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Brain Plasticity and Cortical Dysplasia in Epilepsy: A Common Misconception for Epilepsy Surgery in Children

Fred C C Peng*

Department of Neurosurgery and Neurological Institute, Taipei Veterans General Hospital, Taiwan

***Corresponding Author**: Fred C C Peng, Department of Neurosurgery and Neurological Institute, Taipei Veterans General Hospital, Taiwan.

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Abstract

Purpose: This article is the second one in a series of two, following the publication of "Dementia in Epilepsy: A Clinical Contribution to the Metabesity of Epileptology, Geriatrics and Gerontology" as the first one in which metabesity regarding dementia in epilepsy is included. This second article, therefore, discusses two things: (1) Is the concept of brain plasticity clearly understood by clinicians in neurology and neurosurgery as well as other neuroscientists throughout the world? (2) Is the objective of making patients seizure-free sufficient to warrant epilepsy surgery of any form regardless of the post-operative consequences?

Methods: This second article was inspired or provoked by the results of epilepsy surgery in children reported extensively in Taipei by Dr. A. Simon Harvey of Melbourne, Australia, and the discussions which took place afterwards and during a lunch break with a female neurologist.

Results: As a behavioral neuroscientist, I was horrified by the detailed visual illustrations of the surgical procedures, including EEG and other vivid illustrations of MRI, especially the results of epilepsy surgery to remove multiple cortical dysplasias in children, resulting in frontal lobectomy and/or hemispherectomy. I was also completely bewildered and dumbfounded during a lunch break following the lecture in a casual non-provoked discussion with a pediatric neurologist and a female neurologist, whose erroneous comments on brain plasticity triggered the incentive of presenting this article.

For this reason, I shall discuss what brain plasticity means and present a brief description of neuroanatomy targeting the comments made after the presentation, as I was shocked to become speechless while hearing the female neurologist to boast her knowledge regarding the aftermath of lobectomy and/or hemispherectomy at the age of "seven" then corrected to "five" with no consequential deficits on account of brain plasticity. She seemed to think that brain plasticity is something like food stored in a refrigerator, which can be taken out for consumption during the age of five years. However, she added that adults are excepted because there is no more brain plasticity, that is, no more plasticity (food) is left in the "refrigerator". The pediatric neurologist sitting next to me was apparently also speechless.

Conclusion: I then immediately got the impression that brain plasticity is not at all clearly understood by clinical practitioners, not only in Taiwan but probably elsewhere throughout the world. This article is intended to reveal such a situation in medical practice and to clarify at the same time what brain plasticity means and what it entails anatomo-physiologically in neuroscience and to point out that any epileptic child or an adult undergoing lobectomy or, worse, hemispherectomy for whatever pathological reason, will become a vegetable as a consequence and the child will be short-lived for sure, thereafter, in spite of becoming seizure-free The choice by the parents between intractable seizures of their epileptic child and the unforeseeable consequences of their child becoming a vegetable, in spite of being seizure-free after the drastic surgery, is a very difficult one; once made, there is no going-back with regrets, as I shall show in this article.

Keywords: *Brain Plasticity; Cortical Dysplasia; Epilepsy*

Introduction

The nervous systems are well-connected structurally in adults in order to function properly and adequately to adjust to the internal and external environments. That is, they are structurally interrelated and functionally interdependent. As such, the priority requires that all neurons, especially in the brain, must be intact. Put differently, the nervous systems, starting from the neural tube embryologically, must fulfill four requirements: (1) the neurons at birth have the number in the brain completed, without undergoing further divisions; (2) there must have supporting cells called neuroglial cells or simply glial cells which are more numerous than neurons at the rate of 3 to one; but vary in different locations; (3) there must be arborization by which is meant that neurons must extend their processes, axons and dendrites, for interconnections throughout the brain; and (4) there also must be myelination which is facilitated by two types of glial cells, one in the CNS and other in the PNS.

