Nervous System Surgeries and Procedures

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Chapter 1

Endoscopic Thoracic Sympathectomy and Neurosurgery

Endoscopic thoracic sympathectomy

Intervention:
Endoscopic thoracic sympathectomy

ICD-10 code:
ICD-9 code: 05.2
Other codes:

Endoscopic thoracic sympathectomy (ETS) is a surgical procedure where certain portions of the sympathetic nerve trunk are destroyed. ETS is used to treat hyperhidrosis, facial blushing, Raynaud's disease and reflex sympathetic dystrophy. By far the most common complaint treated with ETS is palmar hyperhidrosis, or "sweaty palms". In this disorder, the palms may constantly shed so much sweat that the affected person is unable to handle paper, sign documents, keep clothes dry, or shake hands. The result is often social phobia so severe as to be disabling.

Sympathectomy physically destroys some tissue anywhere in either of the two sympathetic trunks, which are long chains of nerve ganglia lying along either side of the spine. Each nerve trunk is broadly divided into three regions: cervical (neck), thoracic (chest), and lumbar (lower back). The most common area targeted in sympathectomy is the upper thoracic region, that part of the sympathetic chain lying between the first and fifth thoracic vertebrae.

In addition to the normal risks of surgery, such as bleeding and infection, sympathectomy has several specific risks, such as adverse changes in how nerves function.

Indications

ETS is most commonly used to treat severe hyperhidrosis of the upper body, Raynaud's phenomenon, and facial blushing.

There are reports of ETS being used to achieve cerebral revascularization for patients with moyamoya disease, and to treat headaches, hyperactive bronchial tubes, long QT syndrome and other conditions.

Thoracic sympathectomy can alter many bodily functions, including sweating, vascular responses, heart rate, heart stroke volume, thyroid, baroreflex,lung volume, pupil dilation, skin temperature, goose bumps and other aspects of the autonomic nervous system, like the fight-or-flight response. It may diminish the body's physical reaction to exercise.

Procedure

Sympathectomy involves division of adrenergic, cholinergic and sensory fibers which elaborate adrenergic substances during the process of regulating visceral function. It involves dissection of the main Sympathetic trunk in the upper thoracic region of the sympathetic nervous system, thus interrupting neural messages that ordinarily would travel to many different organs, glands and muscles. It is via these nerves of the autonomic nervous system that the brain is able to make adjustments in the body in response to changing conditions in the environment, changing emotional states, level of exercise, and other factors to maintain the body's homeostasis.

When performed endoscopically, the surgeon penetrates the chest cavity, making holes about the diameter of a straw between ribs. This allows the surgeon to insert the video camera in one hole and a surgical instrument in another.

Sympathectomy is accomplished by dissecting the nerve tissue of the main sympathetic chain. The clamping method, also referred to as **endoscopic sympathetic blockade** (ESB) employs titanium clamps around the nerve tissue, and was developed in an attempt to make the procedure reversible. However, reversal of the clamping procedure must be performed within a short time after clamping (a few days or weeks at most), and recovery may not be complete.

Results

The most common indication for ETS surgery is hyperhidrosis, or excessive sweating. However, one study on sweating before and one month after ETS demonstrated that the procedure increases total sweat production in a hot sauna.

Swedish National Board of Health and Welfare statement on treatment results says: "A large amount of international studies shows that an incision on the sympatikotomi nerve gives a very positive result when it comes to hand perspiration and also that the side

effects are rare." Critics have raised serious questions about the methodology of such studies.

Sympathectomy works by disabling part of the autonomic nervous system, by surgically destroying it, and disrupting the signals to the brain. Many non-ETS doctors find this to be disturbing, as symptoms of the ANS dysfunction go further than the disabled thermoregulation. Sympathectomy prevents the occurrence of a variety of bodily changes, and hence, prevents sensory feedback of those changes.

Exact results of ETS are impossible to predict, because of considerable anatomic variations in sympathetic nerve function from one patient to the next, and also because of variations in surgical technique. The autonomic nervous system is not anatomically exact and connections might exist with different parts of the body. This theory has been proven by the fact that a significant number of patients who have had sympathectomy for hand sweating might notice a reduction or elimination of feet sweating. No reliable operation exists for foot sweating per se except lumber sympathectomy.

Lumbar sympathectomy is largely of historical interest today, being reserved for cases of severe sympathetic dystrophy or selected cases of rest pain, where is it usually done by percutaneous ablation of the lumbar sympathetic chain by phenol injection under imaging guidance. Its original use as an operation for lower limb ischaemia has been superseded by direct revascularisation operations or endovascular revacularisation procedures such as angioplasty or angioplasty with stenting of occuded arteries with reasonable runoff i.e. endovascular surgery.

Studies by ETS surgeons have claimed an initial satisfaction rate around 85-95% with at least 2%-19% regretting the surgery and up to 51% of the patients complaining about decreased quality of life. One study shows a satisfaction rate as low as 28.6. Most patients report various adverse reactions as a result of the surgery. However, ETS surgeon Samuel S. Ahn of UCLA claims "100% success with no negative side effects".

A large study of psychiatric patients treated with this surgery showed significant reductions in fear, alertness and arousal. (Teleranta, Pohjavaara, et al. 2003, 2004). Arousal is essential to consciousness, in regulating attention and information processing, memory and emotion. This study also proves what many patients have claimed, that the surgery caused psychological changes. You cannot reduce 'bad' emotional responses, like fear or anxiety. If you reduce emotional responses, they will affect the whole range of emotions and their intensity. With the elimination of the heart rate variability, emotions are also 'capped'.

ETS patients are being studied using the autonomic failure protocol headed by David Goldstein, M.D. Ph.D., senior investigator at the U.S National Institute of Neurological Disorders and Stroke. He has documented loss of thermoregulatory function, cardiac denervation, and loss of vasoconstriction. Recurrence of the original symptoms due to nerve regeneration or nerve sprouting can occur within the first year post surgery, but regeneration can start years after sympathectomy. Nerve sprouting, or abnormal nerve

growth after damage or injury to the nerves can cause other further damage. Sprouting sympathtetic nerves can form connections with sensory nerves, and lead to pain conditions that are mediated by the SNS. Every time the system is activated, it is translated into pain. This sprouting and its action can lead to Frey's syndrome, a well recognized after effect of sympathectomy, when the growing sympathetic nerves innervate salivary glands. This leads to excessive sweating when eating. For patients different tastes can trigger this abnormal facial sweating (curiously this happens in the area where people who have undergone this procedure can not sweat any more normally). For some it only occurs with hot food, for others, with hot, sour - even by eating an apple, or sweet. Smelling can also cause abnormal reactions, as the signals get mixed up. Nerve regeneration and subsequent abnormal synapses is a well-documented phenomena.

Some patients have required an artificial pacemaker after developing bradycardia (slow heart beat) as a side effect of the surgery.

Risks and controversy

No surgery is risk-free, and ETS has both the normal risks of surgery, such as bleeding and infection, and several specific risks, such as changes in how nerves function. Bleeding during and following the operation may be significant in up to 5% of patients. Pneumothorax (collapsed lung) can occur (2% of patients).

Compensatory hyperhidrosis (sweating) is common over the long term, causing 1-2 percent of patients in one review to regret having had the surgery. The rates of severe compensatory sweating vary widely between studies, ranging from as low as 1.2% and as high as 30.9% of patients. Of those patients that develop this side effect, about a quarter said it was major and disabling.

ETS can cause corposcindosis, in which the patient feels like he or she is living in two separate bodies: one half of the body is numb or "dead," and the other half has hyperactive sympathetic nerve function.

The Finnish Office for Health Care Technology Assessment concluded in a 40 page systematic review that Endoscopic Thoracic Sympathectomy is associated with significant immediate and long-term adverse effects.

Quoting the aforementioned Swedish National Board of Health and Welfare statement: "The method can give permanent side effects that in some cases first will become obvious after some time. One of the side effects might be increased perspiration on different places on your body. Why and how this happens is still unknown. According to the research available about 25-75% of all patients can expect more or less serious perspiration on different places on their body, such as the trunk and groin area, this is *Compensatory sweating*.

However, it is also mentioned in the research that 0-10% regret having the surgery done for this reason. Other documented side effects are the inability to raise the heart rate when

working out physically. This has in some cases led to decreased ability to perform your work and daily activities. Some patients also complained of not being able to control their body temperature and it is experienced from being very uncomfortable to disabling. However description of a changing sweating pattern does not give a comprehensive picture of the permanently disabled thermoregulation. Consequences of this go far beyond some discomfort wearing damp, in some cases dripping clothes and showing up in public.

A reduced efficiency in maintaining normal body temperature in warm environments is consistent with the reduced ability or complete inability to sweat above the nipple line, a common ETS outcome first shown by Dr. Kotzareff. For a fully clothed person, only the hands, cranial region and neck are typically exposed. In a hot environment, a normal person's body is cooled primarily by evaporation of water vapor through the warmest areas of exposed skin. These areas are associated with the head and neck, which under very warm circumstances or vigorous exercise, visibly show moisture (sweat) accumulating as part of the cooling process. For an ETS patient that has lost ability to sweat from cranium, neck, and arms, an increased amount of body heat must be rejected via transpiration/sweating involving skin of the lower body. Unfortunately, this skin is generally at a lower temperature and usually covered by clothing - both factors that reduce the cooling efficiency and result in poor thermoregulation. An uncomfortably warm sensation and accumulation of sweat on large areas of skin underneath clothing can result. This is one theory on the aetiology of the increased sweating phenomenon after sympathectomy. However one of the pioneers of the procedure, Dr Lin, who performed over 7000 procedures, disputes the compensatory nature of the so called Compensatory Sweating. According to him this is a result of the dysregulated thermoregulation and hypothalamus. He objects to using the "Compensatory" term, he sees as misleading. Postoperative sweating phenomenon is a reflex response between sympathetic system and Hypothalamus. "It is absolutely not a compensatory mechanism. The term of “Reflex sweating” instead of compensatory sweating is used. Hypothalamus is the center of Autonomic Nervous System, which influences human mind, mentality and endocrine system. For this sake, Dr. Lin emphasized, “Endoscopic Sympathetic Surgery helps us open a gate to Autonomic Nervous System”.

There is much disagreement among ETS surgeons about the best surgical method, optimal location for nerve dissection, and as to the nature and extent of the consequent primary effects and side effects. The internet now features many websites run by surgeons extolling the benefits of ETS backed by patient testimonials. However, there are also many websites run by disabled ETS victims who complain of severe adverse reactions and lack of adequate informed consent. Several online discussion forums are dedicated to the subject of ETS surgery, where both positive and negative patient testimonials abound, but considering that this is an elective surgery for a benign condition, even a small number of badly affected number of patients is a high number.

In 2003, ETS was banned in its birthplace, Sweden, due to overwhelming complaints by disabled patients. In 2004, Taiwanese health authorities banned the procedure on patients under 20 years of age. In other countries it is highly unregulated procedure. Although it

was never evaluated for safety and adverse effects, sympathectomy is listed on Medical Benefits Scheme, and is freely available to public patients.

In 2006, the FinOHTA group, the Finnish Office for Health Technology Assessment, showed in a review that there were strong indications of side effects as a result of this surgery.

• *No systematic reviews, meta-analyses, or clinical trials that evaluated the* effectiveness of endoscopic thoracic sympathectomy for treating facial blushing were identified. However, we have identified four case series related to the request (Drott et al. 1998, Rex et al. 1998, Telaranta 1998, Yilmaz et al. 1996). These studies were conducted in three countries (Sweden, Finland and the Netherlands).

• The four case series were not critically appraised because they are prone to bias and have significant methodological problems. These studies represent level IV evidence according to the NHMRC criteria and one should not draw firm conclusions from their findings.

• To date, the benefits or side effects associated with endoscopic thoracic sympathectomy for treating facial blushing have not been properly evaluated and reported. (Omar Ahmed PhD Centre for Clinical Effectiveness Monash Medical Centre Australia*)*

Other long term adverse effects: Ultrastructural Changes in the Cerebral Artery Wall Induced by Long-Term Sympathetic Denervation Sympathectomy eliminates the psychogalvanic reflex Cervical sympathectomy reduces the heterogeneity of oxygen saturation in small cerebrocortical veins Sympathetic denervation is one of the causes of Mönckeberg's sclerosis T2-3 sympathectomy suppressed baroreflex control of heart rate in the patients with palmar hyperhidrosis. We should note that baroreflex response for maintaining cardiovascular stability is suppressed in the patients who received the ETS. ETS patients should be warned that these mechanisms may play a role in the development of exertional heat stroke. Morphofunctional changes in the myocardium following sympathectomy.

In none of the limbs studied after sympathectomy could an increase in blood flow be produced reflexly by warming; in the majority of instances the opposite response, a decrease in blood flow was observed. One patient with documented transection of the spinal cord above T5 behaved like subjects after surgical sympathectomy. Retarded adaptation of hemodynamics to a sudden start of exercise after sympathectomy. The significant fall in left circumflex coronary flow was proportional to the decline in external heart work due to sympathectomy both at rest and under exercise. Chemical sympathectomy is associated with increased pulmonary metastases.

History

Sympathectomy developed in the mid-19th century, when it was learned that the autonomic nervous system runs to almost every organ, gland and muscle system in the

body. It was surmised that these nerves play a role in how the body regulates many different body functions in response to changes in the environment, exercise and emotion.

The first sympathectomy was performed by Alexander in 1889. Since the sympathetic nervous system was well known to affect many body systems, the surgery was performed in attempts to treat many conditions, including idiocy, goitre, epilepsy, glaucoma, and angina pectoris. Thoracic sympathectomy has been indicated for hyperhidrosis (excessive sweating) since 1920, when Kotzareff showed it would cause anhidrosis (total inability to sweat) from the nipple line upwards.

A lumbar sympathectomy was also developed and used to treat excessive sweating of the feet and other ailments, and typically resulted in impotence in men. Lumbar sympathectomy is still being offered as a treatment for plantar hyperhidrosis, or as a treatment for patients who have a bad outcome (extreme 'compensatory sweating') after thoracic sympathectomy for palmar hyperhidrosis or blushing; extensive sympathectomy risks hypotension.

Sympathectomy itself is relatively easy to perform; however, accessing the nerve tissue in the chest cavity by conventional surgical methods was difficult, painful, and spawned several different approaches. The posterior approach was developed in 1908, and required resection (sawing off) of ribs. A supraclavical (above the collar-bone) approach was developed in 1935, which was less painful than the posterior, but was more prone to damaging important nerves and blood vessels.

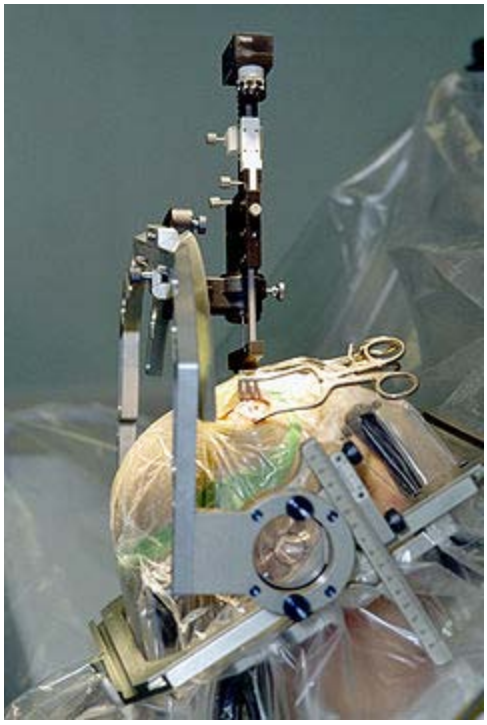
Because of these difficulties, and because of disabling sequelae associated with sympathetic denervation, conventional or "open" sympathectomy was never a very popular procedure, although it continued to be practiced for hyperhidrosis, Raynaud's disease, and various psychiatric disorders. With the popularization of lobotomy in the 1940s, sympathectomy fell out of favor as a form of psychosurgery.

The endoscopic version of thoracic sympathectomy was pioneered by Goren Claes and Christer Drott in Sweden in the late 1980s. The development of endoscopic "minimally invasive" surgical techniques have decreased the recovery time from the surgery and increased its popularity. Today, ETS surgery is practiced in many countries throughout the world.

In the mid-1990s a group of Swedish ETS patients complaining of disabling side effects formed the organization FFSO (people disabled by sympathectomy). The group grew to over 300 members and their work led to the procedure being banned in Sweden. The two surgeons who pioneered the technique, Drott and Claes, moved their practice from Sweden. They still perform the surgery.

Neurosurgery

Neurosurgery



Occupation	
Activity sectors	Surgery
Description	
Education required	Doctor of Medicine
Fields of employment	Hospitals, Clinics

Neurosurgery (or **neurological surgery**) is the medical specialty concerned with the prevention, diagnosis, treatment and rehabilitation of disorders that affect any portion of the nervous system including the brain, spinal column, spinal cord, peripheral nerves, and extra-cranial cerebrovascular system.

Education and training

In the United States, a neurosurgeon must generally complete four years of college, four years of medical school, a year-long internship (PGY-1) that is usually affiliated with their residency program, and five to six years of neurosurgery residency (PGY-2-6). Most, but not all, residency programs have some component of basic science or clinical

research. Neurosurgeons may pursue an additional training in a fellowship, after residency or in some cases, as a senior resident. These fellowships include pediatric neurosurgery, neurocritical care, functional and stereotactic surgery, surgical neuro-oncology, neurovascular surgery, Interventional neuroradiology, or skull base surgery. Neurosurgeons can also pursue fellowship training in neuropathology and neuro-ophthalmology.

In the UK students must earn A*- C Grades at GCSE (General Certificate of Secondary Education), then they must also achieve A*- C at A levels in Chemistry with at least one other Science or Maths. Also a UKCAT (UK Clinical Aptitude Test) or BMAT (BioMedical Admissions Test) can be used to gain access into some Medical Schools. Students have to study medicine for 5 years and achieve an MBBS qualification (Bachelor of Medicine and Bachelor of Surgery). Then the student must perform Foundation training lasting normally 2 years, this is a paid training job in a hospital or clinical situation setting covers a range of Medical specialties including Surgery. Core Surgical training is then taken which lasts for 2 years the difference in this is that the training would be themed towards a particular speciality.

Neurosurgical methods

Neuroradiology methods are used in modern neurosurgical diagnosis and treatment. computer assisted imaging computed tomography (CT), magnetic resonance imaging (MRI), positron emission tomography (PET), magnetoencephalography (MEG) and the development of stereotactic surgery. Some neurosurgical procedures involve the use of MRI and functional MRI intraoperatively.

Microsurgery is utilized in many aspects of neurological surgery. Microvascular anastomosis are required when EC-IC surgery is performed. The clipping of aneurysms is performed using a microscope. Minimally invasive spine surgery utilizes relies on these techniques. Procedures such as microdiscectomy, laminectomy, and artificial discs rely on microsurgery.

Minimally invasive endoscopic surgery is utilized by neurosurgeons. Techniques such as endoscopic endonasal surgery is used for pituitary tumors, craniopharyngiomas, chordomas, and the repair of cerebrospinal fluid leaks. Ventricular endoscopy is used for colloid cysts and neurocysticercosis. Endoscopic techniques can be used to assist in the evaculation of hematomas and trigeminal neuralgia. Repair of craniofacial disorders and disturbance of cerebrospinal fluid circulation is done by neurosurgeons, and depending on the situation, plastic surgeons. Conditions such as chiari malformation, craniosynostosis, and syringomyelia are treated. This is called cranioplasty.

Neurosurgeons are involved in Stereotactic Radiosurgery along with Radiation Oncologists for tumor and AVM treatment. Radiosurgical methods such as Gamma knife and Cyberknife are used.

Neurosurgeons have begun to utilize endovascular image guided procedures for the treatment of aneurysms, AVMs, carotid stenosis, strokes, and spinal malformations, and vasospasms. Also, nonvascular procedures such as Vertoplasty and Kyphoplasty are used by neurosurgeons. Techniques such as angioplasty, stenting, clot retrieval, embolization, and diagnostic angiography are utilized.

Conditions

Other conditions treated by neurosurgeons include:

- Spinal disc herniation
- Cervical spinal stenosis and Lumbar spinal stenosis
- Hydrocephalus
- Head trauma (brain hemorrhages, skull fractures, etc.)
- Spinal cord trauma
- Traumatic injuries of peripheral nerves
- Infections
- Tumours of the spine, spinal cord and peripheral nerves
- Intracerebral hemorrhage, such as subarachnoid hemorrhage, intraparenchymal, and intraventricular hemorrhages
- Some forms of drug-resistant epilepsy
- Some forms of movement disorders (advanced Parkinson's disease, chorea) – this involves the use of specially developed minimally invasive stereotactic techniques (functional, stereotactic neurosurgery) such as ablative surgery and deep brain stimulation surgery
- Intractable pain of cancer or trauma patients and cranial/peripheral nerve pain
- Some forms of intractable psychiatric disorders
- Vascular malformations (i.e., arteriovenous malformations, venous angiomas, cavernous angiomas, capillary telangectasias) of the brain and spinal cord
- Peripheral neuropathies such as carpal tunnel syndrome and ulnar neuropathy
- Moyamoya disease

Chapter 2

Craniotomy and Decompressive Craniectomy

Craniotomy

Intervention:
Craniotomy

ICD-10 code:
ICD-9 code: 01.2
Other codes:

A **craniotomy** is a surgical operation in which a bone flap is (temporarily) removed from the skull, to access the brain. Craniotomies are often a critical operation performed on patients suffering from brain lesions or traumatic brain injury (TBI), and can also allow doctors to surgically implant deep brain stimulators for the treatment of Parkinson's disease, epilepsy and cerebellar tremor. The procedure is also widely used in neuroscience for extracellular recording, brain imaging, and for neurological manipulations such as electrical stimulation and chemical titration.

