicate during the surgical procedure make it difficult to count on the patient's response to electrical stimulation and verbal commands. Therefore, stereotactic surgery for cerebral palsy is usually performed under general anesthesia. The access for the electrode placement is made through a burr hole (Figure 4). The author prefers the burr hole access instead of twist drill because of the cerebral atrophy commonly present in cerebral

palsy patients, with consequent risk of subdural hematoma when blind puncture of the cerebral cortex is performed. It is important to maintain the patient without muscle relaxants during the anesthesia. Electrical stimulation of the internal capsule is detected as twitches of the muscles on the opposite side. Stimulation is performed using 1-ms square pulses and 2 to 10 Hz to elicit muscle response. The current or voltage is monitored to give an idea of the distance of the tip of the electrode to the internal capsule. Using a radiofrequency generator (Model 3FG, Radionics, Burlington, MA), thresholds above 2 V denote a safe distance from the internal capsule. The proximity of structures such as sensory pathways or visual pathways is usually studied with frequencies of 50 Hz or over. When the patient is under general anesthesia, sensory or visual response is monitored by evoked potentials.[21] The 50-Hz stimulation also allows for generation of rigidity by increase of the pallidal output when the globus pallidus internum is stimulated. The electrode is moved depending on the response to stimulation observed.

Radiofrequency lesions are performed in two steps. The temperature is increased to 50°C, and a thorough neurologic examination regarding tonus is carried out. If no undesired effects are observed, the temperature is increased to 80°C and maintained for 60 seconds. This will make a permanent lesion. The author uses a 2-mm exposed tip electrode with 1.8-mm diameter. Three lesions are placed in the trajectory of the electrode spaced 2 mm apart. This usually accomplishes an oval lesion with the long axis (6 to 9 mm) matching the trajectory of the electrode. The transverse diameter of the lesion is usually from 4 to 6 mm. The lesion is seen acutely on a postoperative MRI scan.[35] Several authors are recommending the use of microelectrode recordings to confirm targeting. Although elegant, this technique has not translated to better outcome for the patient. In the author's experience, microelectrode recording offers interesting electrophysiologic findings; however, it significantly increases the length of the operation, increases the need for several passes through the brain for mapping with consequent increase in complications.

RESULTS AND DISCUSSION

Speelman and van Manen reported the longest published follow-up on stereotactic surgery for cerebral palsy.[36] A mean follow-up of 21 years showed objective and subjective improvement in 44% of the patients. They described 64% side effects after the operation. Hemiparesis and speech impairment were the most common complications. These authors did not recommend surgery for patients with tetraplegia or diplegia. They also felt that the surgery had no effect on spasticity. Their results were less favorable than that of other authors (Table 1). They acknowledged that their lesions were apparently larger than those of other authors. They also used a stereotactic technique that is now surpassed by computerized stereotactic techniques. Further work is necessary to finally establish the role of stereotactic surgery in the management of cerebral palsy. It is consensus, however, that patients with unilateral dystonia, tremor, and choreoathetoid symptomatology receive remarkable benefit from surgery.[19,21,22,36–38] Our experience confirmed Speelman and van Manen's recommendations; however, the side effects described by them are no longer observed.

Table 1. Results of Stereotactic Surgery for Cerebral Palsy*

Author (s)	*N*	*Good/Fair, %*	*Poor, %*
Balasubramaniam et al[19]	94	81	19
Broggi et al[37]	33	97	3
De Salles	7	71	29
Laitinen[38]	10	65	35
Narabayashi[21]	117	78	22
Speelman & van Manen[36]	18	44	66
Trejos & Araya[22]	38	92	8

*This is only a sample of the most representative publications. The published results were adapted to the good/fair and poor scale.

In summary, cerebral palsy patients must be evaluated in neurosurgical centers with the capability to treat spasticity, dystonia, and choreoathetoid syndromes. The age of referral for a neurosurgical center is controversial, depending on the classification of the cerebral palsy. In general, if the patient can use the extremity despite the tremor or athetosis, the surgery should be performed during the teens, when the extremity is well developed and the brain is mature. However, if the extremity is not being used because of the symptoms, surgery should be performed at an early age, between 3 and 5 years. This provides the opportunity for useful development of the extremity. The proper surgical approach, either selective posterior rhizotomy, intrathecal baclofen continuous infusion, or stereotactic thalamotomy or pallidotomy, must be determined by a group of experts working in concert. The most effective procedure for specific symptoms is still a subject for research.

References

1. Bax MCO: Terminology and classification of cerebral palsy. *Dev Med Child Neurol* 1964;6:295–307.
2. Schneider RC, Crosby EC: The interplay between cerebral hemispheres and cerebellum in relation to tonus and movements. *J Neurosurg* 1963;20:188–198.
3. Spiegel EA, Wycis HT, Lee AJ: Stereotaxic apparatus for operations on human brain. *Science* 1947;106:349–350.
4. Marsden CD, Obeso JA: The functions of the basal ganglia and the paradox of stereotaxy surgery in Parkinson's disease. *Brain* 1994;117:877–897.
5. Park TS, Owen JH: Surgical management of spastic diplegia in cerebral palsy. *N Engl J Med* 1992;326:745–749.
6. Yokochi K, Aiba K, Horie M, et al: Magnetic resonance imaging in children with spastic diplegia: Correlation with severity of their motor and mental abnormality. *Dev Med Child Neurol* 1991;33:18–25.
7. Huselbosch CE, Coggeshall RE: A comparison of axonal numbers in dorsal roots following spinal cord hemisection in neonate and adult rats. *Brain Res* 1983;264:187–197.
8. Leonard CT, Goldberg ME: Consequences of damage to the sensorimotor cortex in neonatal and adult cats. II. Maintenance of exuberant projections. *Dev Brain Res* 1987;32:15–30.
9. Menkes JH: *Textbook of Child Neurology.* Baltimore, Williams & Wilkins, 1995, pp 325–378.
10. Menkes JH, Curran J: Clinical and MR correlates in children with extrapyramidal cerebral palsy. *AJNR Am J Neuroradiol* 1994;15:451–457.
11. Byrne P, Welh R, Johnson MA, et al: Serial magnetic resonance imaging in neonatal hypoxic-ischemic encephalopathy. *J Pediatr* 1990;117:694–700.