Once these four anatomo-physiological requirements are met and well completed, the nervous system in the brain is said to have completed its growth at age 18 to reach its maturity which lasts for a while, from age 18 to 35, if not interrupted by any cause internally or externally. However, the maturation of brain functions after age 35 is to be followed gradually by another anatomo-physiological process known as aging; it is the ongoing process of wear and tear after maturity of the nervous systems; if for any reason apoptosis of whatever cause happens, inadvertently or not, to disrupt the nervous system, the disruption will affect the brain functions of memory and cognition among others to start the effects of dementia. It is during the flow of such anatomo-physiological processes, from birth to growth, followed by maturation, deterioration (aging), and dementia, because of the ongoing process of wear and tear, that the brain functions of memory and cognition and other body functions change, becoming deteriorated [1].

Where does brain plasticity fit then and how can it be characterized in these continuous anatomo-physiological processes of the brain and other body functions will be the main concern of this article. Simply put, it is these anatomo-physiological processes of (1) neuronal population, (2) growth of glial cells, (3) myelination, and (4) arborization where brain plasticity can be characterized; the first one strengthens the neuronal functions physiologically while the remaining three make sure that all neuronal processes, axons and dendrites, reach the correct locations and are well connected for proper functions. If not, cortical dysplasias will occur during the childhood, which become one form of epileptogenic focus for seizure attacks.

I shall devote the remaining part of this article to describe what such brain plasticity means, in order to characterize it, and what will happen if the nervous system is disrupted in any form to cause apoptosis. I shall also conclude whether or not the objective of making patients seizure-free is sufficient to warrant surgical treatment at all costs regardless of the consequences even at the expense of the patient becoming a vegetable; that is, whether or not such a surgical approach is scientifically, ethically, or even legally sound and justifiable. In so doing, I shall point out the legal consequences which are yet to be fully debated or even sufficiently accounted for.

Results and Discussion

Neuronal Population

It should be pointed out that at birth neurons in the nervous systems are completed in number, and therefore no more mitosis will take place. However, they must be properly distributed and located in the brain; or else, schizencephaly or megaloencephaly will occur; in rare case, anencephaly which means a baby is born without the cerebrum may happen--in the US 11 such cases have been recorded out of 100,000 births. Babies born in one of these conditions will be short-lived. See the case illustrated below that took place in Miami, Florida.

There was a female baby reported in Florida of anencephaly by the *Japan Times*, born without the cerebrum. The mother wanted to donate her organs by appealing to the court for permission. But she died in less than ten days after birth before the mother could get any permission from the court.

Neuroglial Population

Although there are more glial cells, at the rate of 3 to 1 compared to neuronal population, they start before birth, 16 weeks after conception, and continue to increase in number after birth to reach the rate. Five types of neuroglial cell are known in the literature: (1) Astroglial Cell (or Astrocyte), (2) Ependymal Cell, (3) Microglial Cell, (4) Oligodendrocyte, and (5) Schwann Cell.

Astroglial cells are found throughout the CNS. Each astrocyte is a star-shaped cell which has many processes, like a neuron, except that these processes extend from its cell body only to connect the surrounding nerve fibers in the neuronal network without emitting impulses. Their functions are (a) supply of nutrients to neurons, (b) removal of excess neurotransmissions. (c) maintenance of appropriate balances of Ca and K ions which are important for passing impulses at synapses; (d) because axons and dendrites are not well-connected nor are they protected, provision of a wrapping, myelination, is therefore needed; the wrapping of the axon with two coverings of myelin (fatty substance) and a sheath of Schwann cell (in the peripheral nervous system) take place; they form part of the myelin as it circumnavigates the axis cylinder of the axon; in the central nervous system, Schwann cells are replaced by oligodendrocytes for similar functions.

Ependymal cells are somewhat different, because they are found in the tissues of the CNS-- the brain and the spinal cord. Their functions are four: (1) protection by forming the lining of the ventricles of the brain and central canal of the spinal cord; (2) forming the CSF; (3) aiding circulation of CSF; and (4) providing the extremely thin membrane, known as ependyma which lines the ventricles of the brain and choroid plexuses. If for some reasons these functions fail, excessive CSF occurs to expand the ventricles, causing internal as well as external hydrocephaly which disrupt all brain functions.to the detriment of the individual. In that case, a surgical procedure called V-P Shunt is required to treat the hydrocephaly.