Human craniotomy is usually performed under general anesthesia but can be also done with the patient awake using a local anaesthetic; the procedure generally does not involve significant discomfort for the patient. In general, a craniotomy will be preceded by an MRI scan which provides a picture of the brain that the surgeon uses to plan the precise location for bone removal and the appropriate angle of access to the relevant brain areas. The amount of skull that needs to be removed depends to a large extent on the type of surgery being performed. The bone flap is then replaced using titanium plates and screws or another form of fixation (wire, suture, ...etc).

Craniotomy is distinguished from craniectomy (in which the skull flap is not immediately replaced, allowing the brain to swell, thus reducing intracranial pressure) and from trepanation, the creation of a burr hole through the cranium in to the dura mater.

Decompressive craniectomy

Intervention:
Decompressive craniectomy

ICD-10 code:
ICD-9 code: 01.2
Other codes:

Decompressive craniectomy is a neurosurgical procedure in which part of the skull is removed to allow a swelling brain room to expand without being squeezed. It is performed on victims of traumatic brain injury and stroke. Use of the surgery is controversial.

The procedure evolved from a primitive form of surgery known as trephining or trepanning. The older procedure, while common in prehistoric times, was deprecated in favor of other, less invasive treatments as they were developed; although it was still performed with some frequency prior to the twentieth century, its resurgence in modern form became possible only upon the development of precision cutting tools and sophisticated post-operative care such as antibiotics.

Results of clinical trials

Reduction of intracranial pressure

Though the procedure is considered a last resort, some evidence suggests that it does improve outcomes by lowering intracranial pressure (ICP), the pressure within the skull. Raised intracranial pressure is very often debilitating or fatal because it causes compression of the brain and restricts cerebral blood flow. The aim of decompressive craniectomy is to reduce this pressure. The part of the skull that is removed is called a bone flap. A study has shown that the larger the removed bone flap is, the more ICP is reduced.

Other effects

In addition to reducing ICP, studies have found decompressive craniectomy to improve cerebral perfusion pressure and cerebral blood flow in head injured patients.

Decompressive craniectomy is also used to manage major strokes, associated with "malignant" edema and intracranial hypertension. The pooled evidence from three randomised controlled trials in Europe supports the retrospective observations that early (within 48 hours) application of decompressive craniectomy after "malignant" stroke may result in improved survival and functional outcome in patients under the age of 55, compared to conservative management alone.

The procedure is recommended especially for young patients in whom ICP is not controllable by other methods. Age of greater than 50 years is associated with a poorer outcome after the surgery.

Complications

Infections such as meningitis or brain abscess can occur after decompressive craniectomy.

Children

In severely head injured children, a study has shown that decompressive craniectomy resulted in good recovery in all children in the study, suggesting the procedure has an advantage over non-surgical treatment in children. In one of the largest studies on pediatric patients, Jagannathan et al. found a net 65% favorable outcomes rate in pediatric patients for accidental trauma after craniectomy when followed for more than five years. Only three patients were dependent on caregivers. This is the only prospective randomised controlled study to date to support the potential benefit of decompressive craniectomy following traumatic brain injury.

Follow-up treatment

After a craniectomy, the risk of brain injury is increased, particularly after the patient heals and becomes mobile again. Therefore, special measures must be taken to protect the brain, such as a helmet or a temporary implant in the skull.

When the patient has healed sufficiently, the opening in the skull is usually closed with a cranioplasty. If possible, the original skull fragment is preserved after the craniectomy in anticipation of the cranioplasty.

Ongoing trials

Two prospective randomised controlled trials are currently being run in an attempt to provide Class I evidence on the role of surgical decompression in the treatment of raised intracranial pressure after severe head injury. The RESCUEicp study is an international multicentre trial, coordinated by the University of Cambridge Academic Neurosurgery Unit and the European Brain Injury Consortium (EBIC) and the DECRA trial is run and coordinated by the Australian centres.

Chapter 3

Lobotomy

Orbitoclast, used in transorbital lobotomy

Lobotomy (Greek: λοβός – *lobos*: "lobe (of brain)"; τομή – *tome*: "cut/slice") is a neurosurgical procedure, a form of psychosurgery, also known as a **leukotomy** or **leucotomy** (from the Greek λευκός – *leukos*: "clear/white" and *tome*). It consists of cutting the connections to and from the prefrontal cortex, the anterior part of the frontal lobes of the brain. While the procedure, initially termed a leukotomy, has been controversial since its inception in 1935, it was a mainstream procedure for more than two decades, prescribed for psychiatric (and occasionally other) conditions—this despite general recognition of frequent and serious side-effects. The Nobel Prize for Physiology or Medicine of 1949 was awarded to António Egas Moniz "for his discovery of the therapeutic value of leucotomy in certain psychoses". The heyday of its usage was from the early 1940s until the mid-1950s when modern neuroleptic (antipsychotic) medications were introduced. By 1951 almost 20,000 lobotomies had been performed in the United States. The decline of the procedure was gradual rather than precipitous. In Ottawa's psychiatric hospitals, for instance, the 153 lobotomies performed in 1953 were reduced to 58 by 1961, after the arrival in Canada of the antipsychotic drug chlorpromazine in 1954.

Context

The lobotomy was one of a series of radical and invasive physical therapies developed in Europe in the first half of the twentieth century. These psychiatric innovations signaled a break with a culture relegating psychiatric patients to asylums, which had prevailed because most serious forms of mental illness were treated only unsatisfactorily by extreme measure, or as unamenable to treatment. These new early twentieth century physical therapies, described as "heroic" in the sense of a desperate last-ditch act to save a life, included malarial therapy for general paresis of the insane (1917), barbiturate induced deep sleep therapy (1920), insulin shock therapy (1933), cardiazol shock therapy (1934), and electroconvulsive therapy (1938).

The development of the leukotomy procedure by Moniz in 1936, took place at a time when all of the above therapeutic interventions were extreme and experimental forms of therapy and most posed serious risks to the health of the patients who underwent them. Leukotomy was seen by many psychiatrists as no more severe than therapies such as insulin or cardiazol shock; these apparently successful procedures conceived for the treatment of patients suffering severe mental illnesses helped to create the intellectual climate and medical and social warrants that allowed a surgical procedure as radical and irreversible as leukotomy to appear as a viable and even necessary proposition. Moreover, Joel Braslow argues that from malarial therapy onward to lobotomy, physical psychiatric therapies "spiral closer and closer to the interior of the brain" with this organ increasingly taking "centre stage as a source of disease and site of cure." For Roy Porter, these often violent and invasive psychiatric interventions are indicative of both the well-intentioned desire of psychiatrists to find some medical means of alleviating the suffering of the thousands of patients in psychiatric hospitals in the twentieth century and also the relative lack of social power of those same patients to resist the increasingly radical and even reckless interventions of asylum doctors.

Pioneers

Gottlieb Burckhardt

In December 1888 Gottlieb Burckhardt, a psychiatrist with little experience of surgery, made one of the first forays into the field of psychosurgery when he operated on six patients, two women and four men aged between 26 and 51, in a private psychiatric hospital in Switzerland. Their diagnoses were, variously, one of chronic mania, one of primary dementia and four of original paranoia (*primäre Verrücktheit*, an obsolete diagnostic category sometimes anachronistically equated with schizophrenia) and, according to Burckhardt's case notes, they exhibited serious psychiatric symptoms such as auditory hallucinations, paranoid delusions, aggression, excitement and violence. He operated on the frontal, temporal, and tempoparietal lobes of these patients. The results were not overly encouraging as one patient died five days after the operation after experiencing epileptic convulsions, one improved but later committed suicide, another two showed no change, and the last two patients became "quieter". This equated to a success rate of 50%. Complications consequent to the procedure included epilepsy (in

two patients), motor weakness, "word deafness" and sensory aphasia. Only two patients are recorded as having no complications.

The theoretical basis of Burckhardt's action rested on three propositions. The first was that mental illness had a physical basis and that disordered minds were merely a reflection of disordered brains. Next, the associationist viewpoint of nerve functioning which conceived the nervous system as operating according to the following threefold division of labor: an input (or sensory or afferent) system, a connecting system which processed information and an output (or efferent or motor) system. The final assumption of Buckhardt's was that the brain was modular which meant that each mental module or mental faculty could be linked to a specific location in the brain. In accordance with such a viewpoint, Buckhardt postulated that lesions in specific areas of the brain might impact behavior in a specific manner. In other words, he thought that by cutting the connecting system, or second association state of brain's system of communication troubling symptoms might be alleviated without compromising either the nervous system's input or output systems. The procedure was aimed at relieving symptoms, not at curing a given mental disease. Thus, he wrote in 1891:

[I]f excitation and impulsive behaviour are due to the fact that from the sensory surfaces excitations abnormal in quality, quantity and intensity do arise, and do act on the motor surfaces, then an improvement could be obtained by creating an obstacle between the two surfaces. The extirpation of the motor or the sensory zone would expose us to the risk of grave functional disturbances and to technical difficulties. It would be more advantageous to practice the excision of a strip of cortex behind and on both sides of the motor zone creating thus a kind of ditch in the temporal lobe.

Burckhardt attended the Berlin Medical Conference of 1889, which was also attended by such heavyweight alienists as Victor Horsley, Valentin Magnan and Emil Kraepelin, and presented a paper on his brain operations. While his findings were subsequently widely reported in the psychiatric literature, the reviews were unremittingly negative and there was much ill ease generated by the surgical procedures he had performed. Kraepelin, writing in 1893, was scathing of Burckhardt's attempts, and stated that "he [Burckhardt] suggested that restless patients could be pacified by scratching away the cerebral cortex." Whilst Giuseppe Seppilli, the Italian professor of neuropsychiatry, remarked in 1891 that Burckhardt's view of the brain as modular did not "fit in well with the view held by most [experts] that the psychoses reflect a diffuse pathology of the cerebral cortex and [ran counter to] the conception of the psyche as a unitary entity".

Burckhardt wrote in 1891 that "Doctors are different by nature. One kind adheres to the old principle: first, do no harm (primum non nocere); the other one says: it is better to do something than do nothing (melius anceps remedium quam nullum). I certainly belong to the second category". The response to this statement was provided by the French alienist Armand Semelaigne when he wrote that "an absence of treatment was better than a bad treatment". After the publication of his impressive 81 page monograph on the subject in 1891, Burckhardt ended his research and practice of psychosurgery no doubt in part due to the ridicule he received from his colleagues over the methods he had employed.

Commenting on his monograph in 1891 the British psychiatrist William Ireland provided a succinct summation of his position:

Dr. Burckhardt has a firm faith in the view that the mind is made up of a number of faculties, holding their seats in distinct portions of the brain. Where excess or irregularity of function occurs he seeks to check it by ablation of a portion of the irritated centres. He defends himself from the criticisms which are sure to be directed against his bold treatment by showing the desperate character of the prognosis of the patients upon whom the operations were performed ...

Ireland, however, doubted that any English psychiatrist would have the "hardihood" to follow the path taken by Burckhardt.

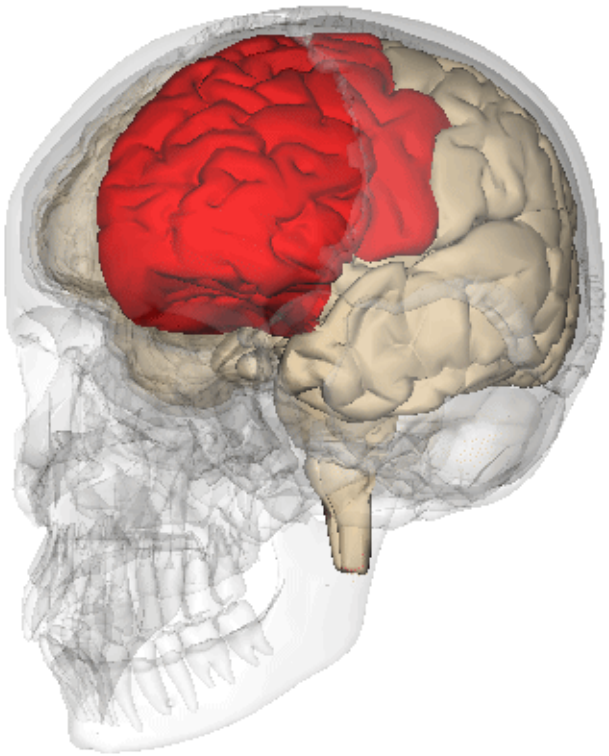
Egas Moniz

The next stage in the development of the procedure was provided by the Portuguese physician and neurologist António Egas Moniz, who was highly acclaimed for his work on cerebral angiography (radiographical visual of the blood vessels in the brain) in 1927. Despite having no clinical psychiatric experience and, indeed, little interest in psychiatry, in 1935 at the Hospital Santa Marta in Lisbon, he devised the surgery called prefrontal leukotomy which was carried out under his direction by the neurosurgeon Pedro Almeida Lima. He was also responsible for coining the term psychosurgery. The procedure involved drilling holes in the patient's head and destroying tissue in the frontal lobes by injecting alcohol. He later changed technique, using a surgical instrument called a leucotome that cut brain tissue by rotating a retractable wire loop (a quite different cutting instrument also used for lobotomies shares the same name). Between November 1935 and February 1936 Moniz and Lima operated on twenty patients, publishing their findings in the same year. Their own assessment was that 35% of the patients improved greatly, 35% improved moderately and that in the remaining 30% there was no change. The patients were aged between 27 and 62 years of age, twelve were female and eight were male. Nine of the patients were diagnosed as suffering from depression, six from schizophrenia, two from panic disorder, and one each from mania, catatonia and manic-depression with the most prominent symptoms being anxiety and agitation. The duration of the illness prior to the procedure varied from as little as four weeks to as much as 22 years, although all but four had been ill for at least one year. The post-operative follow-up assessment took place anywhere from one to ten weeks following surgery. The observed complications were less severe than in Burckhardt's sample as there were no deaths or epileptic convulsions and the most cited complication was fever.

The theoretical underpinnings of Moniz's *avant garde* psychosurgery were largely commensurate with the nineteenth century ones that formed the basis of Burckhardt's theories before him. Although in his later writings he referenced both the neuron theory of Ramón y Cajal and the conditioned reflex of Ivan Pavlov, in essence he simply interpreted this new neurological research in terms of the old psychological theory of associationism. He differed significantly from Burckhardt in that he did not think there

was any physical anatomical pathology in the brains of the mentally ill, but rather that their neural pathways were caught in fixed and destructive circuits As he wrote in 1936:

[The] mental troubles must have [...] a relation with the formation of cellulo-connective groupings, which become more or less fixed. The cellular bodies may remain altogether normal, their cylinders will not have any anatomical alterations; but their multiple liaisons, very variable in normal people, may have arrangements more or less fixed, which will have a relation with persistent ideas and deliria in certain morbid psychic states.



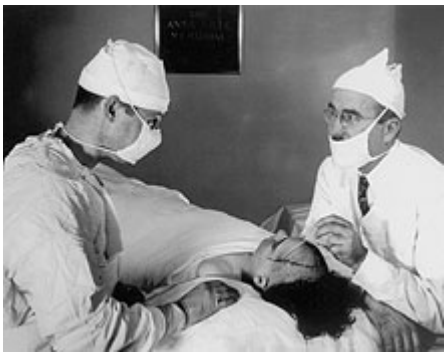
Left frontal lobe highlighted in red. Moniz targeted the frontal lobes in the leucotomy procedure which he first conceived in 1933.

The removal of these aberrant and fixed pathological brain circuits, therefore, might lead to some improvement in mental symptoms. Moniz believed that the brain would functionally adapt to such injury. A significant advantage of this approach was that, unlike the position adopted by Burckhardt, it was unfalsifiable according to the knowledge and technology of the time as the absence of a known correlation between physical brain pathology and mental illness could not disprove his thesis.

Traditionally, the question of why Moniz targeted the frontal lobes in particular has been answered by reference to a presentation by John Fulton and Carlyle Jacobsen at the Second International Congress of Neurology held in London in 1935. Fulton and Carlyle presented two chimpanzees who had undergone frontal lobectomies. The operation had had a pacifying effect on the two primates, who had previously suffered from behavioral disorders. It has been alleged that this provided the impetus and inspiration for Moniz to try the same technique on psychiatric patients. However, as Berrios points out, this conflicts with the fact that Moniz had told his colleague Lima in confidence as early as 1933 of his psychosurgical idea. Nor did he mention Fulton's and Carlyle's presentation as an influence when writing about the procedure in 1936. Indeed, as Kotowicz notes, his attention was drawn more to the case presented by Richard Brickner, at the same conference, of a patient who had had his frontal lobes ablated and, while experiencing a flattening of affect, had suffered no apparent decrease in intellect. Brickner had published on this case in 1932.

Moniz was given the Nobel Prize for medicine in 1949 for this work.

Walter Freeman



James Watts (left) and Walter Freeman performing a lobotomy

The American neurologist and psychiatrist Walter Freeman, who had also attended the London Congress of Neurology in 1935, was intrigued by Moniz's work, and with the help of his close friend, neurosurgeon James W. Watts, he performed the first prefrontal leukotomy in the United States in 1936 at the hospital of George Washington University in Washington. Freeman and Watts gradually refined the surgical technique and created the Freeman-Watts procedure (the "precision method", the standard prefrontal lobotomy).

The Freeman-Watts prefrontal lobotomy still required drilling holes in the scalp, so surgery had to be performed in an operating room by trained neurosurgeons. Walter

Freeman believed this surgery would be unavailable to those he saw as needing it most: patients in state mental hospitals that had no operating rooms, surgeons, or anesthesia and limited budgets. Freeman wanted to simplify the procedure so that it could be carried out by psychiatrists in mental asylums, which housed roughly 600,000 American inpatients at the time.

Inspired by the work of Italian psychiatrist Amarro Fiamberti, Freeman at some point conceived of approaching the frontal lobes through the eye sockets instead of through drilled holes in the skull. In 1945 he took an icepick from his own kitchen and began testing the idea on grapefruit and cadavers. This new "transorbital" lobotomy involved lifting the upper eyelid and placing the point of a thin surgical instrument (often called an orbitoclast or leucotome, although quite different from the wire loop leucotome described above) under the eyelid and against the top of the eyesocket. A mallet was used to drive the orbitoclast through the thin layer of bone and into the brain along the plane of the bridge of the nose, around fifteen degrees toward the interhemispherical fissure. The orbitoclast was mallated five centimetres into the frontal lobes, and then pivoted forty degrees at the orbit perforation so the tip cut toward the opposite side of the head (toward the nose). The instrument was returned to the neutral position and sent a further two centimetres into the brain, before being pivoted around twenty eight degrees each side, to cut outwards and again inwards (In a more radical variation at the end of the last cut described, the butt of the orbitoclast was forced upwards so the tool cut vertically down the side of the cortex of the interhemispherical fissure; the "Deep frontal cut".) All cuts were designed to transect the white fibrous matter connecting the cortical tissue of the prefrontal cortex to the thalamus. The leucotome was then withdrawn and the procedure repeated on the other side.

Freeman performed the first transorbital lobotomy on a live patient in 1946. Its simplicity suggested the possibility of carrying it out in mental hospitals lacking the surgical facilities required for the earlier, more complex procedure (Freeman suggesting that, where conventional anesthesia was unavailable, electroconvulsive therapy be used to render the patient unconscious). In 1947, the Freeman and Watts partnership ended as the latter was disgusted by Freeman's modification of the lobotomy from a surgical operation into a simple "office" procedure. Between 1940 and 1944, 684 lobotomies were performed in the United States. However, because of the fervent promotion of the technique by Freeman and Watts, those numbers increased sharply towards the end of the decade. In 1949, the peak year for lobotomies in the US, 5,074 procedures were undertaken, and by 1951 over 18,608 individuals had been lobotomised in the US.

Prevalence

Most lobotomy procedures were done in the United States, where approximately 40,000 people were lobotomized. In Great Britain, 17,000 lobotomies were performed, and the three Nordic countries of Finland, Norway and Sweden had a combined figure of approximately 9,300 lobotomies. Scandinavian hospitals lobotomized 2.5 times as many people per capita as hospitals in the US. Sweden lobotomized at least 4,500 people between 1944 and 1966, mainly women. This figure includes young children. In Norway

there were 2,500 known lobotomies. In Denmark there were 4,500 known lobotomies, mainly young women, as well as mentally retarded children.

Indications and outcomes: medical literature

According to the *Psychiatric Dictionary* published in 1970:

Prefrontal lobotomy is of value in the following disorders, listed in a descending scale of good results: affective disorders, obsessive-compulsive states, chronic anxiety states and other non-schizophrenic conditions, paranoid schizophrenia, undetermined or mixed type of schizophrenia, catatonic schizophrenia, and hebephrenic and simple schizophrenia. Good results are obtained in about 40 per cent of cases, fair results in some 35 per cent and poor results in 25 per cent are thereabouts. The mortality rate probably does not exceed 3 per cent. Greatest improvement is seen in patients whose premorbid personalities were 'normal', cyclothymic, or obsessive compulsive; in patients with superior intelligence and good education; in psychoses with sudden onset and a clinical picture of affective symptoms of depression or anxiety, and with behaviouristic changes such as refusal of food, overactivity, and delusional ideas of a paranoid nature.

Prefrontal lobotomy has also been used successfully to control pain secondary to organic lesions. In this case, the tendency has been to employ unilateral lobotomy, because of the evidence that a lobotomy extensive enough to reduce psychotic symptoms is not required to control pain.

