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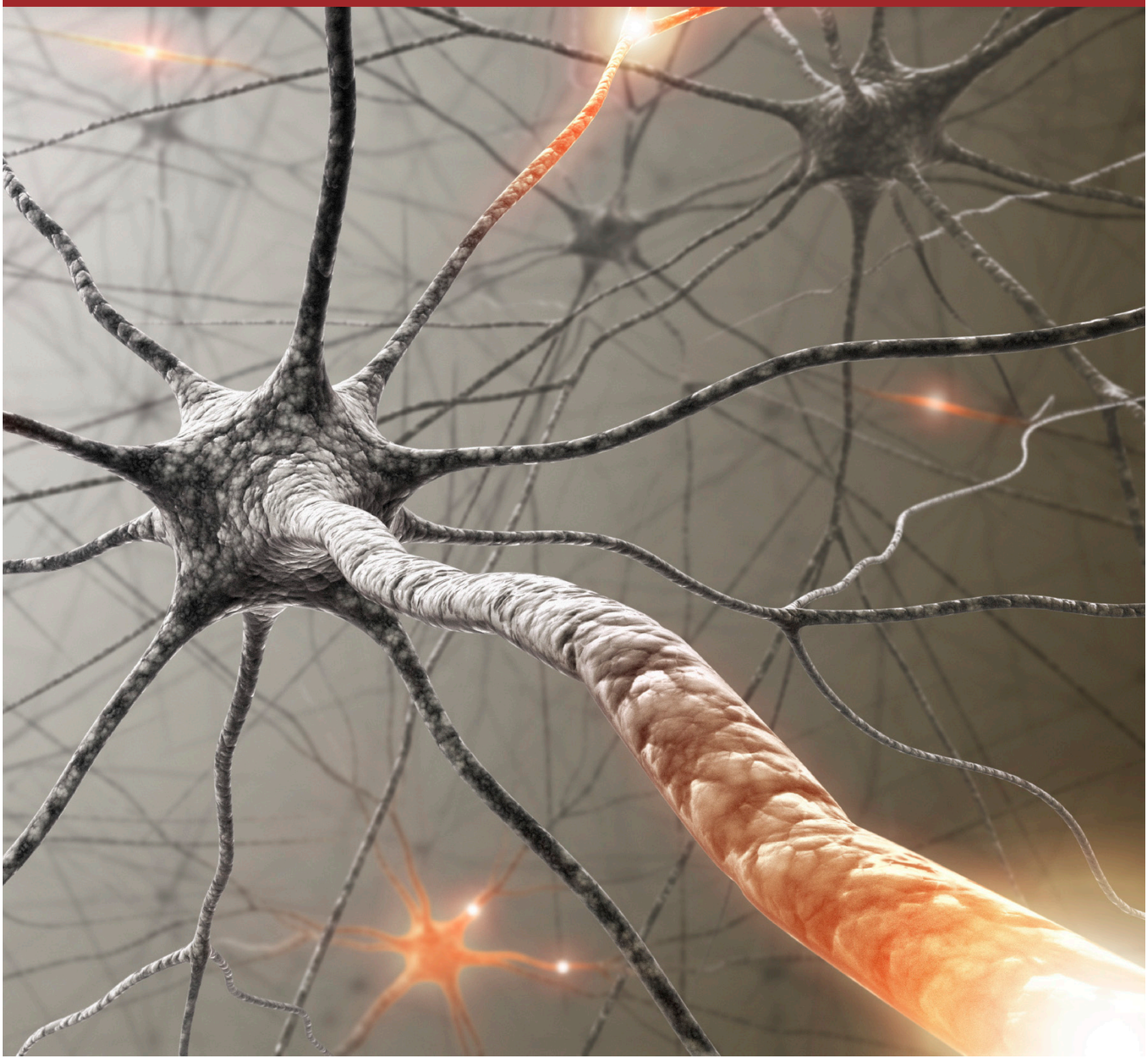
FOUNDATION YEARS JOURNAL

MAY 2011

Volume 5, Issue 4: **Neurology**



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Foundation Years Journal

This journal is an international peer-viewed journal which seeks to be the pre-eminent journal in the field of patient safety and clinical practice for Foundation Years' doctors and educators. The Journal welcomes papers on any aspect of health care and medical education which will be of benefit to doctors in the Foundation training grade in the UK or international equivalents.