When the sheath of Schwann cell circumnavigates the axonal axis cylinder to form the myelin, there are spots where the axonal fibers are not myelinated; these spots are called Nodes of Ranvier. Nodes of Ranvier are very important for the transduction of impulses: they facilitate (i.e., speed up) the impulses in milliseconds; the fastest nerve can transmit impulses at the speed of 130 meters per second or 13 cm per millisecond.

Such a speed is important for many behaviors: 100-yard dash for athletes, fingering and bowing for violinists, and of course speaking in language behavior, for production; it is also very important for reception, as in fencing or *kung fu* and, most of all, listening (in debates, for instance). Imagine what will happen if a person takes minutes to complete a 100-yard dash or ten seconds, if not one minute, to utter a word. Or, if a person in fencing takes five seconds to see or even realize that the opponent's sword is coming, can he/she survive without being killed? But if an athlete has Parkinson's disease, like Mohammed Ali, the former heavyweight champion in boxing, he can no longer box. Or, if a debater takes s minute or so to realize what the opponent is saying, can he/she win the debate?

Be that as it may, high speed is not needed in cortico-cortical connections, especially for the short fibers (i.e., between gyri), where the axons are not heavily myelinated even though the neurons are multipolar neurons. I think that the less heavy myelination is compensated by the multipolarity of neurons in the cortices, and I am inclined to also speculate that there are two interdependent behavioral reasons for having multipolar neurons in the cerebral cortices: (1) function enhancement for the construction and reconstruction of meanings and (2) abstract thinking for forming thoughts in meaning during each extra-pyramidal looping. Both are pertinent to the brain functions of memory and cognition because neuronal axons are myelinated, subserved by CREB (cAMP-responsive element-binding) proteins at the same time. The former has to do with the functions of the six layers of cerebral cortex while the latter has to do with the extra-pyramidal looping in connection with the six layers of neurons for the cerebello-cerebral circuit, the limbic system, and the Papez circuit.

Arborization

Function enhancement and abstract thinking are not accomplished by the myelination of neuronal processes alone; there must be another important anatomo-physiological factor which takes place after birth. That is, all neuronal processes must be interconnected properly through arborization. Put differently, all neuronal processes in the two hemispheres extend to interconnect (1) downward in

each hemisphere from cortices in the cerebrum to subcortical structures, especially the inner-most structures, the brain stem, and the spinal cord, as well as (2) between the two hemispheres, inter-hemispherically, through the corpus callosum to form the corona radiata, and (3) between gyri in each hemisphere, laterally in the neocortex and medially in the paleocortex, as well as in the limbic lobe.

In the first interconnection, the arborization completes the circuit with the two basal ganglia and the brain stem, where the nucleus of each of ten cranial nerves is located, but also with the cortico-cerebellar and cerebellar-spinal connections. In the remaining two interconnections, the arborization completes the corona radiata horizontally through the corpus callosum and vertically to form the two internal capsules as well as to complete the cortico-striato-pallido-thalamo-cortical looping in each hemisphere, for forming thoughts and thinking, an important brain function which I call catalytic mappings of proto-meanings and sound images for production in oral language during the extra-pyramidal looping. The looping, after returning to the motor cortex, is connected to form the cortico-bulbar and cortico-spinal pathways through decussations for expression of behavior. In reception, however, the bulbo-cortical as well as the spino-cortical pathways in three steps through the thalamus reach back to the sensoricortex to enable the individual to reconstruct meanings for what he/she perceives, hears, and sees.