According to the same source, prefrontal lobotomy reduces:

anxiety feelings and introspective activities; and feelings of inadequacy and self-consciousness are thereby lessened. Lobotomy reduces the emotional tension associated with hallucinations and does away with the catatonic state. Because nearly all psychosurgical procedures have undesirable side effects, they are ordinarily resorted to only after all other methods have failed. The less disorganized the personality of the patient, the more obvious are post-operative side effects. ...

Convulsive seizures are reported as sequelae of prefrontal lobotomy in 5 to 10 percent of all cases. Such seizures are ordinarily well controlled with the usual anti-convulsive drugs. Post-operative blunting of the personality, apathy, and irresponsibility are the rule rather than the exception. Other side effects include distractibility, childishness, facetiousness, lack of tact or discipline, and post-operative incontinence.

Criticism

As early as 1944 an author in the *Journal of Nervous and Mental Disease* remarked: "The history of prefrontal lobotomy has been brief and stormy. Its course has been dotted with both violent opposition and with slavish, unquestioning acceptance." Beginning in 1947 Swedish psychiatrist Snorre Wohlfahrt evaluated early trials, reporting that it is "distinctly hazardous to leucotomize schizophrenics" and lobotomy to be "still too

imperfect to enable us, with its aid, to venture on a general offensive against chronic cases of mental disorder" and stating that "Psychosurgery has as yet failed to discover its precise indications and contraindications and the methods must unfortunately still be regarded as rather crude and hazardous in many respects." In 1948 Norbert Wiener, the author of *Cybernetics*, said: "[P]refrontal lobotomy... has recently been having a certain vogue, probably not unconnected with the fact that it makes the custodial care of many patients easier. Let me remark in passing that killing them makes their custodial care still easier."

Concerns about lobotomy steadily grew. The USSR officially banned the procedure in 1950. Doctors in the Soviet Union concluded that the procedure was "contrary to the principles of humanity" and that it turned "an insane person into an idiot." By the 1970s, numerous countries had banned the procedure as had several US states. Other forms of psychosurgery continued to be legally practiced in controlled and regulated US centers and in Finland, Sweden, the UK, Spain, India, Belgium and the Netherlands.

In 1977 the US Congress created the National Committee for the Protection of Human Subjects of Biomedical and Behavioral Research to investigate allegations that psychosurgery—including lobotomy techniques—were used to control minorities and restrain individual rights. It also investigated the after-effects of surgery. The committee concluded that some extremely limited and properly performed psychosurgery could have positive effects.

By the early 1970s the practice of lobotomy had generally ceased, but some countries continued to use other forms of psychosurgery. In 2001 there were, for example, 70 operations in Belgium, about 15 in the UK and about 15 a year at Massachusetts General Hospital in Boston, while France had carried out operations on about 5 patients a year in the early 1980s.

Notable cases

- Rosemary Kennedy, sister of President John F. Kennedy, underwent a lobotomy at age 23 which left her permanently incapacitated.
- Howard Dully wrote a memoir of his late-life discovery that he had been lobotomized at age 12.
- New Zealand author and poet Janet Frame received a literary award the day before a scheduled lobotomy was to take place, and it was never performed.
- French Canadian singer Alys Robi underwent a lobotomy and later resumed singing professionally.
- Swedish modernist painter Sigrid Hjertén died following a lobotomy in 1948.
- Playwright Tennessee Williams's older sister Rose received a lobotomy which left her incapacitated for life; the episode is said to have inspired characters and motifs in certain of his works.

It is often said that when an iron rod was accidentally driven through the head of Phineas Gage in 1848, this constituted an "accidental lobotomy", or that this event somehow

inspired the development of surgical lobotomy a century later. According to the only book-length study of Gage, careful inquiry turns up no such link.

Literary and cinematic portrayals

Lobotomies have been featured in several literary and cinematic presentations that both reflected society's attitude towards the procedure and, at times, changed it. The 1946 novel *All the King's Men* by Robert Penn Warren described a lobotomy, saying it "would have made a Comanche brave look like a tyro [novice] with a scalping knife." The surgeon is portrayed as a repressed man who couldn't change others with love but instead resorted to "high-grade carpentry work." In Tennessee Williams's 1958 play, *Suddenly, Last Summer*, the protagonist is threatened with a lobotomy to stop her from telling the truth about her cousin Sebastian. The surgeon says, "I can't guarantee that a lobotomy would stop her *babbling*." Her aunt responds, "That may be, maybe not, but after the operation who would *believe* her, Doctor?"

A damning portrayal of the procedure is found in Ken Kesey's 1962 novel *One Flew Over the Cuckoo's Nest* and its 1975 movie adaptation. Several patients in the mental ward receive lobotomies in order to discipline or calm them. The operation is described as brutal and abusive, a "frontal-lobe castration". The book's narrator, Chief Bromden, is shocked: "There's nothin' in the face. Just like one of those store dummies." One patient's surgery changes him from an acute to a chronic mental condition. "You can see by his eyes how they burned him out over there; his eyes are all smoked up and gray and deserted inside."

Other sources include Sylvia Plath's 1963 novel *The Bell Jar*, in which the protagonist, Esther, reacts with horror to the "perpetual marble calm" of a lobotomized young woman named Valerie. Elliott Baker's 1964 novel and 1966 film version, *A Fine Madness,* portrays the dehumanizing lobotomy of a womanizing, quarrelsome poet who in the end is just as aggressive as ever. The surgeon is depicted as an inhumane crackpot. The 1982 biopic *Frances* includes a disturbing scene showing actress Frances Farmer undergoing transorbital lobotomy. The claim that a lobotomy was performed on Farmer (and that Freeman performed it) has been criticized as having little or no evidence supporting it.

In the 2010 Martin Scorsese psychological mystery-thriller film *Shutter Island* based on the novel of the same name set in the 1950s when lobotomy was considered an appropriate procedure by many in the psychiatric community, the main character, found to be criminally insane, is given the choice of facing up to the reality that he murdered his wife or be lobotomized. In the novel, it is clear he receives a lobotomy involuntarily after relapsing into insanity whereas the movie is ambiguous as to whether he faked his relapse in order to "die as a good man" by being lobotomized rather than "live as a monster" without the treatment. Dr. James Gilligan, a past director of Massachusetts' prison mental hospital and serving as a technical advisor stated:

“ We worked together to make sure the story reflected a true war that was going on in the mid-20th century within the psychiatric community: a war between those clinicians who wanted to treat these patients with new forms of psychotherapy, education and medicine, and those who regarded the violent mentally ill as incurable and advocated controlling their behavior by inflicting irreversible brain damage, including indiscriminate use of shock treatment and crude forms of brain surgery, such as lobotomies.

Chapter 4

Hemispherectomy and Bilateral Cingulotomy

Hemispherectomy

Intervention:
Hemispherectomy

ICD-10 code:
ICD-9 code: 01.52
Other codes:

Hemispherectomy is a surgical procedure where one cerebral hemisphere (half of the brain) is removed or disabled. This procedure is used to treat a variety of seizure disorders where the source of the epilepsy is localized to a broad area of a single hemisphere of the brain. It is solely reserved for extreme cases in which the seizures have not responded to medications and other less invasive surgeries.

History and changes

Hemispherectomy was first tried on a dog in 1888 by Friedrich Goltz. The first such operation on humans was done by Walter Dandy in 1923 for glioblastoma multiforme. In the 1960s and early 1970s, hemispherectomy involved removing half of the brain, but this resulted in unacceptable complications and side effects in many cases, predominantly filling of excessive body fluids in the skull with subsequent pressure to the remaining brain (known as hydrocephalus). The procedure was revitalized in children in the 1980s by Dr. Ben Carson at The Johns Hopkins Hospital. In many centers, the *functional hemispherectomy* has largely replaced this procedure, in which the temporal lobe is removed; a procedure known as corpus callosotomy is performed; and the frontal and occipital lobes are disconnected from the rest of the brain; however the traditional "anatomic" hemispherectomy has remained a viable procedure, due to its superiority in preventing future seizures compared with functional hemispherectomy.

Results

All hemispherectomy patients suffer at least partial hemiplegia on the side of the body opposite the removed or disabled portion, and may suffer problems with their vision as well.

This procedure is almost exclusively performed in children because their brains generally display more neuroplasticity, allowing neurons from the remaining hemisphere to take over the tasks from the lost hemisphere. This likely occurs by strengthening neural connections which already exist on the unaffected side but which would have otherwise remained small in a normally functioning, uninjured brain. One case, demonstrated by Smith & Sugar, 1975; A. Smith 1987, demonstrated that one patient with this procedure had completed college, attended graduate school and scored above average on intelligence tests. Studies have found no significant long-term effects on memory, personality, or humor after the procedure, and minimal changes in cognitive function overall. Generally, the greater the intellectual capacity of the patient prior to surgery, the greater the decline in function. Most patients end up with mild to severe mental retardation, which is usually already present before surgery. When resectioning the left hemisphere, evidence indicates that some advanced language functions (*e.g.,* higher order grammar) cannot be entirely assumed by the right side. The extent of advanced language loss is often dependent on the patient's age at the time of surgery.

Although initially thought to be limited solely to children, a recent study in 2007 by Dr. Shearwood McClelland III and Dr. Robert E. Maxwell indicated the long-term efficacy of anatomic hemispherectomy in carefully selected adults, with seizure control sustainable over multiple decades.

Foundations

The Hemispherectomy Foundation was formed in 2008 to assist families with children undergoing this procedure.

Bilateral cingulotomy

Intervention:
Bilateral cingulotomy

ICD-10 code:
ICD-9 code: 0.2
Other codes:

Bilateral cingulotomy is a form of psychosurgery, introduced in 1948 as an alternative to lobotomy. Today, it is mainly used in the treatment of obsessive-compulsive disorder, depression and addiction. It is also, rarely, used in the treatment of chronic pain. The objective of this surgical procedure is the severing of the supracallosal fibres of the cingulum bundle, which pass through the anterior cingulate gyrus.

Target

Bilateral Cingulotomy targets the anterior cingulate cortex, which is a part of the limbic system. This system is responsible for the integration of feelings and emotion in the human cortex. It consists of the cingulate gyrus, parahippocampal gyrus and the hippocampal formation.

Studies in patients that were a subject to bilateral cingulotomy, that involved fMRI analyses, showed that the anterior cingulate cortex has a key role in cognitive control and is highly likely to be involved in the control of attentional response, whereas the dorsal part of that region of the brain was not identified to be involved in such a process, although this is still under dispute. The function of the dorsal part of the cingulate cortex was connected to the sorting out and processing of conflicting information signals. In addition, neuroimaging studies also indicated that the anterior cingulate cortex participates in the modulation of cortical regions that are of higher order as well as sensory processing areas.

These findings have also been confirmed by stereotactic microelectrode analysis of single cortical neurons in a study, which involved nine patients undergoing bilateral cingulotomy. The study investigated the effect of performing attention demanding tasks on the activity of 36 neurons located in the anterior cingulate cortex. Upon analyzing of the results of the study it was concluded that the anterior cingulate cortex is indeed involved in the modification of cognitive tasks that require attention based on the fact that there was a change in the basal firing rate of neurons in that region during simulation of such tasks.

Neuroimaging also uncovered different sub-regions in the anterior cingulate cortex itself based on their function. It was proven that the caudal part of the anterior cingulate cortex plays a more important function in cognitive activities that involve attention, salience, interference and response competition. These results combined with electrophysiological investigation of the function of neurons in the anterior cingulate cortex have provided insights that can be used in the improvement of cingulotomy performed on patients treated for OCD. The basis behind this idea is the fact that a variation of certain tasks, Emotional Stroop tasks (ES), which have been particularly identified as exerting effects in OCD patients activate neurons in the more rostral part of the anterior cingulate cortex. Thus, theoretically if bilateral cingulotomy is performed in such patient in the rostral anterior cingulate cortex, better results should be obtained.

Moreover, OCD has been associated with a malformation of the basal ganglia . The function of this part of the human brain has been mapped to be composed of fiber tracks

associated with numerous parallel cortico-striato-thalamocortical circuits (CSTC), which are involved in sensorimotor, motor, oculomotor as well as the cognitive processes that are manifested by the limbic system. This pathway involves GABAergic inhibitory projections that serve as one of the means of communication between the different structures involved. It has been hypothesized that some forms of OCD are a result of disinhibition of a one or several of the circuits that operate in the CSTC. This is also indicated by a finding that showed a significant decrease in intracortical inhibition in OCD patients. Thus, lesions in the anterior cingulate cortex might contribute to the lessening of the disinhibition effect. This theory has been confirmed by another study which assessed the cortical inhibitory and excitatory mechanisms in OCD. The study measured the excitability of motor cortex, as well as intracortical inhibition in OCD patients and a control of healthy individuals. The results showed a significant decrease in intracortical inhibition, which resulted in a slowdown of interstimulus intervals by 3msec. In addition to its proximity to and association with the limbic system and the amygdala in particular, which plays a key role in emotional experience, the anterior cingulate cortex shares afferent and efferent pathways with a number of thalamic nuclei as well as the posterior cingulate and part of some parietal, frontal and supplementary motor cortex. All these underline the high likelihood that the anterior cingulate cortex must be linked to OCD.

Functional MRI analyses of the anterior cingulate cortex have also led to the introduction of bilateral cingulotomy for the treatment of chronic pain. Such application was introduced since the anterior cingulate cortex has been found to be related to the processing nociceptive information input. In particular the role of the anterior cingulate cortex is in the interpretation of how a stimulus affects a person rather than its actual physical intensity.

Procedure

In most cases the procedure starts with the medical team taking a number of CT scan X-ray images of the brain of the patient. This step ensures that the exact target, the cingulate cortex is mapped out, so that the surgeon can identify it. Then burr holes are created into the patient's skull using a drill. Lesions at the targeted tissue are made with the help of fine electrodes inserted very carefully at the right angle into the subject's brain based on plotting charts and making sure important arteries and blood vessels are intact. The electrode is placed in a probe , or a holder, with only its tip projecting. Upon the correct insertion of the holder into the brain tissue, air is injected and more scan images are taken. Then, after the medical team has made sure they are on the right track, the tip of the electrode is advanced to the plane of the cingulate where it is heated to 75-90 C. Once the first lesion is created it serves as a center around which several other lesions are created. In order to confirm whether lesions are made at the right place, scan images are taken postoperatively and analyzed.

Recent technological advances, however, have made bilateral cingulotomy a more precise operation. For example, nowadays a neurosurgical team that has to perform the procedure can use an MRI to identify the location of the anterior and posterior commissures. This

approach allows neurosurgeons to obtain a number of coronal images, which are then used to calculate the stereotactic coordinates of the place in the anterior cingulate cortex, where lesions need to be created. Moreover the MRI enables to differentiate more precisely the cell composition, and thus easily identify the gray matter in that region. This can then be further confirmed with the help of microelectrode recordings.

Side Effects

Patients usually recover from this operation over a period of 4 days. However, there are cases of subjects released from hospital after as few as 48 hours after the operation. The mild shorter postoperative complications that are most commonly related to bilateral cingulotomy are typical of head interventions and include but are not limited to nausea, vomiting, and headaches. However, in some cases patients exhibit seizures that sometimes appear up to two months after the surgical intervention. There is the discussion as to whether this is relevant and can be account to cingulotomy because such seizures were observed in patients that already had a history of this condition.

Case Studies

A recent study conducted at the Massachusetts General Hospital analyzed the outcome of bilateral cingulotomy in 44 patients for the treatment of OCD in the period between 1965 and 1986. Patients were followed up over a long term and evaluated based on several criteria: 1) how many of them were responders after a period of 6 months, 2) how many cingulotomies had a patient undergone before the examination of the effectiveness of the procedure, 3) did the patient show any significant change after the most recent procedure and 4) what were the side effects related to the procedure.

The follow up of the patients produced contradictory results, which indicated that bilateral cingulotomy is not the optimal treatment for OCD as of today. From the 44 patients only 32% could be classified as responders and showed significant improvement compared to the other subjects. Another 14% exhibited some signs of improvement. Multiple cingultomies lead to the increase in responders by 6% and to partial responders by 11%. However, the side effects associated with the procedure were numerous. Among the complaints that patients had after the surgery were deficits in memory and apathy although these were rarely observed. In addition, some of the subjects complained from some form of urinary disturbance ranging from urinary retention to incontinence. Hydrocephalus (2%) and seizures (2%) were also observed .

Another clinical study investigated the effect of bilateral cingulotomy for the treatment of refractory chronic pain. In this case 23 patients that were subject to 28 cingulotomies in total were followed up. The analyses aimed at determining how much the pain of each individual was affected after the procedure with the help of a questionnaire. In addition, the examiners tried to evaluate the social and family relations of the participants in the study. Based on the data obtained, cingulatomy for treatment of chronic pain showed promising results. 72% reported improvement in the level of pain experienced, and 50 % indicated that they no longer required painkillers after cingulotomy. More than half of the

patients also claimed that the surgical procedure was beneficial and contributed to the improvement of their social aspects.

Chapter 5

Hypophysectomy, Amygdalohippocampectomy and Intervertebral Disc Arthroplasty

Hypophysectomy

	Intervention: Hypophysectomy
ICD-10 code:	
ICD-9 code:	07.6
MeSH	D007016
Other codes:	

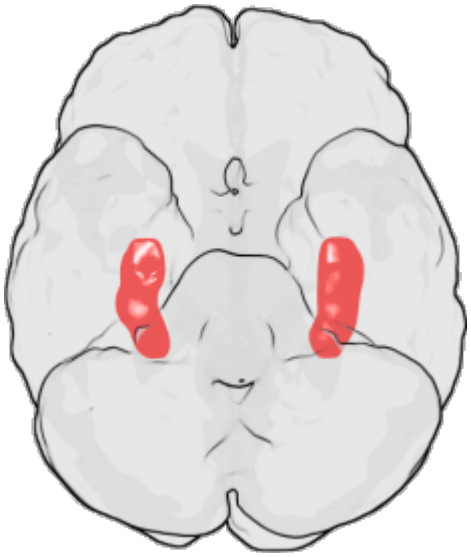
Hypophysectomy is the surgical removal of the hypophysis (pituitary gland). It is most commonly performed to treat tumors, especially craniopharyngioma tumors. Sometimes it is used to treat Cushing's syndrome due to pituitary adenoma. It is also applied in neurosciences (in experiments with lab animals) to understand the functioning of hypophysis.

Medications that are given as hormone replacement therapy following a complete hypophysectomy (removal of the pituitary gland) are Glucocorticoids and Thyroid Medications.

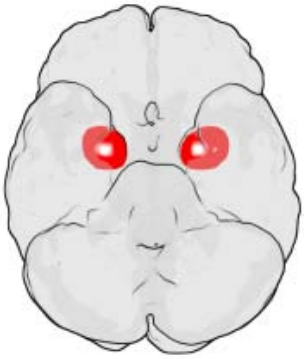
Complications

Hypophysectomy performed at any age causes atrophy of the thyroid and adrenal glands as well as asthenia and cachexia. When the procedure is performed before sexual maturity, the reproductive tract remains undeveloped and non-functional. There is also a general lack of growth. If performed after sexual maturity, there is loss of reproductive function along with atrophy of gonads and accessory reproductive structures.

Amygdalohippocampectomy



Hippocampus



Amygdalae

Amygdalohippocampectomy is a surgical procedure for the treatment of epilepsy. It consists of the removal of the hippocampus, which has a role in memory, spatial awareness, and navigation, and the amygdalae, which have a role in the processing and memory of emotional reactions, both structures forming part of the limbic system of the brain.

Amygdalohippocampectomy is used only when all other treatment options have failed to resolve the epilepsy. It is an effective treatment for most patients. However, possible adverse side effects include impaired memory and defects in visual perception.

Procedure

The amygalohippocampectomy is indicated when the focal point of the seizures can be anatomically localized to the hippocampus and amygdala. Normally, to be considered for this procedure, one must have failed all first-line treatments. The selective amygdalohippocampectomy will remove only the offending portions of the hippocampus and amygdala. When data from studies of the electrophysiology and neuropathy *vis-à-vis* temporal lobe epilepsy determines this area to be the origin of seizure activity, the removal of the hippocampus and amygdala is usually indicated. Computer imaging is sometimes used to guide this procedure. Patients continue normal activity after approximately six to eight weeks.

Statistics and side effects

Of 376 patients who had the amygdalohippocampectomy procedure performed, compared to other types of temporal lobe resections, two thirds of this population were reported free of disabling seizures. Some patients report defects in visual perception and impaired memory function.

Intervertebral disc arthroplasty

Artificial Disc Replacement (ADR), or **Total Disc Replacement (TDR)**, is a type of arthroplasty. It is a surgical procedure in which degenerated intervertebral discs in the spinal column are replaced with artificial devices in the lumbar (lower) or cervical (upper) spine. The procedure is used to treat chronic, severe low back pain and cervical pain resulting from degenerative disc disease.

Artificial disc replacement has been developed as an alternative to spinal fusion, with the goal of pain reduction or elimination, while still allowing motion throughout the spine. Another possible benefit is the prevention of premature breakdown in adjacent levels of the spine, a potential risk in fusion surgeries.

Regulation

United States

Two artificial discs have been approved by the FDA for use in the US: the Charite, manufactured by DePuy for use in the lumbar spine; and the ProDisc, manufactured by Synthes for use in the lumbar spine and cervical spine. They are FDA approved for one-level applications, after clinical trials were said to show patient improvement in motion and pain equivalent to spinal fusion. Two-level disc replacement surgery is considered experimental in the United States, but has been performed in Europe for many years.

While these two discs have received FDA approval, some insurance companies in the United States do not cover the surgery, still classifying it as experimental. Effective

August 14, 2007, the Centers for Medicare & Medicaid Services (CMS) will not cover Lumbar Artificial Disc Replacement (LADR) for patients over the age of 60, on a national basis. Individual localities regulate the use of the procedure in patients 60 and under.