Editor In-Chief

Issue Editor

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Volume 5, Issue 4: Neurology

Foundation Years Journal is the ONLY journal for Foundation Years doctors and educators, specifically written according to the MMC curriculum. It focuses on one or two medical specialties per month, each issue delivers practical and informative articles tailored to the needs of junior doctors. The Journal closely follows the Foundation Years syllabus to provide the best educational value for junior doctors. In addition to good clinical and acute care articles, assessment questions give junior doctors the chance to gauge their learning. The answers will be published in the next issue, but 123Doc will advance answers to clinical tutor subscribers so they can engage their students in the learning process. Each issue provides comprehensive clinical cases for trainees as well as practical teaching assessments for educators. Readers will benefit from:

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Editorial For Neurology Issue Of Foundation Years Journal 2011

Many persons like to hold a book or journal in the hand. The ability to browse by turning pages, for those to annotate who are prepared to deface paper copies to read without needing to find electronic apparatus to enable viewing (whether by computer, by Kindle device or otherwise), all are powerful stimuli to keep to conventional hard copy, paper publications. The feel of a book, the smell of the paper (maybe the binding), the colourful printing and the variations in font and style all contribute to this sensual experience. However, paper copies become dated and cannot easily be amended except in loose-leaf form where they lose much of their aesthetic appeal. They are more expensive to produce at the point of the user. They decay with use, whether aided by fingers, thumbs or by mice, and they are bulky for publishers and readers to transportance, this trend towards electronic publishing. Electronic journals have many advantages and can be accessed from computers worldwide. This journal offers all of these advantages and on this occasion brings to readers aspects of important neurological topics relevant to Foundation Years practitioners.

FOUNDATION YEARS JOURNAL 2011

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The neurosciences, of which everyday clinical neurology forms a part have made miraculous progress over the last couple of decades. The interactions between laboratory and clinical research and with clinical medicine that deals with illness in patients at its most elementary level, have contributed to these advances. However, sometimes research and cutting edge thinking from the laboratory is difficult to apply to some of the immediate clinical problems exhibited by patients. Common sense (whatever that is) and thinking is needed with acute problems and so is rapid decision making. Some of the topics covered in this issue deal with acute medicine, and neurology is now very much part of this since nearly one-fifth of those admitted acutely has neurological problems, and others with less acute matters still get admitted to hospital. Papers published here express some of the most important points that Foundation Years doctors experience during their everyday duties, lessons they wish to share with others in order to help prevent mishaps.

Indeed, such practitioners are encouraged to submit to this journal. There is so much to be learned from our everyday activities and our patients are in many ways our best teachers, using their symptoms and signs to make us think. It is in many ways a moral imperative to share this information with others and to publish for the widest circulation. Specific lessons that may be drawn from the papers in this issue of the Foundation Years Journal include epilepsy and the causes of blackouts together with some useful tips on the use of the EEG in diagnosis, an important supportive test in some patients. Stroke is now an emergency in more ways than previously (since more can be done), a brain attack that needs handling acutely and which can result from venous sinus thrombosis, two more areas covered in this journal. The techniques of lumbar puncture are still important although much that was investigated previously by this technique now is revealed by the increasingly complicated imaging processes that have become available.

Acute neuromuscular weakness is a further presenting feature that has many causes and this condition may be quite puzzling in many patients. Increasingly complicated drugs and drug regimes may lead to toxicity, an important cause of disability that can easily be overlooked; baclofen is a useful drug for spasticity and intoxication is disabling. Trigeminal neuralgia can be treated in many ways; not all being effective and a paper on this topic should help guide those who deal with its early manifestations.

And what of that imperative to publish? Here we are guided in the values of clinic letters and of the role of the doctor as educator. All very important stuff, hopefully interesting, certainly enlightening, and without doubt we hope a stimulus for readers to provide further papers dealing with the many topics in neurology that may perplex all of us including those working in the Foundation Years.

Christopher Gardner-Thorpe
MD, FRCP, FACP, Consultant Neurologist
Editor, Journal of Medical Biography, Royal Society of Medicine
Exeter, UK
cgardnerthorpe@doctors.org.uk

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CEREBRAL VENOUS SINUS THROMBOSIS

Katharine Warburton, Victoria Stokes

Cerebral Venous Sinus Thrombosis.
Patient Management.

Abstract

Headache is a very common complaint encountered by junior doctors. Cerebral venous sinus thrombosis is an important pathology to consider as early recognition and prompt treatment improve outcome. The case described here illustrates the importance of a thorough history and examination, with further discussion around investigation and management.

Case Study

Presentation

Miss M, a 19 year old lady, presented with a 2-week history of constant severe occipital headache made worse by coughing and lying flat. This was initially associated with vomiting. Five days prior to admission, she developed blurred vision and photophobia. There was no history of neck stiffness or fever. Previously she had been well, but had mild asthma for which she took of inhalers. She had been taking the combined oral contraceptive pill (COCP) for the last year. She was a smoker of 10 cigarettes a day. Family history revealed a maternal grandmother who had suffered from a DVT and PE .

On examination, she was found to have bilateral papilledema. Visual acuity was 6/9 left, 6/15 right. She had a mild left lateral rectus palsy. The rest of the cranial nerve, limb examination and systemic examinations were normal.

Investigations

Blood tests were unremarkable with normal inflammatory markers. After her initial management, including anticoagulation, a thrombophilia screen was performed. This showed a low protein S level of 23 u/dL (normal range 50–134). This may indicate an underlying thrombophilia, is discussed later.

A lumbar puncture demonstrated an opening pressure of 40 cm of water and normal cerebrospinal fluid (CSF) constituents. CT brain was normal, but MRI brain demonstrated a sagittal sinus thrombosis with extension to the right transverse sinus.