There are numerous publications regarding cognitive functions of the cerebellum which had been assigned to a mere function of voluntary motor control in the past. For instance, in 1958, Ray S. Snider wrote an article in *Scientific American*, introducing the cerebellum to its readers, by saying "In contrast to the cerebrum, where men have thought and found the centers of so many vital mental activities, the cerebellum remains a region of subtle and tantalizing mystery, its function hidden from investigators". The mystery has continued to be hidden beneath the notion of movement, never going beyond the assertion, until 17 years later, by Rodolfo R.

Llinás who confidently stated in the second *Scientific American* article on the cerebellum that "There is no longer any doubt that the cerebellum is a central control point for the organization of movement" The organization of movement, in my opinion, has very much to do with production and reception in the Individual Aspect of Language for both oral and sign language.

In 2003, however, there is a third article, by Bower and Parsons (2003: 41-7), in which they report that "the cerebellum's function has again become a subject of debate". On the basis of numerous sophisticated cognitive studies by cognitive neuroscientists, they also report that damage to specific areas of the cerebellum can cause unanticipated impairments in non-motor processes, especially in how quickly and accurately people perceive sensory information. I should add that such specific areas of the cerebellum play an important role in the neurophysiological activities of function enhancement for production and reception of behaviors.

The point in focus here, which is not mentioned by Bower and Parsons, is that the cerebellum is also heavily myelinated, although it has been known for decades that a single Purkinje cell in the cerebellum receives 150,000 to 200,000 inputs from the cerebrum, the pons, and the spinal cord through its peduncles, while the Purkinje cell provides the sole output of the cerebellar cortex. These inputs and outputs form enormously complex arborizations (interconnections of cerebellar neurons) which enable the cerebellum to play important roles for the proper adjustments to the internal and external environments beyond the mere motor control. These adjustments include memory and cognition (which are heads and tails of the same coin), attention, and emotion; other findings indicate the adjustments for the ability to schedule and plan tasks in dealing with the sensory inputs just mentioned and in processing information from such inputs (Schmahmann, 2003).

But there is an important point that has been missed in all observations of cerebellar functions; that is, these observations of brain functions, cerebral and cerebellar as well as other inner-most structures of the brain, always concentrate on production of behavior by neglecting reception of behavior, thereby missing a great deal of neurophysiological activities between reception and production, and resulting in the claim of "the centers of so many vital mental activities".

In so doing, such neurophysiological activities are truncated to the extent that only the results of production are considered by ignoring or even conceptually and, therefore, deliberately as well as naively, missing a great deal of cerebellar functions which are of utmost

relevance to reception of behavior in the proper adjustments to the internal and external environments. The fundamental reason for such outcomes is the built-in mind set of looking for a one-to-one correlation between each behavioral outcome and its underlying brain functions, known as regional differences. I shall return on another occasion to the description of contributions of cerebellar functions in behavior from the point of view of both production and reception, wherein I shall discuss "On Regional Differences of the Brain Functions: Fact or Fallacy?" as well as "The Brain Functions of Memory and Cognition Are Heads and Tails of the Same Coin".

Myelination and Demyelination

By now, the importance of myelination and arborization regarding brain functions should be clear. But what if myelin is damaged or destroyed? It is a disease of the nervous system known as demyelination. When a nerve is demyelinated, the consequence is the step-by-step destruction of neurons in the brain, because the nerve becomes hardened, resulting in a medical condition called multiple sclerosis (often abbreviated as MS). It may present many symptoms including: cognitive impairment (or difficulties), depression, impaired body movements, sexual dysfunction, slurred speech (because of impaired body movements), fatigue, dizziness or vertigo, bladder and bowel dysfunction. The individual can no longer walk, or use his/her arms (or hands), or talk, depending on where in the brain demyelination takes place.

When demyelination takes place in one of the temporal lobes (or in both lobes), causing sclerosis of the mesial part of the lobe, involving the hippocampus, the result is called mesial temporal tuberous sclerosis (or hippocampal sclerosis), which invariably triggers temporal lobe epilepsy (often abbreviated as TLE). The seizure attacks may vary from once a day or once a week to several seizure attacks per day. If the seizure attacks cannot be managed by medication, the epilepsy becomes intractable and requires surgical intervention, which may range from anterior hippocampectomy, or amygdalohippocampectomy, to temporal lobectomy, in order to make the patient seizure-free. Either pre- or post-operatively, the patient (intractable or non-intractable) has serious memory impairment; hence, he/she has language disorders ictally (i.e. during a seizure) or interictally (i.e., between seizures). See my "Dementia in Epilepsy: A Clinical Contribution to the Metabesity of Epileptology, Geriatrics, and Gerontology" (2017).