The Maverick, manufactured by Medtronic, has been prevented from entering the US market due to patent infringement litigation, ongoing as of September, 2010.

History

Artificial disc surgery is still relatively new in the United States, but has been used in Europe for more than 15 years.

The first device approved for use in the United States was the Charite artificial disc. Invented at Charite University Hospital in Berlin in the mid-1980s by the East German scientist, two-time Olympic champion in women's artistic gymnastics Karin Büttner-Janz and Kurt Schellnack, the disc received FDA approval in the United States in October 2004, following a four-year clinical trial.

The first surgeon to perform a Charite artificial disc surgery in the United States was Scott Blumenthal, M.D., a spine surgeon at Texas Back Institute in Plano, Texas. Blumenthal served as principal investigator for the Charite study in the US.

Dr. Rudolf Bertagnoli helped to develop Pro Disc and its surgical technique in Europe and has taught more than 2,500 surgeons how to perform the procedure.

Controversy

The New York Times, January 2008, raised concerns relating to the transparency of research being carried out by investors in Prodisc. Questions have been raised about the accuracy of that article.

A statement issued by The American Association of Orthopaedic Surgeons (AAOS) recommends caution in using the new devices, as the studies behind their approval were not designed to show their superiority, only that they produced results equivalent to existing treatments. The data shows that artificial disc replacement patients, when compared to spinal fusion patients, have a shorter recuperation period following surgery, but research also shows that spinal fusion patients show no better outcomes than patients undergoing physical therapy.

The AAOS also states that disc replacement requires a high level of technical skill for accurate placement, and has a significant level of risk if revision surgery is needed.

Members of AAOS and the American Association of Neurological Surgeons joined together as the Association for Ethics in Spine Surgery, formed to raise awareness of the ties between physicians and device manufactureres.

There are several class-action lawsuits pending against the Charite Artificial Disc, and reports of complications with the Pro Disc Artificial Disc implant when used in certain surgical situations.

Chapter 6

Rhizotomy

	Intervention: Rhizotomy
ICD-10 code:	
ICD-9 code:	03.1
MeSH	D019051
Other codes:	

A **rhizotomy** is a neurosurgical procedure that selectively severs problematic nerve roots in the spinal cord, most often to relieve the symptoms of neuromuscular conditions such as spastic diplegia and other forms of spastic cerebral palsy. In extreme cases, a rhizotomy may also be considered for a person suffering from severe back pain or a pinched nerve. The **selective dorsal rhizotomy** (SDR) for spastic cerebral palsy is the main use of rhizotomy for today's neurosurgeons. In this surgery, the spasticity-causing nerves are isolated and then targeted and destroyed. The sensory nerve roots, where spasticity is located, are first separated from the motor ones, and the nerve fibres to be cut are then identified via electromyographic stimulation. The one(s) producing spasticity are then selectively lesioned with tiny electrical pulses.

In spasticity, rhizotomy targets and destroys the damaged nerves that don’t receive gamma amino butyric acid, which is the core problem for people with spastic cerebral palsy. In this case, those nerves which, due to not receiving GABA, generate unusual electrical activity during the testing phase are considered to be the source of hypertonia, and are cut, while the remaining nerves and nerve routes carrying the correct messages remain fully intact.

Background

Dorsal rhizotomy or *selective dorsal rhizotomy* (SDR) is the most widely-used form of rhizotomy and is today a primary treatment for spastic diplegia, best done in the youngest years before bone/joint deformities from the pull of spasticity take place, but it can be performed safely and effectively on adults as well.

SDR is a permanent procedure that addresses the spasticity at its neuromuscular root: i.e., in the central nervous system that contains the misfiring nerves that cause the spasticity of those certain muscles in the first place. After a rhizotomy, assuming no complications, the person's spasticity is usually completely eliminated, revealing the "real" strength (or lack thereof) of the muscles underneath.

Because the muscles may have been depending on the spasticity to function, there is almost always extreme weakness after a rhizotomy, and the patient will have to work very hard to strengthen the weak muscles with intensive physical therapy, and to learn habits of movement and daily tasks in a body without the spasticity. Rhizotomy's result is fundamentally unlike orthopedic surgical procedures, where any release in spasticity is essentially temporary.

Rhizotomy is usually performed on the pediatric spastic cerebral palsy population between the ages of 2 and 6, since this is the age range where orthopedic deformities from spasticity have not yet occurred, or are minimal. However, recent cases of successful SDR procedures among those with spastic diplegia across all major age ranges (years 3-40) has finally proven its universal effectiveness and safety regardless of the age of the spastic diplegic patient.

Although the concept of rhizotomy was conceived, tested and tried as early as, or possibly even earlier than, the 1930s, it was not in wide use for the treatment of spasticity until the last quarter of the 20th Century. Dr. Warwick Peacock of South Africa helped to begin the modern era of rhizotomy procedure in the early 1980s, and soon trained many other neurosurgeons in his technique; both Peacock and these other surgeons then went on to develop the procedure further using both their own intellectual refinements and refinements in medical equipment and technology that occurred from the 1980s through the 2000s.

Today, St. Louis Children's Hospital in St. Louis, Missouri has a "Center for Cerebral Palsy Spasticity" that is the only internationally-known clinic in the world to have conducted concentrated first-hand clinical research on SDR over an extended period. Its chief neurosurgeon in the field, Doctor T.S. Park (who was initially trained by Dr. Peacock), has performed thousands of SDR surgeries, some of them on adults, and is the originator of the L1-laminectomy modification to the SDR surgery in 1991, which sections the first dorsal root and enables the removal of significantly less spine-bone than in surgeries performed before 1991, as well as inherent release of the hip flexor muscles specifically as a result of that particular sectioning (prior to that, total hip flexor release was not necessarily possible). That L1-laminectomy modification has since become the standard, and SLCH has become internationally known as a major provider of the SDR surgery to those in need of it (for example, it is one of the first Google search results when inputting the word string "selective dorsal rhizotomy"). It is this clinic's opinion that patients with spastic diplegia or quadriplegia should have spasticity reduced first through SDR before undergoing muscle release or tendon release procedures, and other surgeons today share this view. A major qualifier in the cases taken on at SLCH, however, is that all of its adults have had only *mild* cases of spastic diplegia.

In September 2008, a ground-breaking SDR was performed that 'closed the gap' on concerns regarding age of the patient in SDR: Columbia-Presbyterian Children's Hospital's Richard C.E. Anderson performed an SDR surgery on a 28-year-old male with *moderate* spastic diplegia, which by the patient's own report has reduced his muscle tone nearly to the level of a "normal" person and enabled him to walk and exercise much more efficiently; also, Dr. Anderson in the past performed an SDR on a 16-year-old wheelchair-using female with *severe* spastic diplegia. Reportedly, that particular SDR enabled the young woman to ambulate, whereas before the surgery, she was too tight to do so.

Procedural outline

SDR begins with a 1- to 2-inch incision along the center of the lower back just above the waist. An L1 laminectomy is then performed: a section of the spine's bone, the spinous processes together with a portion of the lamina, are removed, like a drain-cap, to expose the spinal cord and spinal nerves underneath. Ultrasound and an x-ray locate the tip of the spinal cord, where there is a natural separation between sensory and motor nerves. A rubber pad is then placed to separate the motor from the sensory nerves. The sensory nerve roots, each of which will be tested and selectively eliminated, are placed on top of the pad, while the motor nerves are beneath the pad, away from the operative field.

After the sensory nerves are exposed, each sensory nerve root is divided into 3-5 rootlets. Each rootlet is tested with electromyography, which records electrical patterns in muscles. Rootlets are ranked from 1 (mild) to 4 (severe) for spasticity. The severely abnormal rootlets are cut. This technique is repeated for rootlets between spinal nerves L2 and S2. Half of the L1 dorsal root fibers are cut without EMG testing.

When testing and corresponding elimination are complete, the dura mater is closed, and fentanyl is given to bathe the sensory nerves directly. The other layers of tissue, muscle, fascia, and subcutaneous tissue are sewn. The skin is typically now closed with glue, but there are sometimes stitches to be removed from the back after 3 weeks. The surgery takes approximately 4 hours and typically involves one neurosurgeon, one anesthesiologist, and possibly an assortment of assisting physicians (as in the New York City September 2008 case). The patient then goes to the recovery room for 1–2 hours before being transferred to the intensive care unit overnight. Transfer from the ICU to a recovery room in the hospital is then done to enable direct post-surgical observation by the neurosurgeon and surgical team, but this usually lasts only about 3 days, during which the team performs range-of-motion tests that they record and compare to pre-surgery levels. After that short period, the patient, depending on circumstances and appropriateness, is either transferred to inpatient recovery or is linked to an intense outpatient exercise program and discharged from the hospital.

According to clinicians, it usually takes about one year from the date of surgery to achieve maximum results from SDR. However, videos from St. Louis Children's Hospital website have shown continued marked improvement as much as 5 years post-surgery, and presumably, if the person keeps exercising intensely, potential for continued

improvement and strengthening is, just as in a person born with normal muscle tone and range of motion, unlimited.

Complications

There is always abnormal sensitivity and tingling of the skin on the feet and legs after SDR because of the nature of the nerves that have been worked on, but this usually resolves within 6 weeks. There is no way to prevent the abnormal sensitivity in the feet. Transient change in bladder control may occur, but this also resolves within a few weeks.

If a certain degree of permanent numbness remains in certain leg-muscles, such as the quadriceps, ankles, and feet, this is usually not enough to prevent feeling and sensation, sensing of changes in temperature or pressure, etc. The affected muscle-areas simply feel less than before, and the trade-off in ease of movement is said to be immensely worth this change, should it occur.

In general, there is a *combined 5-10%* risk of any of the following more serious risks happening as a result of SDR. Because of technological advances in both the technology used in the surgery and also in the procedure itself, there have been no major cases of SDR that have had these side-effects.

- Permanent paralysis of the legs and bladder.
- Permanent impotence
- Sensory loss and/or numbness that is severe enough to not feel anything any more in the legs (not paralysis; movement is retained)
- Wound infection and meningitis - usually controlled with antibiotics
- Leakage of the spinal fluid through the wound, also repairable; the surgical team watches very closely post-surgery for this

A few patients in St. Louis experienced urinary tract infections and pneumonia, but these were successfully treated.

Those who walk independently before SDR

After the surgery all patients who were walking independently before surgery regained the independent walking within a few weeks after surgery. Patients maintain independent walking for the long term; when some have more difficulty walking independently they may eventually need an assistive device—however, in nearly all cases spasticity can be eliminated and the quality of independent walking improves; in many patients, physical therapy and braces become unnecessary after SDR. Orthopedic surgery is rarely required after SDR.

Those who walk with walkers or crutches before SDR

In children who are 2–7 years old and walk with a walker or crutches before SDR, independent walking after the procedure is possible. Once they have achieved independent walking, they can maintain it.

In children who are older than 7 years and walks with crutches, independent walking (inside or outside house) is possible. If they walk with walker at the age, they will most likely walk with a walker or crutches after the procedure, though it improves the quality of assisted walking and transition movements, and alleviates deformities of the legs. Many of these patients will need orthopedic surgeries after SDR.

Candidates for rhizotomy

Not all patients with spastic cerebral palsy benefit from SDR. For those under 18 years of age, rhizotomy requires that they be:

- At least 2 years of age
- Diagnosis of spastic diplegia, spastic quadriplegia or spastic hemiplegia
- Some form of independent mobility; for example, crawling or walking with or without an assistive device
- History of premature birth; if born at full term, child must have typical signs of spastic diplegia
- No severe damage to the basal ganglia on MRI examination
- Potential for improvement in functional skills

For adults between 19 and 40 years of age, rhizotomy requires:

- Diagnosis of spastic diplegia
- History of premature birth
- Currently ambulates independently *without* assistive device
- No fixed orthopedic deformities that either prevent current walking or would prevent walking after SDR; in these cases orthopedic releases are to be done first, after which SDR can be discussed.
- Potential for functional gains after SDR
- Intense motivation to attend intensive physical therapy and perform home exercise program

On the limited number of adult spastic diplegic people treated with rhizotomy, satisfactory functional gains in adult patients are similar to those in children.

Required circumstances

All candidates for rhizotomy must have good muscle strength in the legs and trunk. There must also be evidence of adequate motor control, or the ability to make reciprocal movements for crawling or walking, and to move reasonably quickly from one posture to

another. Chiefly, pediatric rhizotomy candidates are people with CP who have shown age-appropriate progression in motor development, but spasticity hampers the development of skills and/or causes gait patterns like the scissors gait. In adults, the primary requirements are that the person is able to ambulate independently, but spasticity limits energy, flexibility, walking speed and balance and sometimes causes pain/muscle spasms.

Conditions that preclude SDR

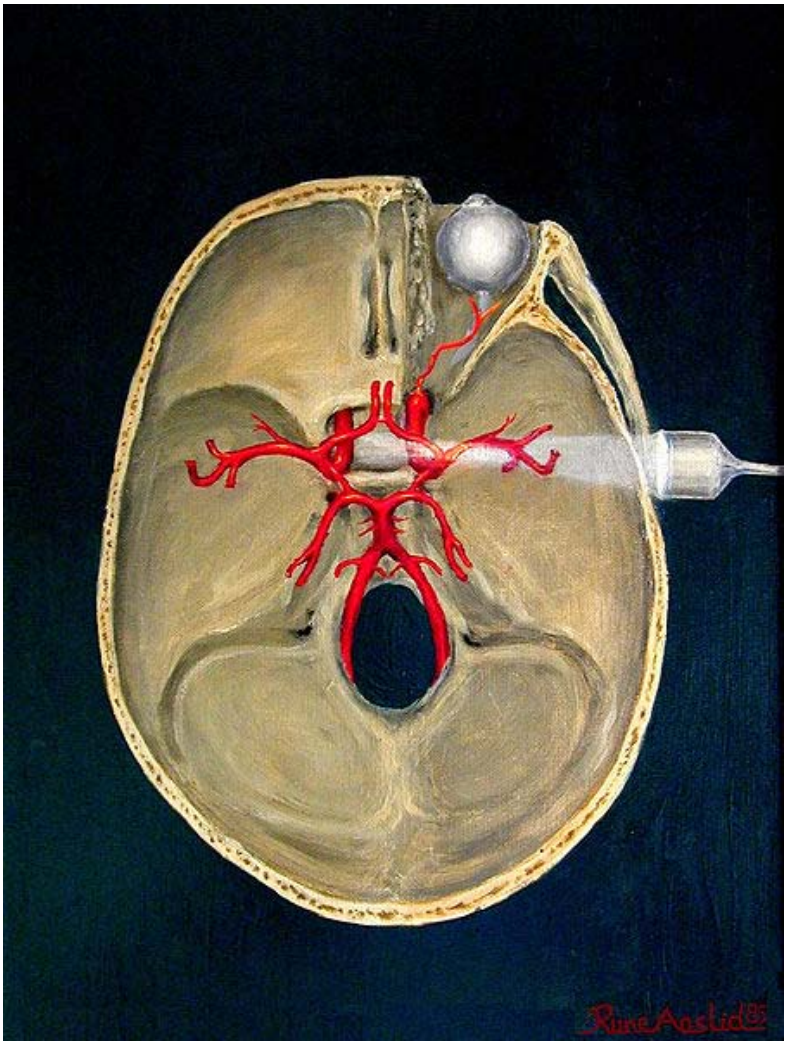
There are a few clinical situations in which it is likely that someone may not be a candidates for the surgery. These situations include those who have suffered meningitis, a congenital (birth-originating) brain infection, congenital hydrocephalus unrelated to the person's premature birth, a person who has suffered head trauma, or a person with some sort of familial disease (e.g., those with hereditary spastic paraplegia are said to not be SDR candidates). Also precluded are people who have a "mixed" CP with predominant rigidity or dystonia, significant athetosis, or ataxia; and those who have very severe scoliosis. However, as with any procedure, an individual evaluation is needed in all instances to determine eligibility.

Post-surgical restrengthening

Most rehabilitation from SDR is done on an outpatient basis, though it may also include an initial several-week inpatient component (but typically does not). Typical base restrengthening and restoration of full ambulatory function takes about twelve weeks (3 months) of intensive physical therapy 4-5 times per week, but subsequent buildup and maintenance beyond that initial several-week period is just as necessary, and may require continued 4-5 times per week therapy as much as 6 months postoperatively, for a total of about a year and four months after surgery in order to achieve *maximum basic* functionary movement from the surgery. Beyond that point, any continued strengthening is, as with any person's exercise regimen, undertaken strictly by the individual's own choice and direction.

Chapter 7

Transcranial Doppler



Transcranial doppler insonation of the cerebral circulation

Transcranial doppler ultrasound analyzer of blood velocity

Transcranial Doppler (TCD) is a test that measures the velocity of blood flow through the brain's blood vessels. Used to help in the diagnosis of emboli, stenosis, vasospasm from a subarachnoid hemorrhage (bleeding from a ruptured aneurysm), and other problems, this relatively quick and inexpensive test is growing in popularity in the United States. The equipment used for these tests is becoming increasingly portable, making it possible for a clinician to travel to a hospital, doctor's office or nursing home for both inpatient and outpatient studies. It is often used in conjunction with other tests such as MRI, MRA, carotid duplex ultrasound and CT scans.

Methods

Two methods of recording may be used for this procedure. The first uses "B-mode" imaging, which displays a 2-dimensional image as seen by the ultrasound probe. Once the desired blood vessel is found, blood flow velocities may be measured with a pulsed doppler probe, which graphs velocities over time. Together, these make a duplex test. The second method of recording uses only the second probe function, relying instead on the training and experience of the clinician in finding the correct vessels.

Applications of TCD

Clinical routine transcranial Doppler (TCD) ultrasound examination of the intracranial arteries was demonstrated to be possible in 1982 by Aaslid and colleagues. The value obtained for a particular artery is the velocity of blood flowing through the vessel, and unless the diameter of that vessel is established by some other means it is not possible to determine the actual blood flow. Thus TCD is primarily a technique for measuring relative changes in flow. The clinical utility of the technique is now well established for a number of different disease processes. The technology assessment report of the American Academy of Neurology published in 1990 stated that TCD has established value in the assessment of patients with intracranial stenosis, collaterals, subarachnoid hemorrhage, and brain death .

How it works

Blood flow velocity is recorded by emitting a high-pitched sound wave from the ultrasound probe, which then bounces off of various materials to be measured by the same probe. A specific frequency is used (usually a multiple of 2 MHz), and the speed of the blood in relation to the probe causes a phase shift, wherein the frequency is increased or decreased. This frequency change directly correlates with the speed of the blood, which is then recorded electronically for later analysis. Normally a range of depths and angles must be measured to ascertain the correct velocities, as recording from an angle to the blood vessel yields an artificially low velocity.

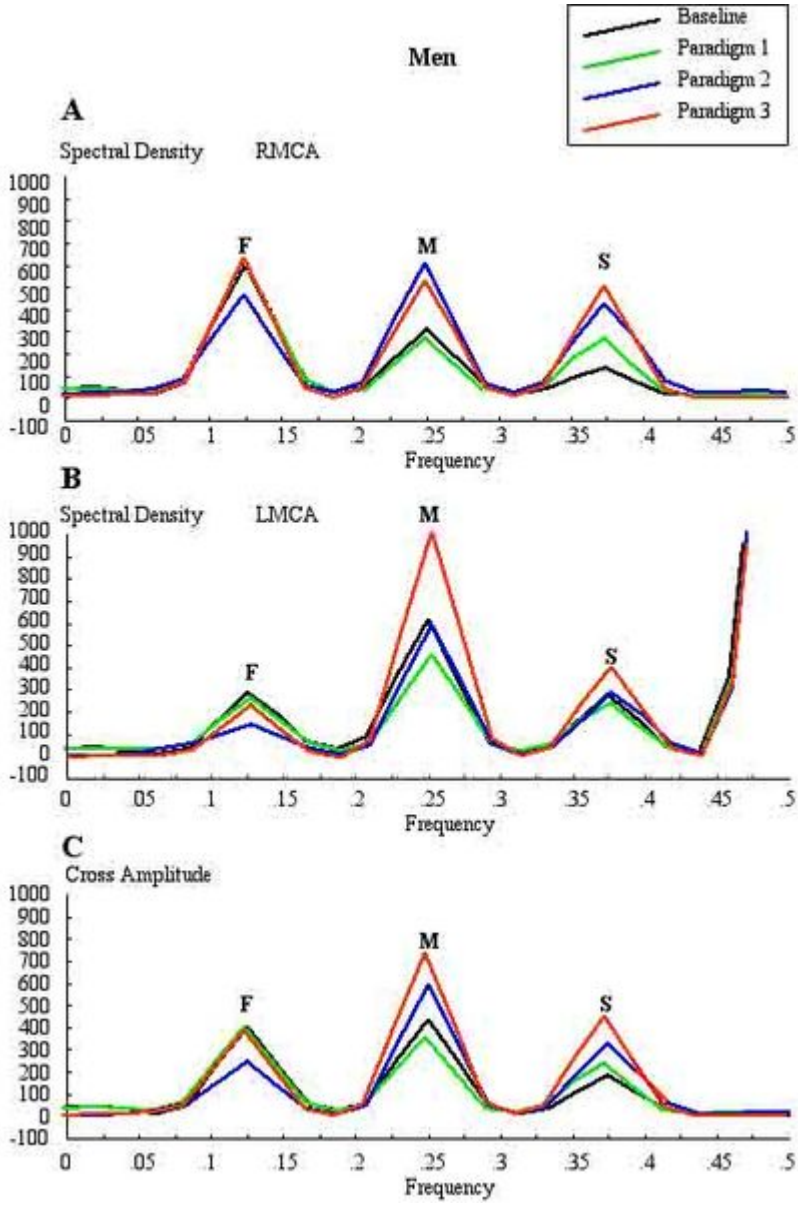
Because the bones of the skull block the transmission of ultrasound, regions with thinner walls - insonation windows - must be used for analyzing. For this reason, recording is performed in the temporal region above the cheekbone/zygomatic arch, through the eyes, below the jaw, and from the back of the head. Patient age, gender, race and other factors affect bone thickness, making some examinations more difficult or even impossible. Most can still be performed to obtain acceptable responses, sometimes requiring using alternate sites from which to view the vessels.