12. Kupsky WJ, Drozd MA, Barlow CF: Selective injury of the globus pallidus in children with post cardiac surgery choreic syndrome. *Dev Med Child Neurol* 1995;37:134–144.
13. Carpenter MB, Whittier JR, Mettler FA: Analysis of choreoid hyperkinesia in the rhesus monkey: Surgical and pharmacological analysis of hyperkinesia resulting from lesions of the subthalamic nucleus of Luys. *J Comp Neurol* 1950;92:293.
14. Bergman H, Wichmann T, DeLong MR: Reversal of experimental parkinsonism by lesions of the subthalamic nucleus. *Science* 1990;249:1436–1438.
15. Laitinen LV, Bergenheim AT, Hariz MI: Leksell's posteroventral pallidotomy in the treatment of Parkinson's disease. *J Neurosurg* 1992;76:53–61.
16. Iacono RP, Shima F, Lonser RR, et al: The results, indications, and physiology of posteroventral pallidotomy for patients with Parkinson's disease. *Neurosurgery* 1995;36:1118–1127.
17. Lozano A, Hutchinson W, Kiss Z, et al: Methods for microelectrode-guided posteroventral pallidotomy. *J Neurosurg* 1996;84:194–202.
18. Sterio D, Beric A, Dogali M, et al: Neurophysiological properties of pallidal neurons in Parkinson's disease. *Ann Neurol* 1994;35:586–591.
19. Balasubramaniam V, Kanaka TB, Ramanujan PB: Stereotaxic surgery for cerebral palsy. *J Neurosurg* 1974;40:577–582.
20. Laitinen LV: Short term results of stereotaxic treatment for infantile cerebral palsy. *Confin Neurol* 1965;26:258–263.
21. Narabayashi H: Choreoathetosis and spasticity, in Schaltenbrand, Walker (eds): *Stereotaxy of the Human Brain.* New York, Thieme-Stratton, 1982, pp 532–542.
22. Trejos H, Araya R: Stereotactic surgery for cerebral palsy. *Stereotac Funct Neurosurg* 1990;54+55:130–135.
23. Canavan AGM, Nixon PD, Passingham RE: Motor learning in monkeys (*Macaca fascicularis*) with lesions in motor thalamus. *Exp Brain Res* 1989;77:113–126.
24. De Salles AAF, Brekhus SD, De Souza EC, et al: Early postoperative appearance of radiofrequency lesions on magnetic resonance imaging. *Neurosurgery* 1995;36:932–936.
25. Nashold BS, Slaughter DG: Effects of stimulating or destroying the deep cerebellar regions in man. *J Neurosurg* 1969;31:172–186.
26. De Salles AAF, Bittar G: Thalamic pain syndrome: Anatomic and metabolic correlation. *Surg Neurol* 1994;41:147–151.
27. Heimburger FR: The cerebellum and spasticity. *Int J Neurol* 1970;7:232–243.
28. Albright AL, Cervi A, Singletary J: Intrathecal baclofen for spasticity in cerebral palsy. *JAMA* 1991;25:1418–1422.
29. Fasano VA, Broggi G, Barolat-Romana G, Sguazzi A: Surgical treatment of spasticity in cerebral palsy. *Childs Brain* 1978;4:289–305.
30. Peacock WJ, Staudt LA: Functional outcomes following selective posterior rhizotomy in children with cerebral palsy. *J Neurosurg* 1991;74:380–385.
31. Ohye C, Miyazaki M, Hirai T, et al: Stereotactic selective thalamotomy for the treatment of tremor type cerebral palsy in adolescence. *Childs Nerv Syst* 1983;10:157–167.
32. Maciunas RJ, Galloway RL Jr, Latimer J, et al: An independent application accuracy evaluation of stereotactic frame systems. *Stereotact Funct Neurosurg* 1992;58:103–107.
33. Sumanaweera TS, Glover GH, Hemler PF, et al: MR geometric distortion correction for improved frame-based stereotaxic localization accuracy. *Magn Reson Med* 1995;34:106–113.
34. De Salles AAF, Hariz M: Functional radiosurgery, in De Salles AAF, Goetsch SJ (eds): *Stereotactic Surgery and Radiosurgery.* Madison, WI, Medical Physics, 1993, pp 389–406.
35. Talairach J, Tournoux P: *Co-planar Stereotactic Atlas of the Human Brain.* New York, Thieme, 1988, p 99.
36. Speelman JD, van Manen J: Cerebral palsy and stereotactic neurosurgery: Long term results. *J Neurol Neurosurg Psychiatry* 1989;52:23–30.
37. Broggi G, Angelini L, Bono R, et al: Long term results of stereotactic thalamotomy for cerebral palsy. *Neurosurgery* 1983;12:195–202.
38. Laitinen LV: Neurosurgery in cerebral palsy. *J Neurol Neurosurg Psychiatry* 1970;33:513–518.