But what if arborization does not work properly as expected during childhood? The result is now known as cortical dysplasia; the consequence is another form of epileptofocus for seizure attacks. In the case of my discussion here, the point at issue is the surgical treatment of such cortical dysplasias, resulting in the consequences of frontal lobectomy of either hemisphere and/or hemispherectomy in children as reported by Dr. Simon Harvey; his aim is to make the patients seizure-free at all costs.

Three years or so ago, at the Annual Meeting of Taiwan Epilepsy Society, a serious debate took place between an epilepsy neurosurgeon from Taiwan, who advocated epilepsy surgery at all costs regardless of locations, and an epilepsy neurosurgeon from South Korea, who opposed vigorously the claim of surgery at all costs. I for one took part in the debate to side with the latter and pointed out that if epilepsy surgery makes the patient seizure-free at all costs regardless of locations, the patient will have a heck of a lot of psychiatric problems postoperatively to deal with. I was glad that most people in the audience, including the neurosurgeon in Taiwan, seemed to agree with me.

On another occasion, when a German epilepsy neurosurgeon was invited to Taipei Veterans General Hospital to give a lecture on surgery of Cortical Dysplasia in Germany, it was elegantly presented. During the Q and A session, I asked him: "What would you do if the patient postoperatively developed psychiatric problems?" His answer was, "We send the patient to psychologists".

It should therefore be clear to everybody by now that myelination as well as demyelination can change the brain functions of memory and cognition, and that alterations by epilepsy surgery at all costs to make the patient seizure-free regardless will definitely cause serious damage to the patient's postoperative behaviors, even to the irreparable detriment to the patient's brain functions of memory and cognition; in the case of frontal lobectomy or hemispherectomy, the patient may be seizure-free but will be short-lived postoperatively, becoming a vegetable before death. For this reason, I believe Dr. Simon Harvey did NOT dare mention in his lectures even a single word or show the behavioral result of a single case of the postoperative consequence of either frontal lobectomy or hemispherectomy.

It should be cautioned that myelin is not the source or origin of the brain functions of memory. However, proper arborization is the foundation of all brain functions; because the lack of such proper arborization for the interconnections of neurons or any disruptions of such interconnections in the nervous systems, surgically or otherwise, will result in serious consequences.

However, it would be absurd to correlate or associate, as many neuroscientists would do, a particular lesion or lesioned site in the brain (e.g., a hardened neural tissue in the case of MS) with a behavioral deficit or a group of such deficits (as indicated above), as if the lesioned site was originally responsible for the lost functions when it was intact, that is, before demyelination. Apparently, this is the mistake of seeking a one-to-one correlation that some geneticists have done; they attempted to associate the mutation of FOXp2, claimed to be the language gene they discovered on chromosome 7 in a small pedigree of English speakers, with the grammatical errors of the tense markers some of the speakers made; they then erroneously jumped to the conclusion that they discovered the language gene.

Such a mistake, as was pointed out above, is also quite prevalent among researchers in neuroscience. For instance, recently researchers from a research center in Taiwan claim that lesion of the amygdaloid complex nuclei in a mouse causes a behavioral alteration in the mouse that looks like autism of the mouse; they then assert that in humans such nuclei "control" socializing behavior and a damage to the human amygdaloid complex nuclei results in human autism. The claim misses the whole point, for there is no such thing as the center of socialization in the brain. Socialization involves a great deal of structures in the nervous system, central and peripheral, and autism in humans is not just lack of talking.