Functional Transcranial Doppler (fTCD)

Functional transcranial Doppler sonography (fTCD) is a neuroimaging tool for measuring cerebral blood flow velocity changes due to neural activation during cognitive tasks. Functional TCD utilizes pulse-wave Doppler technology to record blood flow velocities in the anterior, middle, and posterior cerebral arteries. Similar to other neuroimaging techniques such as functional magnetic resonance imaging or positron emission tomography, fTCD is based on a close coupling between regional cerebral blood flow changes and neural activation. Due to a continuous monitoring of blood flow velocity, TCD offers an excellent temporal resolution in comparison to other neuroimaging techniques. The technique is noninvasive and easy to apply. Blood flow velocity measurements are robust against movement artifacts. Since its introduction the technique has contributed substantially to the elucidation of the hemispheric organization of cognitive, motor, and sensory functions in adults and children. fTCD has been

particularly useful for the study of cerebral lateralization of major brain functions such as language, facial processing, color processing, intelligence processing and gender-related differences . Moreover, most established neuroanatomical substrates for brain function are perfused by the major cerebral arteries that could be directly insonated.

Functional Transcranial Doppler Spectroscopy (fTCDS)



Spectral Density Plots Right and Left Middle Cerebral Arteries Cross Amplitude Plots in Men.

Conventional fTCD has limitations for the study of cerebral lateralization. For example, it may not differentiate the lateralising effects due to stimulus characteristics from those due to light responsiveness, and does not distinguish between flow signals emanating from cortical and subcortical branches of the cerebral arteries of the circle of Willis. Each

basal cerebral artery of the circle of Willis gives origin to two different systems of secondary vessels. The shorter of these two is called the ganglionic system, and the vessels belonging to it supply the thalami and corpora striata; the longer is the cortical system, and its vessels ramify in the pia mater and supply the cortex and subjacent brain substance. Furthermore, the cortical branches are divisible into two classes: long and short. The long or medullary arteries pass through the grey substance and penetrate the subjacent white substance to the depth of three or four centimetres. The short vessels are confined to the cortex. Both cortical and ganglionic systems do not communicate at any point in their peripheral distribution, but are entirely independent of each other, having between the parts supplied by the two systems, a borderline of diminished nutritive activity . While, the vessels of the ganglionic system are terminal vessels, the vessels of the cortical arterial system are not so strictly ‘‘terminal’’. Blood flow in these two systems in the MCA territory supplies 80% of both hemispheres , including most neural substrates implicated in facial processing, language processing and intelligence processing at cortical and subcortical structures. The measurements of mean blood flow velocity (MFV) in the MCA main stem could potentially provide information about downstream changes at cortical and subcortical sites within the MCA territory. Each distal arm of the MCA vascular system could be separated into ‘‘near’’ and ‘‘far’’ distal reflection sites for the cortical and ganglionic (subcortical) systems, respectively. To accomplish this objective, one method is to apply Fourier analysis to the periodic time series of MFV acquired during cognitive stimulations. Fourier analysis would yield peaks representing pulsatile energy from reflection sites at various harmonics, which are multiples of the fundamental frequency ;. McDonald in 1974 showed that the first five harmonics usually contain 90% of the entire pulsatile energy within the system of pressure/flow oscillations in the peripheral circulation. It could be presumed that each arm of the vascular system represents a single viscoelastic tube terminated by impedance, creating a single reflection site . Psychophysiologic stimulation induced vasomotor activity at each terminal site sets up a standing sinusoidal wave oscillation, comprising a summation of waves due to effects of incident, reflected, and re-reflected waves from distal to proximal point of measurement. fTCDS studies are performed with the participant placed in a supine posture with their head up at about 30 degrees. The probe holder headgear (e.g LAM-RAK (DWL, Sipplingen, Germany) are used with a base support on two earplugs and on the nasal ridge. Two 2-MHz probes are affixed in the probe holder and insonation performed to determine the optimal position for continuous monitoring of both MCA main stems at 50 mm depth from the surface of the probe. A serial recording of MFV for each stimulus is acquired and latter used for Fourier analysis. Fourier transform algorithm uses standard software (for example, Time series and forecasting module, Statistica for Macintosh, StatSoft, OK, USA). The most efficient standard Fourier algorithm requires that the length of the input series is equal to a power of 2. If this is not the case, additional computations have to be performed. To derive the required time series, the data were averaged in 10-second segments for 1-minute duration or each stimulus; yielding 6 data points for each participant and a total of 48 data points for all eight men and women, respectively. Smoothing the periodogram values was accomplished using a weighted moving average transformation. Hamming window was applied as a smoother ;. The spectral density estimates, derived from single series Fourier analysis, were plotted, and the frequency regions with the highest estimates were marked

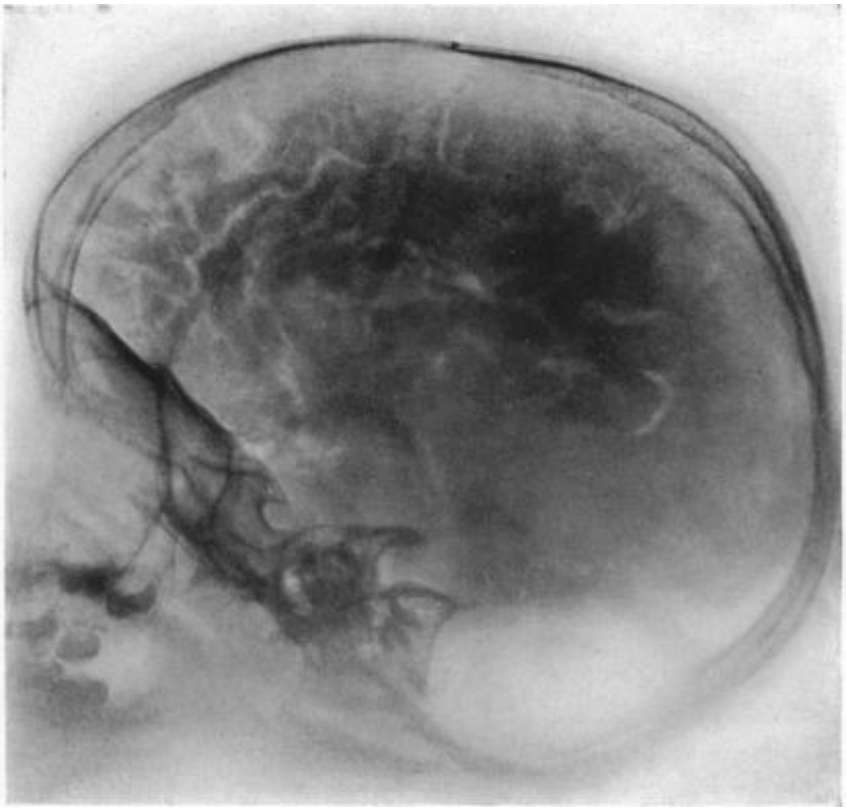
as peaks. The origins of the peaks are of interest in order to determine the reliability of the present technique. The fundamental (F), cortical (C) or memory (M), and subcortical (S) peaks occurred at regular frequency intervals of 0.125, 0.25, and 0.375, respectively. These frequencies could be converted to cycles per second (Hz), assuming that the fundamental frequency of cardiac oscillation was the mean heart rate. The fundamental frequency (F) of the first harmonic could be determined from the mean heart rate per second. For example, a heart rate of 74 bpm, suggests 74 cycles/60 or 1.23 Hz. In other words, the F-, C-, and S-peaks occurred at multiples of the first harmonic, at second and third harmonics, respectively. The distance of the reflection site for F-peak could be presumed to emanate from a site at $D1 = 1 / 4 * Wavelength$, or $D1 = c / 4f$, or $6.15(m / sec) / (4 * 1.23Hz) = 125cm$; where c is the assumed wave propagation velocity of the peripheral arterial tree according to McDonald, 1974. Given the vascular tortuosity, the estimated distance approximates that from the measurement site in the MCA main stem, to an imaginary site of summed reflections from the upper extremities, close to the finger tips when stretched sideways (Njemanze, 2007). The C-peak occurred at the second harmonic, such that the estimated arterial length (using common carotid $c = 5.5m / sec$) was given by $D2 = 1 / 8 * Wavelength$ or $c / 8 * 2f = 28cm$; and a frequency f2 of 2.46 Hz. The distance approximates the visible arterial length from the main stem of the MCA, through vascular tortuosity and around the cerebral convexity, to the end vessels at distal cortical sites such as the occipito-temporal junction on carotid angiograms of adults (Njemanze, 2007). The S-peak occurred at the third harmonic, and may have arisen from an estimated site at $D3 = 1 / 16 * Wavelength$ or $c / 16 * / 3f = 9.3cm$; and a frequency f3 of 3.69 Hz. The latter approximates the visible arterial length of the lenticulostriate vessels from the main stem of the MCA on carotid angiograms . Although not displayed, the fourth harmonic would be expected to arise from the MCA bifurcation in closest proximity to the measurement site in the main stem of the MCA. The pre-bifurcation length from the measurement point would be given by $D4 = 1 / 32 * Wavelength$ or $c / 32 * 4f = 3.5cm$; and a frequency f4 of 4.92 Hz. The calculated distance approximates that of the segment of MCA main stem just after the carotid bifurcation, where probably the ultrasound sample volume was placed, to the MCA bifurcation. Thus, these estimates approximate actual lengths. However, it has been suggested that the estimated distances may not correlate exactly with known morphometric dimensions of the arterial tree according to Campbell et al., 1989. The method was first described by Philip Njemanze in 2007, and was referred to as functional transcranial Doppler spectroscopy (fTCDS). fTCDS examines spectral density estimates of periodic processes induced during mental tasks, and hence offers a much more comprehensive picture of changes related to effects of a given mental stimulus. The spectral density estimates would be least affected by artefacts that lack periodicity, and filtering would reduce the effect of noise . The changes at the C-peak may show cortical longterm potential (CLTP) or cortical longterm depression (CLTD), which has been proposed to be suggest equivalents of cortical activity during learning (Njemanze, 2007) and cognitive processes>;. The flow velocity tracings are monitored during paradigm 1 comprising a checkerboard square as object perception are compared to whole face (paradigm 2) and facial element sorting task (paradigm 3). Fast Fourier transform calculations are used to obtain the spectral density and cross amplitude plots in the left and right middle cerebral arteries. The C-peak also called memory (M-peak) cortical peak could be seen arising during paradigm 3, a facial

element sorting task requiring iterative memory recall as a subject constantly spatially fits the puzzle by matching each facial element in paradigm 3 to that stored in memory (Paradigm 2) before proceeding to form the picture of the whole face.

Chapter 8

Pneumoencephalography and Pallidotomy

Pneumoencephalography



Pneumoencephalography

Pneumoencephalography (sometimes abbreviated PEG) is a medical procedure in which cerebrospinal fluid is drained to a small amount from around the brain and replaced with air, oxygen, or helium to allow the structure of the brain to show up more clearly on an X-ray picture. It is derived from ventriculography, an earlier and more primitive method where the air is injected through holes drilled in the skull.

The procedure was introduced in 1919 by the American neurosurgeon Walter Dandy.

Pneumoencephalography was performed extensively throughout the 20th century, but it was extremely painful and, as researchers would later discover, very dangerous. The test was generally not well tolerated by patients. Headaches and severe vomiting were common side effects. Replacement of the spinal fluid was by natural generation and therefore required recovery for as long as 2-3 months before normal movement was restored. Modern imaging techniques such as MRI and Computed tomography have largely replaced Pneumoencephalography.

By the late 1980s the procedure was largely abandoned by the medical community, having been supplanted by the CT scan and metrizamide cisternography. Today, pneumoencephalography is limited to the research field and is used under rare circumstances. A related procedure is pneumomyelography, where gas is used similarly to investigate the spinal canal.

Pneumoencephalography appears in popular culture in the movie *The Exorcist* (1973), when Linda Blair's Regan MacNeil character undergoes the procedure. It is also referred to in Episode 7, Season 7 of House M.D.

Pallidotomy

Pallidotomy is a procedure where a tiny electrical probe is placed in the globus pallidus (one of the basal ganglia of the brain), which is then heated to 80 degrees celsius for 60 s, to destroy a small area of brain cells. Pallidotomy is used to treat dyskinesias in patients with Parkinson's disease.

Technique

In a pallidotomy, the surgeon destroys a tiny part of the globus pallidus by creating a scar. This reduces the brain activity in that area, which may help relieve movement symptoms such as tremor and rigidity.

Before surgery, detailed brain scans using MRI are done to identify the precise location for treatment.

The patient is awake during the surgery, but the scalp area where instruments are inserted is numbed with a local anesthetic. The surgeon inserts a hollow probe through a small hole drilled in the skull to the target location. Liquid nitrogen is then circulated inside the probe. The cold probe destroys the targeted brain tissue. The probe is then removed, and the wound is closed.

The surgery usually requires a 2-day hospital stay. Most people recover completely within about 6 weeks.

Indications

Parkinsonism

Pallidotomy may be considered when a patient with advanced Parkinson's disease has:

- Developed severe motor fluctuations, such as dyskinesias and on-off responses, as a result of long-term levodopa treatment.
- Severe or disabling tremor, stiffness (rigidity), or slow movement (bradykinesia) that medication can no longer control.
- Pallidotomy is not a good choice for treatment when a person has not responded to levodopa. Some studies suggest that people with parkinsonian symptoms who do not improve with levodopa therapy do not gain much benefit from pallidotomy.

The most striking effect of pallidotomy is a reduction in dyskinesias that are caused by long-term levodopa therapy. This improvement can be seen almost immediately. By reducing these side effects, pallidotomy enables some patients to adjust their levodopa dosage, allowing for better symptom control.

Pallidotomy may reduce tremor, muscle rigidity, bradykinesis, and other motor symptoms. Balance and speech may also be improved.

It is not known how long the effects of pallidotomy can be expected to last. Benefits may fade over time in some people.

Reduction in use

Doctors rarely perform pallidotomy anymore. Instead, doctors use deep brain stimulation, a procedure that does not destroy brain tissue and has fewer risks than pallidotomy.

This type of brain surgery has less risk today than in the past because technology allows the surgeon to identify with great precision the area of the brain that will be treated. Serious permanent complications are not common, although less serious side effects are.

Complications

Complications of pallidotomy can include a stroke caused by bleeding in the brain.

Many people who have a stroke recover fully and benefit from pallidotomy. Pallidotomy has caused problems with thought and memory (cognitive impairment) in some people.

Other risks include:

- Infection.
- Seizures.

History

Stereotactic pallidotomy was pioneered by Dr. Hirotaro Narabayashi.

Chapter 9

Nerve Conduction Study and Mini–Mental State Examination

Nerve conduction study

A **nerve conduction study** (NCS) is a test commonly used to evaluate the function, especially the ability of electrical conduction, of the motor and sensory nerves of the human body.

Nerve conduction velocity (NCV) is a common measurement made during this test. The term NCV often is used to mean the actual test, but this may be misleading since velocity is only one measurement in the test suite.

Purposes

Nerve conduction studies are used mainly for evaluation of paresthesias (numbness, tingling, burning) and/or weakness of the arms and legs. The type of study required is dependent in part by the symptoms presented. A physical exam and thorough history also help to direct the investigation. Some of the common disorders which can be diagnosed by nerve conduction studies are:

- Peripheral neuropathy
- Carpal tunnel syndrome
- Ulnar neuropathy
- Guillain-Barré syndrome
- Facioscapulohumeral muscular dystrophy
- Spinal disc herniation

Description

The nerve conduction study consists of the following components:

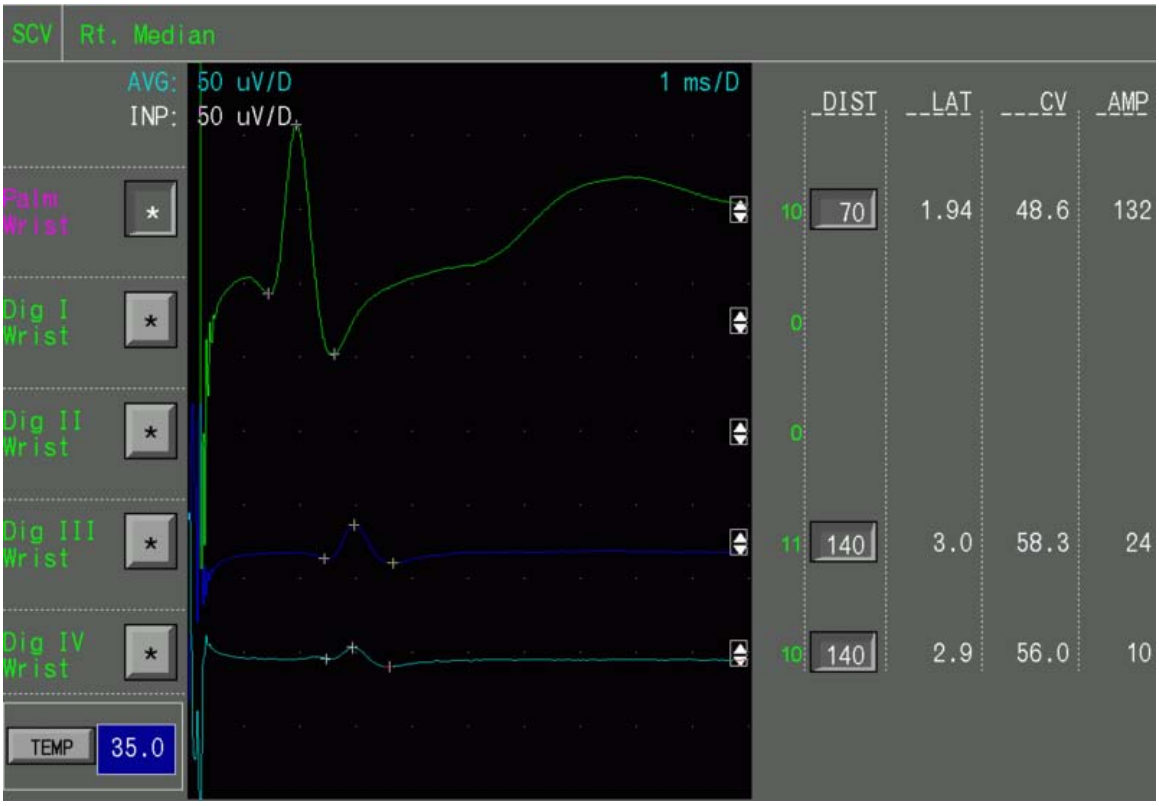
- Motor NCS
- Sensory NCS
- F-wave study
- H-reflex study

Motor NCS

Motor NCS are performed by electrical stimulation of a peripheral nerve and recording from a muscle supplied by this nerve. The time it takes for the electrical impulse to travel from the stimulation to the recording site is measured. This value is called the latency and is measured in milliseconds (ms). The size of the response - called the amplitude - is also measured. Motor amplitudes are measured in millivolts (mV). By stimulating in two or more different locations along the same nerve, the NCV across different segments can be determined. Calculations are performed using the distance between the different stimulating electrodes and the difference in latencies.

Sensory NCS

Sensory NCS are performed by electrical stimulation of a peripheral nerve and recording from a purely-sensory portion of the nerve, such as on a finger.The recording electrode is the more proximal of the two. Like the motor studies, sensory latencies are on the scale of milliseconds. Sensory amplitudes are much smaller than the motor amplitudes, usually in the microvolt (μV) range. The sensory NCV is calculated based upon the latency and the distance between the stimulating and recording electrode.



Sensory NCS: An example screenshot showing the results of a sensory nerve conduction velocity study of the right median nerve.

F-wave study

F-wave study uses supramaximal stimulation of a motor nerve and recording of action potentials from a muscle supplied by the nerve. This is not a reflex, per se, in that the action potential travels from the site of the stimulating electrode in the limb to the spinal cord's anterior horn cell and back to the limb in the same nerve that was stimulated. The F-wave latency can be used to derive the conduction velocity of nerve between the limb and spine, whereas the motor and sensory nerve conduction studies evaluate conduction in the segment of the limb. F waves vary in latency and an abnormal variance is called "chrono dispersion". Conduction velocity is derived by measuring the limb length in millimeters from the stimulation site to the corresponding spinal segment (C7 spinous process to wrist crease for median nerve). This is multiplied by 2 as it goes to the cord and returns to the muscle (2D). 2D is divided by the latency difference between mean F and M and 1 millisecond subtracted (F-M-1). The formula is 2D/(F-M-1).

H-reflex study

H-reflex study uses stimulation of a nerve and recording the reflex electrical discharge from a muscle in the limb. This also evaluates conduction between the limb and the spinal cord, but in this case, the afferent impulses (those going towards the spinal cord)

are in sensory nerves while the efferent impulses (those coming from the spinal cord) are in motor nerves. This process cannot be changed.

Small-pain-fibers method

In 1998 a small-pain-fibers (spf-NCS) method was cleared by the FDA. This method uses an electrical stimulus with a neuroselective frequency to determine the minimum voltage causing conduction. Rather than comparing the data with population averages on a bell-shaped curve, which at best has about 65% sensitivity, the patient is his own control. In a three year LSU Pain Center study it was found that the nerve requiring the greatest voltage to cause conduction of the A-delta (Fast Pain) fibers identified nerve root pathology with 95% sensitivity. Besides being painless, the test is fast. A new version, uses a potentiometer to objectively measure the amplitude of the action potential at a distant site along the nerve being tested. The previous version relied on the patient reporting a sensation when the nerve fired. The spf-NCS does not require myelin loss to detect function change, so velocity is not measured.

Interpretation of nerve conductions

The interpretation of nerve conduction studies is complex, but in general, different pathological processes result in changes in latencies, motor and/or sensory amplitudes, or slowing of the conduction velocities to differing degrees. For example, slowing of the NCV usually indicates there is damage to the myelin. Another example, slowing across the wrist for the motor and sensory latencies of the median nerve indicates focal compression of the median nerve at the wrist, called carpal tunnel syndrome. On the other hand, slowing of all nerve conductions in more than one limb indicates generalized diseased nerves, or generalized peripheral neuropathy. People with diabetes mellitus often develop generalized peripheral neuropathy.

Patient risk

Nerve conduction studies are very helpful to diagnose certain diseases of the nerves of the body. The test is not invasive, but can be a little painful due to the electrical shocks. The shocks are associated with a low amount of electrical current so they are not dangerous to anyone. Patients with a permanent pacemaker or other such implanted stimulators such as deep brain stimulators or spinal cord stimulators must tell the examiner prior to the study. This does not prevent the study, but special precautions are taken.

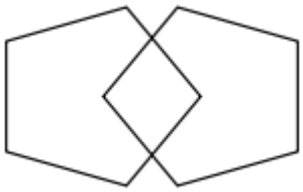
Mini–mental state examination

The **mini–mental state examination** (**MMSE**) or **Folstein test** is a brief 30-point questionnaire test that is used to screen for cognitive impairment. It is commonly used in medicine to screen for dementia. It is also used to estimate the severity of cognitive impairment at a given point in time and to follow the course of cognitive changes in an individual over time, thus making it an effective way to document an individual's response to treatment.

In the time span of about 10 minutes it samples various functions including arithmetic, memory and orientation. It was introduced by Folstein *et al.* in 1975,. This test is not the same thing as a mental status examination. The standard MMSE form which is currently published by Psychological Assessment Resources is based on its original 1975 conceptualization, with minor subsequent modifications by the authors.

Various other tests are also used, such as the Hodkinson abbreviated mental test score (1972, geriatrics) or the General Practitioner Assessment Of Cognition as well as longer formal tests for deeper analysis of specific deficits.

Test features



Interlocking pentagons used for the last question

The MMSE test includes simple questions and problems in a number of areas: the time and place of the test, repeating lists of words, arithmetic such as the serial sevens, language use and comprehension, and basic motor skills. For example, one question asks to copy a drawing of two pentagons (shown on the right).

Although consistent application of identical questions increases the reliability of comparisons made using the scale, the test is sometimes customized (for example, for use on patients that are intubated, blind, or partially immobilized. Also, some have questioned the use of the test on the deaf.) However, the number of points assigned per category is usually consistent:

Category	Possible points	Description
Orientation to time	5	From broadest to most narrow. Orientation to time has been correlated with future decline.
Orientation to place	5	From broadest to most narrow. This is sometimes narrowed down to streets, and sometimes to floor.
Registration	3	Repeating named prompts
Attention and calculation	5	Serial sevens, or spelling "world" backwards It has been suggested that serial sevens may be more appropriate in a population where English is not the first language.
Recall	3	Registration recall
Language	2	Name a pencil and a watch
Repetition	1	Speaking back a phrase
Complex commands	6	Varies. Can involve drawing figure shown.

Interpretation

Any score greater than or equal to 25 points (out of 30) is effectively normal (intact). Below this, scores can indicate severe (≤9 points), moderate (10-20 points) or mild (21-24 points). The raw score may also need to be corrected for educational attainment and age. Low to very low scores correlate closely with the presence of dementia, although other mental disorders can also lead to abnormal findings on MMSE testing. The presence of purely physical problems can also interfere with interpretation if not properly noted; for example, a patient may be physically unable to hear or read instructions properly, or may have a motor deficit that affects writing and drawing skills.

Copyright issues

The MMSE was first published in 1975 as an appendix to an article written by Dr. Marshal F. Folstein, Dr. Susan E. Folstein, and Dr. Paul R. McHugh. It was published in Volume 12 of the *Journal of Psychiatric Research*, a journal published by Pergamon Press, Ltd. at the time. While the MMSE was attached as an appendix to the article, the copyright ownership of the MMSE remained with Dr. Folstein and his two coauthors. Pergamon Press was subsequently taken over by Elsevier Science, Ltd., and Elsevier took over Pergamon Press' copyright ownership of the Journal of Psychiatric Research.

The authors later transferred all their intellectual property rights, including the copyright of the MMSE, to MiniMental, LLC. A copyright registration form for the MMSE designating MiniMental, LLC as the owner of the MMSE copyright was registered with the U.S. Copyright Office on June 8, 2000 (Form TX 5-228-282). In March 2001, MiniMental, LLC entered into an exclusive agreement with Psychological Assessment Resources, Inc. (PAR), granting PAR the exclusive rights to publish, license, and manage all intellectual property rights to the MMSE in all media and languages in the world.

Despite the many free versions of the test that are available on the internet, the official version is copyrighted and must be ordered through PAR. The enforcement of the copyright on the MMSE has been compared to "stealth", or "submarine" patents, where a patent applicant would wait until an invention gains widespread popularity until allowing the patent to issue and only then commencing enforcement (such patent applications are no longer possible with changes made to the patent term). The enforcement of the copyright has led to researchers looking for alternative strategies in assessing cognition.

In February 2010, PAR released a second edition of the MMSE; 10 foreign language translations (French, German, Dutch, Spanish for the US, Spanish for Latin America, European Spanish, Hindi, Russian, Italian, and Simplified Chinese) were also created.

Chapter 10

Electromyography

Electromyography (EMG) is a technique for evaluating and recording the electrical activity produced by skeletal muscles. EMG is performed using an instrument called an **electromyograph**, to produce a record called an **electromyogram**. An electromyograph detects the electrical potential generated by muscle cells when these cells are electrically or neurologically activated. The signals can be analyzed to detect medical abnormalities, activation level, recruitment order or to analyze the biomechanics of human or animal movement.

Electrical characteristics

The electrical source is the muscle membrane potential of about -90 mV. Measured EMG potentials range between less than 50 μV and up to 20 to 30 mV, depending on the muscle under observation.

Typical repetition rate of muscle motor unit firing is about 7–20 Hz, depending on the size of the muscle (eye muscles versus seat (gluteal) muscles), previous axonal damage and other factors. Damage to motor units can be expected at ranges between 450 and 780 mV.

History

The first documented experiments dealing with EMG started with Francesco Redi's works in 1666. Redi discovered a highly specialized muscle of the electric ray fish (Electric Eel) generated electricity. By 1773, Walsh had been able to demonstrate that the Eel fish's muscle tissue could generate a spark of electricity. In 1792, a publication entitled *De Viribus Electricitatis in Motu Musculari Commentarius* appeared, written by Luigi Galvani, in which the author demonstrated that electricity could initiate muscle contractions. Six decades later, in 1849, Dubois-Raymond discovered that it was also possible to record electrical activity during a voluntary muscle contraction. The first actual recording of this activity was made by Marey in 1890, who also introduced the term electromyography. In 1922, Gasser and Erlanger used an oscilloscope to show the

electrical signals from muscles. Because of the stochastic nature of the myoelectric signal, only rough information could be obtained from its observation. The capability of detecting electromyographic signals improved steadily from the 1930s through the 1950s, and researchers began to use improved electrodes more widely for the study of muscles. Clinical use of surface EMG (sEMG) for the treatment of more specific disorders began in the 1960s. Hardyck and his researchers were the first (1966) practitioners to use sEMG. In the early 1980s, Cram and Steger introduced a clinical method for scanning a variety of muscles using an EMG sensing device.

It is not until the middle of the 1980s that integration techniques in electrodes had sufficiently advanced to allow batch production of the required small and lightweight instrumentation and amplifiers. At present, a number of suitable amplifiers are commercially available. In the early 1980s, cables that produced signals in the desired microvolt range became available. Recent research has resulted in a better understanding of the properties of surface EMG recording. Surface electromyography is increasingly used for recording from superficial muscles in clinical or kinesiological protocols, where intramuscular electrodes are used for investigating deep muscles or localized muscle activity.

There are many applications for the use of EMG. EMG is used clinically for the diagnosis of neurological and neuromuscular problems. It is used diagnostically by gait laboratories and by clinicians trained in the use of biofeedback or ergonomic assessment. EMG is also used in many types of research laboratories, including those involved in biomechanics, motor control, neuromuscular physiology, movement disorders, postural control, and physical therapy.

Procedure

There are two kinds of EMG in widespread use: surface EMG and intramuscular (needle and fine-wire) EMG. To perform intramuscular EMG, a needle electrode or a needle containing two fine-wire electrodes is inserted through the skin into the muscle tissue. A trained professional (such as a neurologist, physiatrist, or physical therapist) observes the electrical activity while inserting the electrode. The insertional activity provides valuable information about the state of the muscle and its innervating nerve. Normal muscles at rest make certain, normal electrical signals when the needle is inserted into them. Then the electrical activity when the muscle is at rest is studied. Abnormal spontaneous activity might indicate some nerve and/or muscle damage. Then the patient is asked to contract the muscle smoothly. The shape, size, and frequency of the resulting motor unit potentials are judged. Then the electrode is retracted a few millimeters, and again the activity is analyzed until at least 10–20 units have been collected. Each electrode track gives only a very local picture of the activity of the whole muscle. Because skeletal muscles differ in the inner structure, the electrode has to be placed at various locations to obtain an accurate study.

Intramuscular EMG may be considered too invasive or unnecessary in some cases. Instead, a surface electrode may be used to monitor the general picture of muscle

activation, as opposed to the activity of only a few fibres as observed using an intramuscular EMG. This technique is used in a number of settings; for example, in the physiotherapy clinic, muscle activation is monitored using surface EMG and patients have an auditory or visual stimulus to help them know when they are activating the muscle (biofeedback).

A motor unit is defined as one motor neuron and all of the muscle fibers it innervates. When a motor unit fires, the impulse (called an action potential) is carried down the motor neuron to the muscle. The area where the nerve contacts the muscle is called the neuromuscular junction, or the motor end plate. After the action potential is transmitted across the neuromuscular junction, an action potential is elicited in all of the innervated muscle fibers of that particular motor unit. The sum of all this electrical activity is known as a motor unit action potential (MUAP). This electrophysiologic activity from multiple motor units is the signal typically evaluated during an EMG. The composition of the motor unit, the number of muscle fibres per motor unit, the metabolic type of muscle fibres and many other factors affect the shape of the motor unit potentials in the myogram.

Nerve conduction testing is also often done at the same time as an EMG to diagnose neurological diseases.

Some patients can find the procedure somewhat painful, whereas others experience only a small amount of discomfort when the needle is inserted. The muscle or muscles being tested may be slightly sore for a day or two after the procedure.

Normal results

Muscle tissue at rest is normally electrically inactive. After the electrical activity caused by the irritation of needle insertion subsides, the electromyograph should detect no abnormal spontaneous activity (i.e., a muscle at rest should be electrically silent, with the exception of the area of the neuromuscular junction, which is, under normal circumstances, very spontaneously active). When the muscle is voluntarily contracted, action potentials begin to appear. As the strength of the muscle contraction is increased, more and more muscle fibers produce action potentials. When the muscle is fully contracted, there should appear a disorderly group of action potentials of varying rates and amplitudes (a complete recruitment and interference pattern).

Abnormal results

EMG is used to diagnose diseases that generally may be classified into one of the following categories: neuropathies, neuromuscular junction diseases and myopathies.

Neuropathic disease has the following defining EMG characteristics:

- An action potential amplitude that is twice normal due to the increased number of fibres per motor unit because of reinnervation of denervated fibres

- An increase in duration of the action potential
- A decrease in the number of motor units in the muscle (as found using motor unit number estimation techniques)

Myopathic disease has these defining EMG characteristics:

- A decrease in duration of the action potential
- A reduction in the area to amplitude ratio of the action potential
- A decrease in the number of motor units in the muscle (in extremely severe cases only)

Because of the individuality of each patient and disease, some of these characteristics may not appear in every case.

Abnormal results may be caused by the following medical conditions (please note this is nowhere near an exhaustive list of conditions that can result in abnormal EMG studies):

- Alcoholic neuropathy
- Amyotrophic lateral sclerosis
- Anterior compartment syndrome of the lower leg
- Axillary nerve dysfunction
- Becker's muscular dystrophy
- Brachial plexopathy
- Carpal tunnel syndrome
- Centronuclear myopathy
- Cervical spondylosis
- Charcot-Marie-Tooth disease
- Chronic Immune Demyelinating Poly[radiculo]neuropathy (CIDP)
- Common peroneal nerve dysfunction
- Denervation (reduced nervous stimulation)
- Dermatomyositis
- Distal median nerve
- Duchenne muscular dystrophy
- Facioscapulohumeral muscular dystrophy (Landouzy-Dejerine)
- Familial periodic paralysis
- Femoral nerve dysfunction
- Fields condition
- Friedreich's ataxia
- Guillain-Barre
- Lambert-Eaton Syndrome
- Mononeuritis multiplex
- Mononeuropathy
- Motor neurone disease
- Multiple system atrophy
- Myasthenia gravis
- Myopathy (muscle degeneration, which may be caused by a number of disorders, including muscular dystrophy)
- Myotubular myopathy
- Neuromyotonia
- Peripheral neuropathy
- Poliomyelitis
- Polymyositis
- Radial nerve dysfunction
- Sciatic nerve dysfunction
- Sensorimotor polyneuropathy
- Sleep bruxism
- Spinal stenosis
- Thyrotoxic periodic paralysis
- Tibial nerve dysfunction
- Ulnar nerve dysfunction

dysfunction

EMG signal decomposition

EMG signals are essentially made up of superimposed motor unit action potentials (MUAPs) from several motor units. For a thorough analysis, the measured EMG signals can be decomposed into their constituent MUAPs. MUAPs from different motor units tend to have different characteristic shapes, while MUAPs recorded by the same electrode from the same motor unit are typically similar. Notably MUAP size and shape depend on where the electrode is located with respect to the fibers and so can appear to be different if the electrode moves position. EMG decomposition is non-trivial, although many methods have been proposed.

Applications of EMG

EMG signals are used in many clinical and biomedical applications. EMG is used as a diagnostics tool for identifying neuromuscular diseases, assessing low-back pain, kinesiology, and disorders of motor control. EMG signals are also used as a control signal for prosthetic devices such as prosthetic hands, arms, and lower limbs.

EMG can be used to sense isometric muscular activity where no movement is produced. This enables definition of a class of subtle motionless gestures to control interfaces without being noticed and without disrupting the surrounding environment. These signals can be used to control a prosthesis or as a control signal for an electronic device such as a mobile phone or PDA.

EMG signals have been targeted as control for flight systems. The Human Senses Group at the NASA Ames Research Center at Moffett Field, CA seeks to advance man-machine interfaces by directly connecting a person to a computer. In this project, an EMG signal is used to substitute for mechanical joysticks and keyboards. EMG has also been used in research towards a "wearable cockpit," which employs EMG-based gestures to manipulate switches and control sticks necessary for flight in conjunction with a goggle-based display.

Unvoiced speech recognition recognizes speech by observing the EMG activity of muscles associated with speech. It is targeted for use in noisy environments, and may be helpful for people without vocal cords and people with aphasia.

EMG has also been used as a control signal for computers and other devices. An interface device based on EMG could be used to control moving objects, such as mobile robots or an electric wheelchair. This may be helpful for individuals that cannot operate a joystick-controlled wheelchair. Surface EMG recordings may also be a suitable control signal for some interactive video games.

A joint project involving Microsoft, the University of Washington in Seattle, and the University of Toronto in Canada has explored using muscle signals from hand gestures as an interface device. A patent based on this research was submitted on June 26, 2008.

Chapter 11

Anterior Temporal Lobectomy and Glasgow Coma Scale

Anterior temporal lobectomy

Anterior temporal lobectomy is the complete removal of the anterior portion of the temporal lobe of the brain. It is a treatment option in temporal lobe epilepsy for those in whom anticonvulsant medications do not control epileptic seizures.

The techniques for removing temporal lobe tissue vary from resection of large amounts of tissue, including lateral temporal cortex along with medial structures, to more restricted anterior temporal lobectomy (ATL) to more restricted removal of only the medial structures (selective amygdalohippocampectomy, SAH).

Nearly all reports of seizure outcome following these procedures indicate that the best outcome group includes patients with MRI evidence of mesial temporal sclerosis (hippocampal atrophy with increased T-2 signal.) The range of seizure-free outcomes for these patients is reported to be between 80 and 90%, which is typically reported as a sub-set of data within a larger surgical series.

Open surgical procedures such as ATL have inherent risks including damage to the brain (either directly or indirectly by injury to important blood vessels), bleeding (which can require re-operation), blood loss (which can require transfusion), and infection. Furthermore, open procedures require several days of care in the hospital including at least one night in an intensive care unit. Although such treatment can be costly, multiple studies have demonstrated that ATL in patients who have failed at least two anticonvulsant drug trials (thereby meeting the criteria for medically intractable temporal lobe epilepsy) has lower mortality, lower morbidity and lower long-term cost in comparison with continued medical therapy without surgical intervention.

The strongest evidence supporting ATL over continued medical therapy for medically refractory temporal lobe epilepsy is a prospective, randomized trial of ATL compared to

best medical therapy (anticonvulsants), which convincingly demonstrated that the seizure-free rate after surgery was ~ 60% as compared to only 8% for the medicine only group. Furthermore, there was no mortality in the surgery group, while there was seizure-related mortality in the medical therapy group. Therefore, ATL is considered the standard of care for patients with medically-intractable mesial temporal lobe epilepsy.

Glasgow Coma Scale

Glasgow Coma Scale or **GCS** is a neurological scale that aims to give a reliable, objective way of recording the conscious state of a person for initial as well as subsequent assessment. A patient is assessed against the criteria of the scale, and the resulting points give a patient score between 3 (indicating deep unconsciousness) and either 14 (original scale) or 15 (the more widely used modified or revised scale).

GCS was initially used to assess level of consciousness after head injury, and the scale is now used by first aid, EMS, and doctors as being applicable to all acute medical and trauma patients. In hospitals it is also used in monitoring chronic patients in intensive care.

The scale was published in 1974 by Graham Teasdale and Bryan J. Jennett, professors of neurosurgery at the University of Glasgow's Institute of Neurological Sciences at the city's Southern General Hospital.

GCS is used as part of several ICU scoring systems, including APACHE II, SAPS II, and SOFA, to assess the status of the central nervous system. A similar scale, the Rancho Los Amigos Scale is used to assess the recovery of traumatic brain injury patients.

Elements of the scale

Glasgow Coma Scale

	1	2	3	4	5	6
Eyes	Does not open eyes	Opens eyes in response to painful stimuli	Opens eyes in response to voice	Opens eyes spontaneously	N/A	N/A
Verbal	Makes no sounds	Incomprehensible sounds	Utters inappropriate words	Confused, disoriented	Oriented, converses normally	N/A
Motor	Makes no movements	Extension to painful stimuli (decerebrate response)	Abnormal flexion to painful stimuli (decorticate response)	Flexion / Withdrawal to painful stimuli	Localizes painful stimuli	Obeys commands

The scale comprises three tests: eye, verbal and motor responses. The three values separately as well as their sum are considered. The lowest possible GCS (the sum) is 3 (deep coma or death), while the highest is 15 (fully awake person).

Best eye response (E)

There are 4 grades starting with the most severe:

1. No eye opening
2. Eye opening in response to pain. (Patient responds to pressure on the patient's fingernail bed; if this does not elicit a response, supraorbital and sternal pressure or rub may be used.)
3. Eye opening to speech. (Not to be confused with an awaking of a sleeping person; such patients receive a score of 4, not 3.)
4. Eyes opening spontaneously

Best verbal response (V)

There are 5 grades starting with the most severe:

1. No verbal response
2. Incomprehensible sounds. (Moaning but no words.)
3. Inappropriate words. (Random or exclamatory articulated speech, but no conversational exchange)
4. Confused. (The patient responds to questions coherently but there is some disorientation and confusion.)
5. Oriented. (Patient responds coherently and appropriately to questions such as the patient's name and age, where they are and why, the year, month, etc.)

Best motor response (M)

There are 6 grades starting with the most severe:

1. No motor response
2. Extension to pain (abduction of arm, internal rotation of shoulder, pronation of forearm, extension of wrist, *decerebrate response*)
3. Abnormal flexion to pain (adduction of arm, internal rotation of shoulder, pronation of forearm, flexion of wrist, *decorticate response*)
4. Flexion/Withdrawal to pain (flexion of elbow, supination of forearm, flexion of wrist when supra-orbital pressure applied ; pulls part of body away when nailbed pinched)
5. Localizes to pain. (Purposeful movements towards painful stimuli; e.g., hand crosses mid-line and gets above clavicle when supra-orbital pressure applied.)
6. Obeys commands. (The patient does simple things as asked.)

Interpretation

Individual elements as well as the sum of the score are important. Hence, the score is expressed in the form "GCS 9 = E2 V4 M3 at 07:35".