Brain Plasticity

What is brain plasticity, then? It starts from birth and continues to expand by way of increasing neuroglial cells 3 to 1 and through myelination and arborization to reach maturation as discussed above. Is there any limitation of brain plasticity? Yes, there is. It stops to expand when glial cells for myelination and arborization in the nervous systems are complete, but ependymal cells must remain active even in adults. The age of completion varies from one individual to another but I shall call this period, from birth till the completion of myelination and arborization, the period of growth which starts from birth till age 18 [2,3].

One important aspect of arborization is that all neurons must be in their proper places before birth in order to be ready for the total arborization after birth. During fetal life before birth, neurons are specialized; those which have similar functions 'stick together' to form a cluster or a layer (especially in the cerebral cortex and the cerebellum) or a ganglion (in the basal ganglia or the peripheral nervous system).

I have already mentioned gyri, each one of which is a strip of neurons sticking together to form a fold. As there are many such folds, they are divided by ditches, so that the folds look like 'wrinkles'. The more wrinkled up the brain surface is the more advanced the brain functions are. Thus, the brain of a lower form of vertebrates, such as a chicken, does not have gyri and an adult chimpanzee's brain has far fewer gyri. than does an adult human brain.

Within each gyrus the neurons have come into their respective positions lined up in the cerebral cortices or stratified in six layers during fetal life. Mechanically, it may be thought that neurons of the first (top) layer will take their position first and neurons of the last (bottom) layer will take their position last. However, it has been found recently by researchers from Stanford that the actual ordering of layers in fetal life is the reverse of the 'common sense' mechanical ordering. It was shown at one of the annual conferences of the American Epilepsy Society with a video taken from a lab that neurons of the bottom layer take their position first and neurons of the fifth (i.e., second to the bottom) layer take their position next, and so on, and finally neurons of the first layer take their position last. After that, all neurons begin to arborize, that is, extend their processes vertically and horizontally, to interconnect with one another.

Now, what will happen to the fetus if neurons during the developing stages, collectively called neurogenesis, are misplaced, or do not get to their appropriate positions in time, or are overproduced? It is known that the growth in all animals takes place in two processes:

the cells within the organism increase either in number (through mitosis), called hyperplasia, or in size, called hypertrophy. The brain is no exception. But the neurons in the brain, in addition, must migrate to their appropriate positions in time. If not, the consequences are very serious. Examples as I have mentioned are cortical dysplasia, as in double cortex (for gray matter), cortical dysplasia of white matter, megaloencephaly (enlarged cortex), or schizencephaly (one oversized hemisphere), or progeria (premature aging syndrome, which is caused by a mutation of DNA arising from unknown factors).

Progeria, however, is a very rare disease, one in every eight million infants, and there are only about 40 such reported cases. The newborn at first looks 'normal' but ages very fast, at the pace of one year equivalent to 8-10 years in normal aging. Thus, a child with progeria at the physical age of 10, if he/she lives that long, would look like an old person of 80, although that individual would have no problem learning the rudimentary form of a language so long as there is no other neurological complication in his/her brain, for instance, a stroke, just as an old person has learned his/her language but may have a stroke; the difference is that the child with progeria has reached the age of 80 ten times faster than a normal person. In such cases, there is no brain plasticity of any kind, as the fetus in such conditions, if born, will be short-lived.

However, in the case of multiple cortical dysplasias of white matter, the baby born will have pediatric epilepsy which was what Dr. Simon Harvey's lectures were all about. His method to make those children seizure-free was epilepsy surgery to remove those tubers at all costs, each one of which refers to the shape of the sclerosis, a scar, caused by the cortical dysplasias.

The Consequences of Epilepsy Surgery to Remove Tubers

The removal of such multiple cortical dysplasias surgically through frontal lobectomy or hemispherectomy will make the patient postoperatively a vegetable, albeit seizure-free for a few years, because the patient will be short-lived. Let me, therefore, point out the damages of brain structures and tissues, first through hemispherectomy and then through frontal lobectomy.in the pretext of making the patient seizure-free. However, in the absence of detailed surgical data from Dr. Harvey, I can only grossly summarize the brain structures and tissues surgically damaged and the behavioral consequences.