Generally, brain injury is classified as:

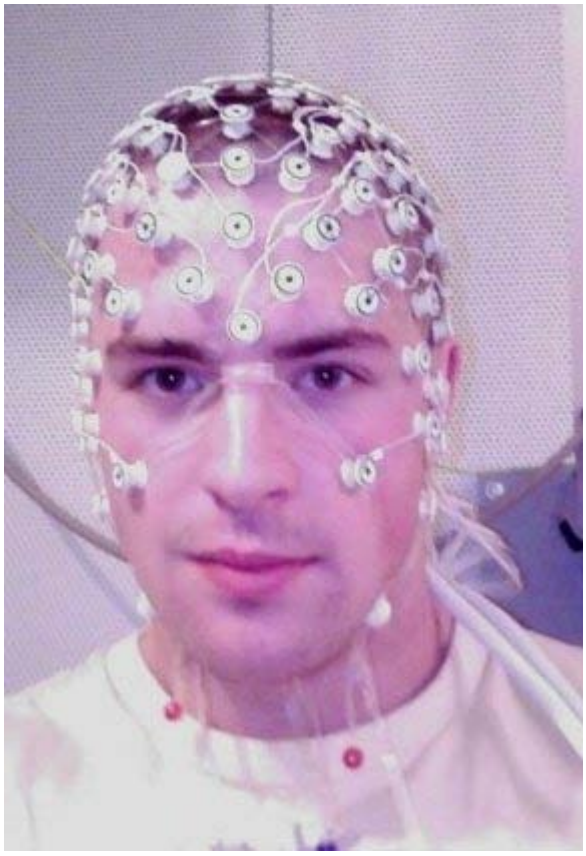
- Severe, with GCS ≤ 8
- Moderate, GCS 9 - 12
- Minor, GCS ≥ 13.

Tracheal intubation and severe facial/eye swelling or damage make it impossible to test the verbal and eye responses. In these circumstances, the score is given as 1 with a modifier attached e.g. 'E1c' where 'c' = closed, or 'V1t' where t = tube. A composite might be 'GCS 5tc'. This would mean, for example, eyes closed because of swelling = 1, intubated = 1, leaving a motor score of 3 for 'abnormal flexion'. Often the 1 is left out, so the scale reads Ec or Vt.

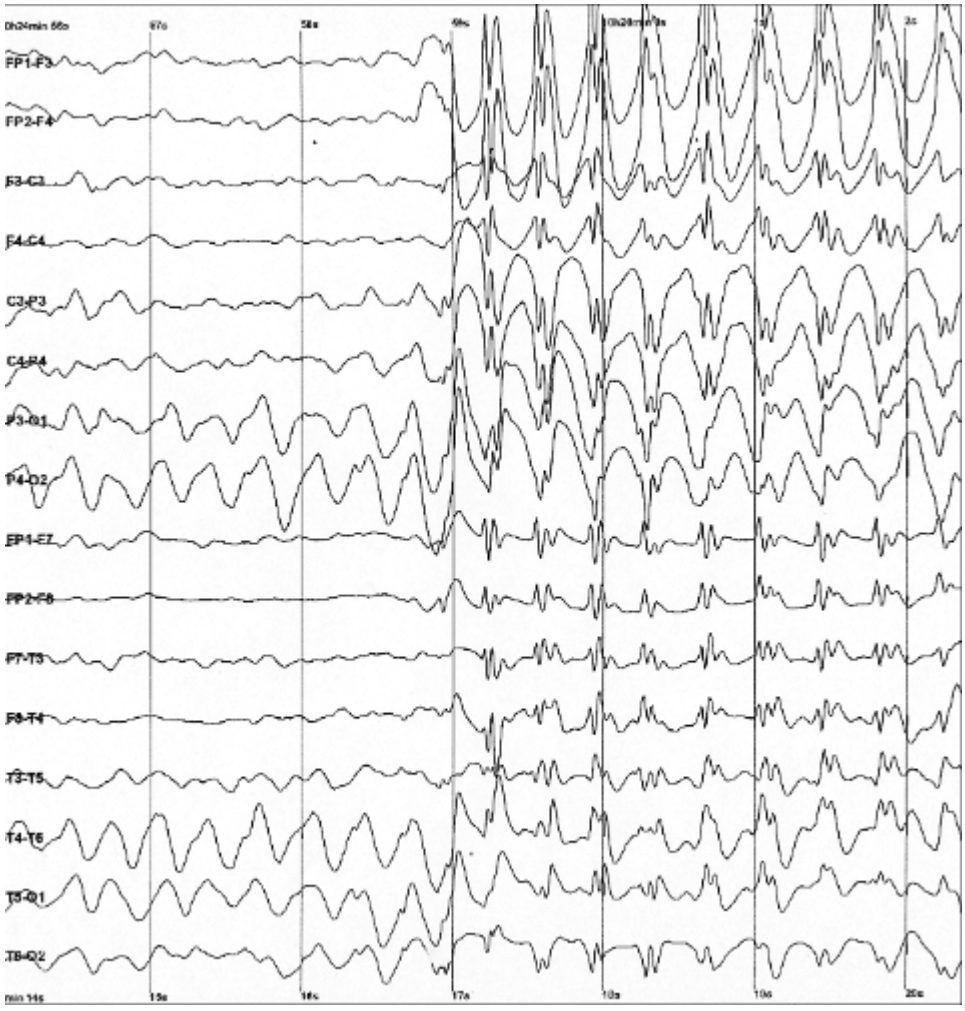
The GCS has limited applicability to children, especially below the age of 36 months (where the verbal performance of even a healthy child would be expected to be poor). Consequently the Pediatric Glasgow Coma Scale, a separate yet closely related scale, was developed for assessing younger children.

Chapter 12

Electroencephalography



An EEG recording net (Electrical Geodesics, Inc.) being used on a participant in a brain wave study.



Epileptic spike and wave discharges monitored with EEG.

Electroencephalography (**EEG**) is the recording of electrical activity along the scalp produced by the firing of neurons within the brain. In clinical contexts, EEG refers to the recording of the brain's spontaneous electrical activity over a short period of time, usually 20–40 minutes, as recorded from multiple electrodes placed on the scalp. In neurology, the main diagnostic application of EEG is in the case of epilepsy, as epileptic activity can create clear abnormalities on a standard EEG study. A secondary clinical use of EEG is in the diagnosis of coma, encephalopathies, and brain death. EEG used to be a first-line method for the diagnosis of tumors, stroke and other focal brain disorders, but this use has decreased with the advent of anatomical imaging techniques such as MRI and CT.

Derivatives of the EEG technique include evoked potentials (EP), which involves averaging the EEG activity time-locked to the presentation of a stimulus of some sort (visual, somatosensory, or auditory). Event-related potentials (ERPs) refer to averaged EEG responses that are time-locked to more complex processing of stimuli; this technique is used in cognitive science, cognitive psychology, and psychophysiological research.

Source of EEG activity

The brain's electrical charge is maintained by billions of neurons. Neurons are electrically charged (or "polarized") by membrane transport proteins that pump ions across their membranes. When a neuron receives a signal from its neighbor via an action potential, it responds by releasing ions into the space outside the cell. Ions of like charge repel each other, and when many ions are pushed out of many neurons at the same time, they can push their neighbors, who push their neighbors, and so on, in a wave. This process is known as volume conduction. When the wave of ions reaches the electrodes on the scalp, they can push or pull electrons on the metal on the electrodes. Since metal conducts the push and pull of electrons easily, the difference in push, or voltage, between any two electrodes can be measured by a voltmeter. Recording these voltages over time gives us the EEG.

The electric potentials generated by single neurons are far too small to be picked by EEG or MEG. EEG activity therefore always reflects the summation of the synchronous activity of thousands or millions of neurons that have similar spatial orientation. If the cells do not have similar spatial orientation, their ions do not line up and create waves to be detected. Pyramidal neurons of the cortex are thought to produce most EEG signal because they are well-aligned and fire together. Because voltage fields fall off with the square of the distance, activity from deep sources is more difficult to detect than currents near the skull.

Scalp EEG activity shows oscillations at a variety of frequencies. Several of these oscillations have characteristic frequency ranges, spatial distributions and are associated with different states of brain functioning (e.g., waking and the various sleep stages). These oscillations represent synchronized activity over a network of neurons. The neuronal networks underlying some of these oscillations are understood (e.g., the thalamocortical resonance underlying sleep spindles), while many others are not (e.g., the system that generates the posterior basic rhythm). Research that measures both EEG and neuron spiking finds the relationship between the two is complex with the power of surface EEG only in two bands that of gamma and delta relating to neuron spike activity.

Clinical use

A routine clinical EEG recording typically lasts 20–30 minutes (plus preparation time) and usually involves recording from scalp electrodes. Routine EEG is typically used in the following clinical circumstances:

- to distinguish epileptic seizures from other types of spells, such as psychogenic non-epileptic seizures, syncope (fainting), sub-cortical movement disorders and migraine variants.
- to differentiate "organic" encephalopathy or delirium from primary psychiatric syndromes such as catatonia
- to serve as an adjunct test of brain death
- to prognosticate, in certain instances, in patients with coma

- to determine whether to wean anti-epileptic medications

At times, a routine EEG is not sufficient, particularly when it is necessary to record a patient while he/she is having a seizure. In this case, the patient may be admitted to the hospital for days or even weeks, while EEG is constantly being recorded (along with time-synchronized video and audio recording). A recording of an actual seizure (i.e., an ictal recording, rather than an inter-ictal recording of a possibly epileptic patient at some period between seizures) can give significantly better information about whether or not a spell is an epileptic seizure and the focus in the brain from which the seizure activity emanates.

Epilepsy monitoring is typically done:

- to distinguish epileptic seizures from other types of spells, such as psychogenic non-epileptic seizures, syncope (fainting), sub-cortical movement disorders and migraine variants.
- to characterize seizures for the purposes of treatment
- to localize the region of brain from which a seizure originates for work-up of possible seizure surgery

Additionally, EEG may be used to monitor certain procedures:

- to monitor the depth of anesthesia
- as an indirect indicator of cerebral perfusion in carotid endarterectomy
- to monitor amobarbital effect during the Wada test

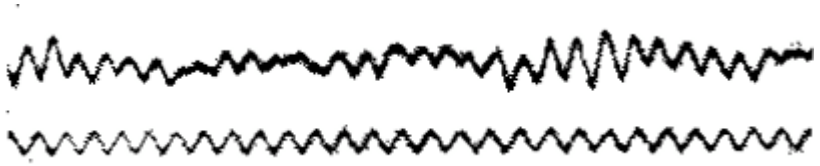
EEG can also be used in intensive care units for brain function monitoring:

- to monitor for non-convulsive seizures/non-convulsive status epilepticus
- to monitor the effect of sedative/anesthesia in patients in medically induced coma (for treatment of refractory seizures or increased intracranial pressure)
- to monitor for secondary brain damage in conditions such as subarachnoid hemorrhage (currently a research method)

If a patient with epilepsy is being considered for resective surgery, it is often necessary to localize the focus (source) of the epileptic brain activity with a resolution greater than what is provided by scalp EEG. This is because the cerebrospinal fluid, skull and scalp *smear* the electrical potentials recorded by scalp EEG. In these cases, neurosurgeons typically implant strips and grids of electrodes (or penetrating depth electrodes) under the dura mater, through either a craniotomy or a burr hole. The recording of these signals is referred to as electrocorticography (ECoG), subdural EEG (sdEEG) or intracranial EEG (icEEG)--all terms for the same thing. The signal recorded from ECoG is on a different scale of activity than the brain activity recorded from scalp EEG. Low voltage, high frequency components that cannot be seen easily (or at all) in scalp EEG can be seen clearly in ECoG. Further, smaller electrodes (which cover a smaller parcel of brain

surface) allow even lower voltage, faster components of brain activity to be seen. Some clinical sites record from penetrating microelectrodes.

Research use



The first human EEG recording obtained by Hans Berger in 1924. The upper tracing is EEG, and the lower is a 10 Hz timing signal.

EEG, and its derivative, ERPs, are used extensively in neuroscience, cognitive science, cognitive psychology, and psychophysiological research. Many techniques used in research contexts are not standardized sufficiently to be used in the clinical context.

A different method to study brain function is functional magnetic resonance imaging (fMRI). Some benefits of EEG compared to fMRI include:

- Hardware costs are significantly lower for EEG sensors versus an fMRI machine
- EEG sensors can be deployed into a wider variety of environments than can a bulky, immobile fMRI machine
- EEG enables higher temporal resolution, on the order of milliseconds, rather than seconds
- EEG is relatively tolerant of subject movement versus an fMRI (where the subject must remain completely still)
- EEG is silent, which allows for better study of the responses to auditory stimuli
- EEG does not aggravate claustrophobia

Limitations of EEG as compared with fMRI include:

- Significantly lower spatial resolution
- ERP studies require relatively simple paradigms, compared with block-design fMRI studies

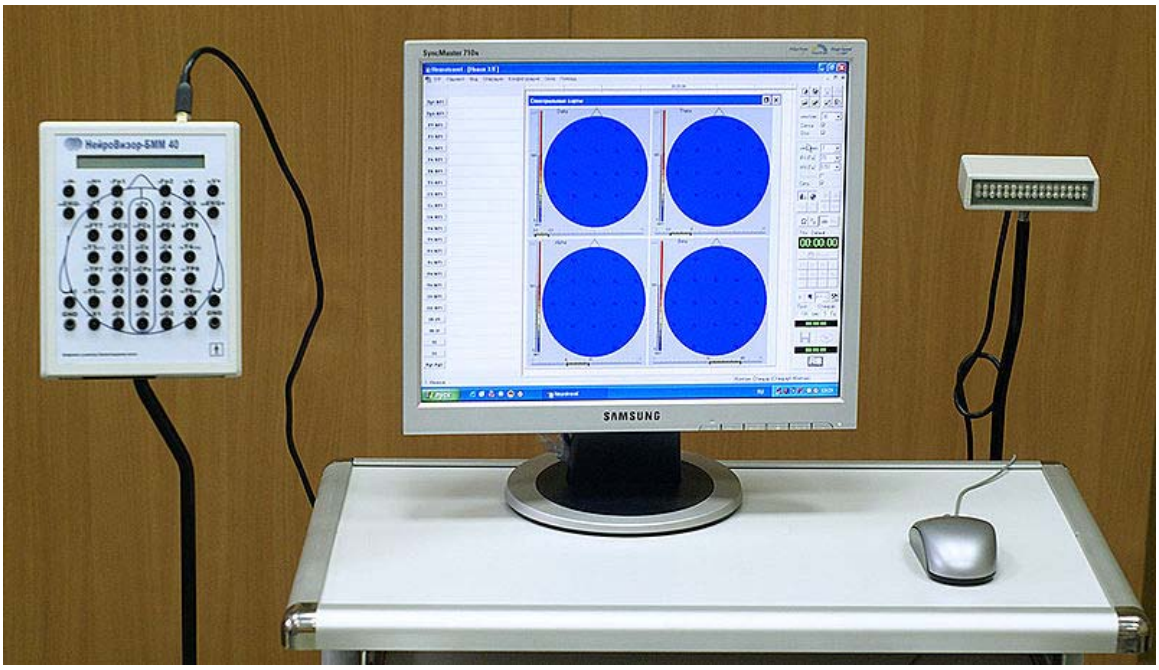
EEG recordings have been successfully obtained simultaneously with fMRI scans, though successful simultaneous recording requires that several technical issues be overcome, such as the presence of ballistocardiographic artifact, MRI pulse artifact and the induction of electrical currents in EEG wires that move within the strong magnetic fields of the MRI.

EEG also has some characteristics that compare favorably with behavioral testing:

- EEG can detect covert processing (i.e., processing that does not require a response)
- EEG can be used in subjects who are incapable of making a motor response

- Some ERP components can be detected even when the subject is not attending to the stimuli
- As compared with other reaction time paradigms, ERPs can elucidate stages of processing (rather than just the final end result)

Method



Computer Electroencephalograph *Neurovisor-BMM 40*

In conventional scalp EEG, the recording is obtained by placing electrodes on the scalp with a conductive gel or paste, usually after preparing the scalp area by light abrasion to reduce impedance due to dead skin cells. Many systems typically use electrodes, each of which is attached to an individual wire. Some systems use caps or nets into which electrodes are embedded; this is particularly common when high-density arrays of electrodes are needed.

Electrode locations and names are specified by the International 10–20 system for most clinical and research applications (except when high-density arrays are used). This system ensures that the naming of electrodes is consistent across laboratories. In most clinical applications, 19 recording electrodes (plus ground and system reference) are used. A smaller number of electrodes are typically used when recording EEG from neonates. Additional electrodes can be added to the standard set-up when a clinical or research application demands increased spatial resolution for a particular area of the brain. High-density arrays (typically via cap or net) can contain up to 256 electrodes more-or-less evenly spaced around the scalp.

Each electrode is connected to one input of a differential amplifier (one amplifier per pair of electrodes); a common system reference electrode is connected to the other input of

each differential amplifier. These amplifiers amplify the voltage between the active electrode and the reference (typically 1,000–100,000 times, or 60–100 dB of voltage gain). In analog EEG, the signal is then filtered (next paragraph), and the EEG signal is output as the deflection of pens as paper passes underneath. Most EEG systems these days, however, are digital, and the amplified signal is digitized via an analog-to-digital converter, after being passed through an anti-aliasing filter. Analog-to-digital sampling typically occurs at 256–512 Hz in clinical scalp EEG; sampling rates of up to 20 kHz are used in some research applications.

During the recording, a series of activation procedures may be used. These procedures may induce normal or abnormal EEG activity that might not otherwise be seen. These procedures include hyperventilation, photic stimulation (with a strobe light), eye closure, mental activity, sleep and sleep deprivation. During (inpatient) epilepsy monitoring, a patient's typical seizure medications may be withdrawn.

The digital EEG signal is stored electronically and can be filtered for display. Typical settings for the high-pass filter and a low-pass filter are 0.5-1 Hz and 35–70 Hz, respectively. The high-pass filter typically filters out slow artifact, such as electrogalvanic signals and movement artifact, whereas the low-pass filter filters out high-frequency artifacts, such as electromyographic signals. An additional notch filter is typically used to remove artifact caused by electrical power lines (60 Hz in the United States and 50 Hz in many other countries). As part of an evaluation for epilepsy surgery, it may be necessary to insert electrodes near the surface of the brain, under the surface of the dura mater. This is accomplished via burr hole or craniotomy. This is referred to variously as "electrocorticography (ECoG)", "intracranial EEG (I-EEG)" or "subdural EEG (SD-EEG)". Depth electrodes may also be placed into brain structures, such as the amygdala or hippocampus, structures, which are common epileptic foci and may not be "seen" clearly by scalp EEG. The electrocorticographic signal is processed in the same manner as digital scalp EEG (above), with a couple of caveats. ECoG is typically recorded at higher sampling rates than scalp EEG because of the requirements of Nyquist theorem—the subdural signal is composed of a higher predominance of higher frequency components. Also, many of the artifacts that affect scalp EEG do not impact ECoG, and therefore display filtering is often not needed.

A typical adult human EEG signal is about 10μV to 100 μV in amplitude when measured from the scalp and is about 10–20 mV when measured from subdural electrodes.

Since an EEG voltage signal represents a difference between the voltages at two electrodes, the display of the EEG for the reading encephalographer may be set up in one of several ways. The representation of the EEG channels is referred to as a *montage.*

Bipolar montage

> Each channel (i.e., waveform) represents the difference between two adjacent electrodes. The entire montage consists of a series of these channels. For example, the channel "Fp1-F3" represents the difference in voltage between the Fp1 electrode and the F3 electrode. The next channel in the montage, "F3-C3,"

represents the voltage difference between F3 and C3, and so on through the entire array of electrodes.

Referential montage
: Each channel represents the difference between a certain electrode and a designated reference electrode. There is no standard position for this reference; it is, however, at a different position than the "recording" electrodes. Midline positions are often used because they do not amplify the signal in one hemisphere vs. the other. Another popular reference is "linked ears," which is a physical or mathematical average of electrodes attached to both earlobes or mastoids.

Average reference montage
: The outputs of all of the amplifiers are summed and averaged, and this averaged signal is used as the common reference for each channel.

Laplacian montage
: Each channel represents the difference between an electrode and a weighted average of the surrounding electrodes.

When analog (paper) EEGs are used, the technologist switches between montages during the recording in order to highlight or better characterize certain features of the EEG. With digital EEG, all signals are typically digitized and stored in a particular (usually referential) montage; since any montage can be constructed mathematically from any other, the EEG can be viewed by the electroencephalographer in any display montage that is desired.

The EEG is read by a neurologist, optimally one who has specific training in the interpretation of EEGs. This is done by visual inspection of the waveforms, called graphoelements. The use of computer signal processing of the EEG—so-called quantitative EEG—is somewhat controversial when used for clinical purposes (although there are many research uses).

Limitations

EEG has several limitations. Most important is its poor spatial resolution. EEG is most sensitive to a particular set of post-synaptic potentials: those generated in superficial layers of the cortex, on the crests of gyri directly abutting the skull and radial to the skull. Dendrites, which are deeper in the cortex, inside sulci, in midline or deep structures (such as the cingulate gyrus or hippocampus), or producing currents that are tangential to the skull, have far less contribution to the EEG signal.

The meninges, cerebrospinal fluid and skull "smear" the EEG signal, obscuring its intracranial source.

It is mathematically impossible to reconstruct a unique intracranial current source for a given EEG signal, as some currents produce potentials that cancel each other out. This is referred to as the inverse problem. However, much work has been done to produce remarkably good estimates of, at least, a localized electric dipole that represents the recorded currents.