The Consequences of Hemispherectomy

Before I point out the destructions of brain tissues and structures, I should mention that the child's cranium is grossly altered; that is, one big piece of the skull must be removed, leading to a big concavity of the skull, one half of the cranium, which undoubtedly alters the facial expressions postoperatively. Not only that, beneath the concavity, let me now mention the destructions of the following brain tissues and structures:

(1) One side of the corona radiata no longer exists, with probable damage to the corpus callosum, thereby cutting off laterally any inter-hemispheric commissures, good and bad, as there are no longer any brain tissues left from the lesioned side to the intact side.

(2) In so doing, the limbic lobe of the removed hemisphere no longer exists, thereby affecting the patient's limbic system and the Papez circuit, which overlap structurally and are interrelated functionally, for postoperative behaviors.

(3) Vertically the internal capsule of the lesioned side as an extension of the original intact corona radiata is removed, thereby affecting one side of the anterior and posterior limbs of the internal capsule.

(4) One of the two basal ganglia-the lesioned side – is destroyed surgically, disrupting the extra pyramidal functions of the cortico-striato-pallido-thalamo-cortical looping for the catalytic mappings needed for thinking and formation of thoughts.

(5) As a result, the connections of cortico-bulbar and cortico-spinal pathways are disrupted, affecting one side of the ten pairs of cranial nerves in the brain stem, thereby jeopardizing the patient's articulation of oral language.

(6) If the first and second cranial nerves of the lesioned side are also affected, because there is no report from Dr. Harvey regarding details of the epilepsy surgery other than a gross mention and a slide of hemispherectomy, the child becomes postoperatively hemianopia and the loss of one side of olfactory function.

(7) Since one hemisphere is completely removed to stop the child's seizure attacks at all costs, the patient's sensorimotor cortices of the lesioned side are destroyed, thereby making the child grossly hemiplegic postoperatively.

The Consequences of Frontal Lobectomy

Either it is the left or right frontal lobe that was removed, the consequences are also very serious, coupled with some of the consequences already mentioned above, resulting from destructions of the corona radiata, one of the internal capsules, as well as the first and second cranial nerve in the lesioned side.

(1) Hemiplegia of the limbs opposite to the lesioned side follows.
(2) Disruption of the cortico-striato-pallido-thalamo-cortical looping, which grossly damages the child's brain functions of memory and cognition.
(3) As the motor cortex of the lesioned side as well as the supplemental motor area no longer exist, the cortico-bulbar pathways and the cortico-spinal pathways are disrupted, affecting the brain stem for language production (articulation), and body movements for gesticulation, walking, and/or even eating.

Conclusion

Given the descriptions above, several serious questions arise. (1) Should epilepsy surgery be performed to make the patient seizure-free at all costs? (2) If so, should epilepsy neurosurgeon have the obligation to explain in detail to the parent(s) the postoperative consequences in the child's postoperative behavior? (3) In the case of frontal lobectomy and hemispherectomy, is the neurosurgeon aware of the patient's postoperative consequence as a vegetable or can the neurosurgeon anticipate such a consequence? (4) Who should bear the legal or moral responsibility if such a postoperative consequence happens?

I do not think there is any sound answer to each of these questions, because these questions involve legal controversies which may not be anticipated by either the neurosurgeon performing the surgery or the patient's parent(s). The reason is twofold: (1) the eagerness to stop the seizure attacks, at all costs on the part of the surgeon and the parents, as well as (2) the lack of comparison between the pains to see the child's repeated seizure attacks preoperatively and the burden and regrets of seeing postoperatively the child's behavioral consequences of becoming a vegetable, albeit seizure-free. When the postoperative consequence happens, and the parents are shocked when facing it, there is no way in which to regret. Of course, the neurosurgeon will only be glad to see that the objective of making the patient seizure-free is accomplished, an objective that is kept by the neurosurgeon as his/her mind-set for a great success, whereas the alternative view of the "success" may be a butcher's deed.

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