EEG vs fMRI and PET

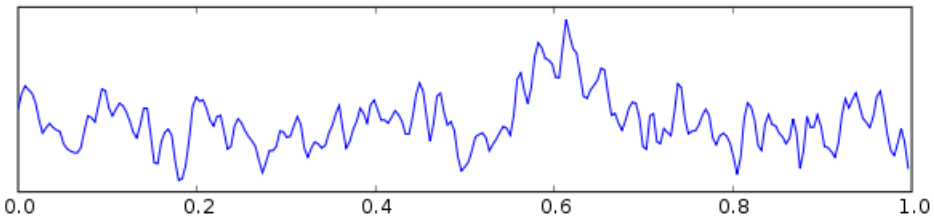
EEG has several strong points as a tool for exploring brain activity. EEG's can detect changes within a millisecond timeframe, excellent considering an action potential takes approximately 0.5-130 milliseconds to propagate across a single neuron, depending on the type of neuron. Other methods of looking at brain activity, such as PET and fMRI have time resolution between seconds and minutes. EEG measures the brain's electrical activity directly, while other methods record changes in blood flow (e.g., SPECT, fMRI) or metabolic activity (e.g., PET), which are indirect markers of brain electrical activity. EEG can be used simultaneously with fMRI so that high-temporal-resolution data can be recorded at the same time as high-spatial-resolution data, however, since the data derived from each occurs over a different time course, the data sets do not necessarily represent exactly the same brain activity. There are technical difficulties associated with combining these two modalities, including the need to remove the *MRI gradient artifact* present during MRI acquisition and the ballistocardiographic artifact (resulting from the pulsatile motion of blood and tissue) from the EEG. Furthermore, currents can be induced in moving EEG electrode wires due to the magnetic field of the MRI.

EEG vs MEG

EEG reflects correlated synaptic activity caused by post-synaptic potentials of cortical neurons. The ionic currents involved in the generation of fast action potentials may not contribute greatly to the averaged field potentials representing the EEG . More specifically, the scalp electrical potentials that produce EEG are generally thought to be caused by the extracellular ionic currents caused by dendritic electrical activity, whereas the fields producing magnetoencephalographic signals are associated with intracellular ionic currents .

EEG can be recorded at the same time as MEG so that data from these complementary high-time-resolution techniques can be combined.

Normal activity



One second of EEG signal

The EEG is typically described in terms of (1) rhythmic activity and (2) transients. The rhythmic activity is divided into bands by frequency. To some degree, these frequency bands are a matter of nomenclature (i.e., any rhythmic activity between 8–12 Hz can be described as "alpha"), but these designations arose because rhythmic activity within a certain frequency range was noted to have a certain distribution over the scalp or a certain

biological significance. Frequency bands are usually extracted using spectral methods (for instance Welch) as implemented for instance in freely available EEG software such as EEGLAB.

Most of the cerebral signal observed in the scalp EEG falls in the range of 1–20 Hz (activity below or above this range is likely to be artifactual, under standard clinical recording techniques).

Comparison table

Comparison of EEG bands

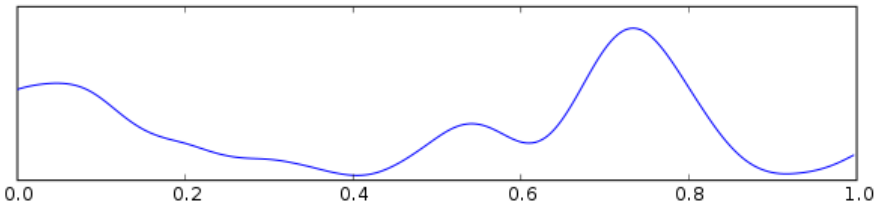
Type	Frequency (Hz)	Location	Normally	Pathologically
Delta	up to 4	frontally in adults, posteriorly in children; high amplitude waves	• adults slow wave sleep • in babies • Has been found during some continuous attention tasks (Kirmizi-Alsan et al. 2006)	• subcortical lesions • diffuse lesions • metabolic encephalopathy hydrocephalus • deep midline lesions
Theta	4 – <8	Found in locations not related to task at hand	• young children • drowsiness or arousal in older children and adults • idling • Associated with inhibition of elicited responses (has been found to spike in situations where a person is actively trying to repress a response or action) (Kirmizi-Alsan et al. 2006).	• focal subcortical lesions • metabolic encephalopathy • deep midline disorders • some instances of hydrocephalus
Alpha	8 – 13	posterior regions of head, both sides, higher in amplitude on dominant side. Central sites (c3-c4) at rest .	• relaxed/reflecting • closing the eyes • Also associated with inhibition control, seemingly with the purpose of timing inhibitory activity in different locations	• coma

			across the brain (Klimesch, Sauseng, & Hanslmayr 2007; Coan & Allen 2008).	
Beta	>13 – 30	both sides, symmetrical distribution, most evident frontally; low amplitude waves	• alert/working • active, busy or anxious thinking, active concentration	• benzodiazepines
Gamma	30 – 100+	Somatosensory cortex	• Displays during cross-modal sensory processing (perception that combines two different senses, such as sound and sight) (Kisley & Cornwell 2006; Kanayama, Sato, & Ohira 2007; Nieuwenhuis, Yeung, & Cohen 2004) • Also is shown during short term memory matching of recognized objects, sounds, or tactile sensations (Herrmann, Frund, & Lenz 2009)	• A decrease in gamma band activity may be associated with cognitive decline, especially when related the theta band; however, this has not been proven for use as a clinical diagnostic measurement yet (Moretti et al. 2009).
Mu	8 – 13	Sensorimotor cortex	• Shows rest state motor neurons (Gastaut, 1952).	• Mu suppression could be indicative for motor mirror neurons working, and deficits in Mu suppression, and thus in mirror neurons, might play a role in autism. (Oberman et al.,

2005)

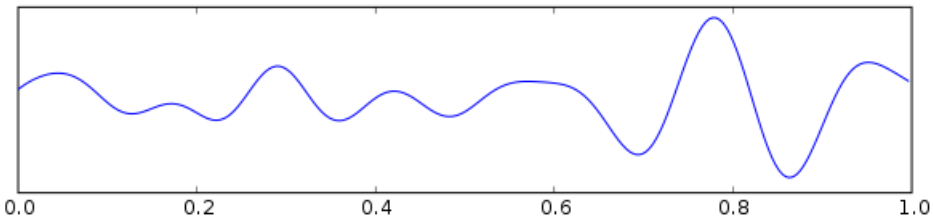
It should be noted that while these are the universally recognized ranges, they are not concrete definitions of the range of brain-waves. While researchers tend to follow these guidelines, many scholars use their own specific boundaries depending on the range they choose to focus on. Additionally, some researchers define the bands using decimal values rather than rounding to whole numbers (for example, one researcher may define the lower Beta band cut-off as 12.1, while another may use the value 13), while still others sometimes divide the bands into sub-bands. Generally, this is only done for the sake of analysis.

Wave patterns



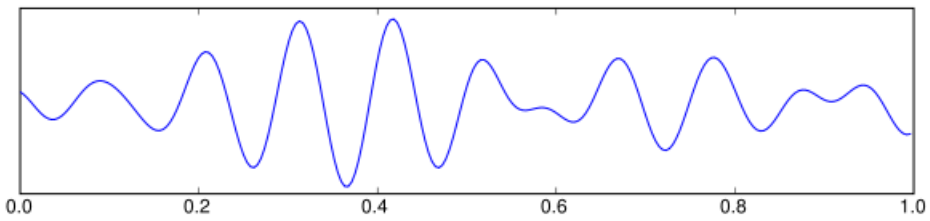
delta waves.

- Delta is the frequency range up to 4 Hz. It tends to be the highest in amplitude and the slowest waves. It is seen normally in adults in slow wave sleep. It is also seen normally in babies. It may occur focally with subcortical lesions and in general distribution with diffuse lesions, metabolic encephalopathy hydrocephalus or deep midline lesions. It is usually most prominent frontally in adults (e.g. FIRDA - Frontal Intermittent Rhythmic Delta) and posteriorly in children (e.g. OIRDA - Occipital Intermittent Rhythmic Delta).



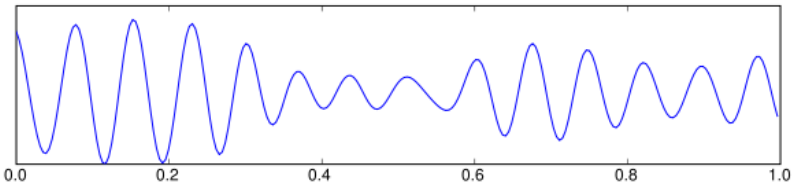
theta waves.

- Theta is the frequency range from 4 Hz to 7 Hz. Theta is seen normally in young children. It may be seen in drowsiness or arousal in older children and adults; it can also be seen in meditation. Excess theta for age represents abnormal activity. It can be seen as a focal disturbance in focal subcortical lesions; it can be seen in generalized distribution in diffuse disorder or metabolic encephalopathy or deep midline disorders or some instances of hydrocephalus. On the contrary this range has been associated with reports of relaxed, meditative, and creative states.



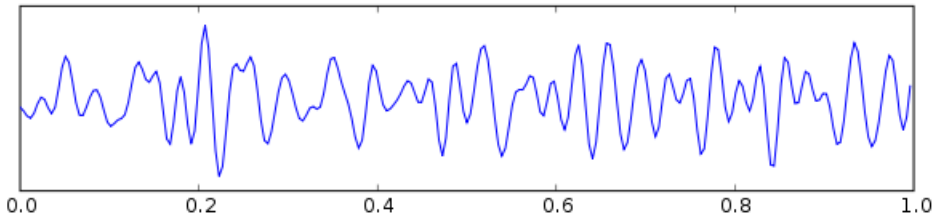
alpha waves.

- Alpha is the frequency range from 8 Hz to 12 Hz. Hans Berger named the first rhythmic EEG activity he saw as the "alpha wave". This was the "posterior basic rhythm" (also called the "posterior dominant rhythm" or the "posterior alpha rhythm"), seen in the posterior regions of the head on both sides, higher in amplitude on the dominant side. It emerges with closing of the eyes and with relaxation, and attenuates with eye opening or mental exertion. The posterior basic rhythm is actually slower than 8 Hz in young children (therefore technically in the theta range).



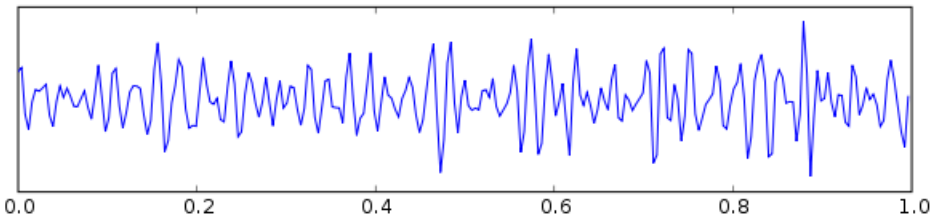
sensorimotor rhythm aka mu rhythm.

In addition to the posterior basic rhythm, there are other normal alpha rhythms such as the mu rhythm (alpha activity in the contralateral sensory and motor cortical areas that emerges when the hands and arms are idle; and the "third rhythm" (alpha activity in the temporal or frontal lobes). Alpha can be abnormal; for example, an EEG that has diffuse alpha occurring in coma and is not responsive to external stimuli is referred to as "alpha coma".



beta waves.

- Beta is the frequency range from 12 Hz to about 30 Hz. It is seen usually on both sides in symmetrical distribution and is most evident frontally. Beta activity is closely linked to motor behavior and is generally attenuated during active movements. Low amplitude beta with multiple and varying frequencies is often associated with active, busy or anxious thinking and active concentration. Rhythmic beta with a dominant set of frequencies is associated with various pathologies and drug effects, especially benzodiazepines. It may be absent or reduced in areas of cortical damage. It is the dominant rhythm in patients who are alert or anxious or who have their eyes open.



gamma waves.

- Gamma is the frequency range approximately 30–100 Hz. Gamma rhythms are thought to represent binding of different populations of neurons together into a network for the purpose of carrying out a certain cognitive or motor function.

- Mu ranges 8–13 Hz., and partly overlaps with other frequencies. It reflects the synchronous firing of motor neurons in rest state. Mu suppression is thought to reflect motor mirror neuron systems, because when an action is observed, the pattern extinguishes, possibly because of the normal neuronal system and the mirror neuron system "go out of sync", and interfere with each other.

"Ultra-slow" or "near-DC" activity is recorded using DC amplifiers in some research contexts. It is not typically recorded in a clinical context because the signal at these frequencies is susceptible to a number of artifacts.

Some features of the EEG are transient rather than rhythmic. Spikes and sharp waves may represent seizure activity or interictal activity in individuals with epilepsy or a predisposition toward epilepsy. Other transient features are normal: vertex waves and sleep spindles are seen in normal sleep.

Note that there are types of activity that are statistically uncommon, but not associated with dysfunction or disease. These are often referred to as "normal variants." The mu rhythm is an example of a normal variant.

The normal Electroencephalography (EEG) varies by age. The neonatal EEG is quite different from the adult EEG. The EEG in childhood generally has slower frequency oscillations than the adult EEG.

The normal EEG also varies depending on state. The EEG is used along with other measurements (EOG, EMG) to define sleep stages in polysomnography. Stage I sleep (equivalent to drowsiness in some systems) appears on the EEG as drop-out of the posterior basic rhythm. There can be an increase in theta frequencies. Santamaria and Chiappa cataloged a number of the variety of patterns associated with drowsiness. Stage II sleep is characterized by sleep spindles—transient runs of rhythmic activity in the 12–14 Hz range (sometimes referred to as the "sigma" band) that have a frontal-central maximum. Most of the activity in Stage II is in the 3–6 Hz range. Stage III and IV sleep are defined by the presence of delta frequencies and are often referred to collectively as "slow-wave sleep." Stages I-IV comprise non-REM (or "NREM") sleep. The EEG in REM (rapid eye movement) sleep appears somewhat similar to the awake EEG.

EEG under general anesthesia depends on the type of anesthetic employed. With halogenated anesthetics, such as halothane or intravenous agents, such as propofol, a rapid (alpha or low beta), nonreactive EEG pattern is seen over most of the scalp, especially anteriorly; in some older terminology this was known as a WAR (widespread anterior rapid) pattern, contrasted with a WAIS (widespread slow) pattern associated with high doses of opiates. Anesthetic effects on EEG signals are beginning to be understood at the level of drug actions on different kinds of synapses and the circuits that allow synchronized neuronal activity.

Artifacts

Biological artifacts

Electrical signals detected along the scalp by an EEG, but that originate from non-cerebral origin are called artifacts. EEG data is almost always contaminated by such artifacts. The amplitude of artifacts can be quite large relative to the size of amplitude of the cortical signals of interest. This is one of the reasons why it takes considerable experience to correctly interpret EEGs clinically. Some of the most common types of biological artifacts include:

- Eye-induced artifacts (includes eye blinks, eye movements and extra-ocular muscle activity)
- EKG (cardiac) artifacts
- EMG (muscle activation)-induced artifacts
- Glossokinetic artifacts

The most prominent eye-induced artifacts are caused by the potential difference between the cornea and retina, which is quite large compared to cerebral potentials. When the eyes and eyelids are completely still, this corneo-retinal dipole does not affect EEG. However, blinks occur several times per minute, the eyes movements occur several times per second. Eyelid movements, occurring mostly during blinking or vertical eye movements, elicit a large potential seen mostly in the difference between the Electrooculography (EOG) channels above and below the eyes. An established explanation of this potential regards the eyelids as sliding electrodes that short-circuit the positively charged cornea to

the extra-ocular skin. Rotation of the eyeballs, and consequently of the corneo-retinal dipole, increases the potential in electrodes towards which the eyes are rotated, and decrease the potentials in the opposing electrodes. Eye movements called saccades also generate transient electromyographic potentials, known as saccadic spike potentials (SPs). The spectrum of these SPs overlaps the gamma-band, and seriously confounds analysis of induced gamma-band responses, requiring tailored artifact correction approaches. Purposeful or reflexive eye blinking also generates electromyographic potentials, but more importantly there is reflexive movement of the eyeball during blinking that gives a characteristic artifactual appearance of the EEG.

Eyelid fluttering artifacts of a characteristic type were previously called Kappa rhythm (or Kappa waves). It is usually seen in the prefrontal leads, that is, just over the eyes. Sometimes they are seen with mental activity. They are usually in the Theta (4–7 Hz) or Alpha (8–13 Hz) range. They were named because they were believed to originate from the brain. Later study revealed they were generated by rapid fluttering of the eyelids, sometimes so minute that it was difficult to see. They are in fact noise in the EEG reading, and should not technically be called a rhythm or wave. Therefore, current usage in electroencephalography refers to the phenomenon as an eyelid fluttering artifact, rather than a Kappa rhythm (or wave).

Some of these artifacts can be useful in various applications. The EOG signals, for instance, can be used to detect and track eye-movements, which are very important in polysomnography, and is also in conventional EEG for assessing possible changes in alertness, drowsiness or sleep.

EKG artifacts are quite common and can be mistaken for spike activity. Because of this, modern EEG acquisition commonly includes a one-channel EKG from the extremities. This also allows the EEG to identify cardiac arrhythmias that are an important differential diagnosis to syncope or other episodic/attack disorders.

Glossokinetic artifacts are caused by the potential difference between the base and the tip of the tongue. Minor tongue movements can contaminate the EEG, especially in parkinsonian and tremor disorders.

Environmental artifacts

In addition to artifacts generated by the body, many artifacts originate from outside the body. Movement by the patient, or even just settling of the electrodes, may cause *electrode pops*, spikes originating from a momentary change in the impedance of a given electrode. Poor grounding of the EEG electrodes can cause significant 50 or 60 Hz artifact, depending on the local power system's frequency. A third source of possible interference can be the presence of an IV drip; such devices can cause rhythmic, fast, low-voltage bursts, which may be confused for spikes.

Artifact correction

Recently, independent component analysis techniques have been used to correct or remove EEG contaminates. These techniques attempt to "unmix" the EEG signals into some number of underlying components. There are many source separation algorithms, often assuming various behaviors or natures of EEG. Regardless, the principle behind any particular method usually allow "remixing" only those components that would result in "clean" EEG by nullifying (zeroing) the weight of unwanted components. Fully automated artifact rejection methods, which use ICA, have also been developed.

Abnormal activity

Abnormal activity can broadly be separated into epileptiform and non-epileptiform activity. It can also be separated into focal or diffuse.

Focal epileptiform discharges represent fast, synchronous potentials in a large number of neurons in a somewhat discrete area of the brain. These can occur as interictal activity, between seizures, and represent an area of cortical irritability that may be predisposed to producing epileptic seizures. Interictal discharges are not wholly reliable for determining whether a patient has epilepsy nor where his/her seizure might originate.

Generalized epileptiform discharges often have an anterior maximum, but these are seen synchronously throughout the entire brain. They are strongly suggestive of a generalized epilepsy.

Focal non-epileptiform abnormal activity may occur over areas of the brain where there is focal damage of the cortex or white matter. It often consists of an increase in slow frequency rhythms and/or a loss of normal higher frequency rhythms. It may also appear as focal or unilateral decrease in amplitude of the EEG signal.

Diffuse non-epileptiform abnormal activity may manifest as diffuse abnormally slow rhythms or bilateral slowing of normal rhythms, such as the PBR.

Intracortical Encephalogram electrodes and sub-dural electrodes can be used in tandem to discriminate and discretize artifact from epileptiform and other severe neurological events.

More advanced measures of abnormal EEG signals have also recently received attention as possible biomarkers for different disorders such as Alzheimer's disease.

History

A timeline of the history of EEG is given by Swartz. Richard Caton (1842–1926), a physician practicing in Liverpool, presented his findings about electrical phenomena of the exposed cerebral hemispheres of rabbits and monkeys in the British Medical Journal in 1875. In 1890, Polish physiologist Adolf Beck published an investigation of

spontaneous electrical activity of the brain of rabbits and dogs that included rhythmic oscillations altered by light.

In 1912, Russian physiologist, Vladimir Vladimirovich Pravdich-Neminsky published the first animal EEG and the evoked potential of the mammalian (dog). In 1914, Napoleon Cybulski and Jelenska-Macieszyna photographed EEG-recordings of experimentally induced seizures.

German physiologist and psychiatrist Hans Berger (1873–1941) recorded the first human EEG in 1924. Expanding on work previously conducted on animals by Richard Caton and others, Berger also invented the electroencephalogram (giving the device its name), an invention described "as one of the most surprising, remarkable, and momentous developments in the history of clinical neurology". His discoveries were first confirmed by British scientists Edgar Douglas Adrian and B. H. C. Matthews in 1934 and developed by them.

In 1934, Fisher and Lowenback first demonstrated epileptiform spikes. In 1935 Gibbs, Davis and Lennox described interictal spike waves and the 3 cycles/s pattern of clinical absence seizures, which began the field of clinical electroencephalography. Subsequently, in 1936 Gibbs and Jasper reported the interictal spike as the focal signature of epilepsy. The same year, the first EEG laboratory opened at Massachusetts General Hospital.

Franklin Offner (1911–1999), professor of biophysics at Northwestern University developed a prototype of the EEG that incorporated a piezoelectric inkwriter called a Crystograph (the whole device was typically known as the Offner Dynograph).

In 1947, The American EEG Society was founded and the first International EEG congress was held. In 1953 Aserinsky and Kleitman describe REM sleep.

In the 1950s, William Grey Walter developed an adjunct to EEG called EEG topography, which allowed for the mapping of electrical activity across the surface of the brain. This enjoyed a brief period of popularity in the 1980s and seemed especially promising for psychiatry. It was never accepted by neurologists and remains primarily a research tool.