Management

She was anticoagulated with warfarin and low-molecular weight heparin until INR was therapeutic at target 2.5. Her headache and nausea were controlled with opiates and an anti-emetic. She was advised to stop the COCP and to stop smoking. Her symptoms settled over 2 weeks, and she was discharged on warfarin. Miss M was followed up by neurologists with repeat imaging to check for resolution of the thrombosis. The haemotologists further investigated her abnormal thrombophilia screen.

Discussion

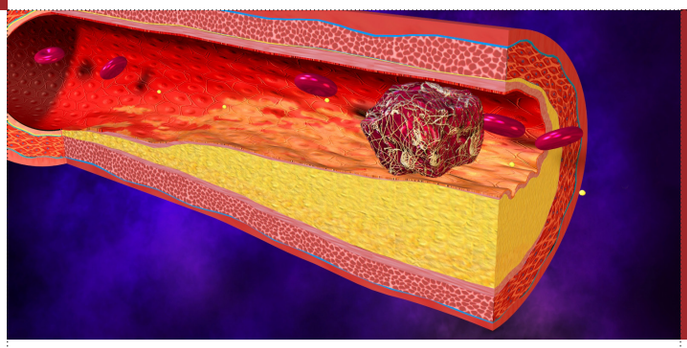
Cerebral venous sinus thrombosis is simply a blood clot that is formed in the dural venous sinuses that drain blood from the brain. The resulting "back pressure" on the cerebral circulation can cause small haemorrhages, venous infarcts and raised intracranial pressure (ICP).

Clinical assessment of patients with sinus thrombosis should begin with a thorough history and examination. Headaches are a common presenting complaint and are usually benign. However, there were some concerning features to Miss M's headache that should have prompted further investigation.

When a patient with a headache complains of vomiting and photophobia, then intracranial infections such as meningitis or encephalitis should always be considered. Miss M's headache was also exacerbated by lying flat and coughing. This is characteristic of raised ICP where vomiting and visual disturbance are also common features.

CEREBRAL VENOUS SINUS THROMBOSIS

Katharine Warburton, Victoria Stokes



Miss M had several risk factors for cerebral venous sinus thrombosis. She was on the COCP and had a positive family history of venous thrombosis. A thorough history will elicit these factors and give clues towards diagnosis.

On examination, Miss M had bilateral papilloedema and a left lateral rectus palsy. Papilloedema indicates raised ICP. A left lateral rectus palsy can be caused by a discrete lesion along the course of the left abducens nerve or may be a "false localising sign". In addition to signs of raised ICP, patients may have true focal neurology from a venous infarct or haemorrhage. They may also become encephalopathic which may range from a disturbance in consciousness to coma.

Differential diagnosis

It was clear from the history and examination that Miss M had raised ICP. Possible causes of this include a space occupying lesion, intracranial haemorrhage, idiopathic intracranial hypertension and cerebral venous sinus thrombosis. Her risk factors made venous sinus thrombosis the most likely in this list.

Investigations

In the first instance, the cause of the raised ICP should be sought, with neuroimaging performed before lumbar puncture. This is due to the small risk that the patient will have non-communicating hydrocephalus and will cone from the pressure shift as CSF is drained.

Miss M's initial CT was normal. This is the case in up to 30% of sinus thrombosis patients. However, it is a useful screen for other causes of headache. MRI with MR venography is the most sensitive technique and was able to detect the thrombus and occluded sinus.[1]

Lumbar puncture may be normal or show non-specific abnormalities. However, a normal CSF examination can be helpful to exclude other differentials such as meningitis and subarachnoid haemorrhage. A raised opening pressure supports the clinical suspicion of raised ICP, and draining CSF will relieve this, but only temporarily.

Cerebral Venous Sinus Thrombosis. Patient Management.

Once the diagnosis of cerebral venous sinus thrombosis is made, then the patient should be investigated for underlying causes. Even with extensive investigation, no cause will be found in around a quarter of patients.[2] Any of the following may predispose a patient to develop cerebral sinus thrombosis:

- Oral contraceptive pill or hormonal replacement therapy
- Pregnancy and the puerperium
- Malignancy
- Thrombophilia, e.g., factor V Leiden deficiency
- Infections, either local such as meningitis or infections of the ear, nose and throat, or generalised sepsis
- Head injury and mechanical precipitants
- Blood disorders, eg polycythaemia rubra vera
- Chronic inflammatory diseases, e.g., inflammatory bowel disease
- Nephrotic syndrome

Miss M was on the COCP and had a family history of venous thrombosis. Her thrombophilia screen revealed a low free protein S level, which may indicate that she has a thrombophilic condition. However, it is important to perform thrombophilia screens before anticoagulation and interpret the results with caution. Free protein S can be transiently reduced when clotting occurs and also with anticoagulation. Miss M's test was performed after anticoagulation and during her period of illness, making these results hard to interpret.

Treatment

Anticoagulation is the mainstay of treatment. This is to attempt to recanalise the sinus to prevent the thrombus from propagating and where relevant to treat the underlying prothrombotic state. Patients should be anticoagulated with heparin initially and will require long-term anticoagulation with warfarin. The duration of treatment will depend on any underlying cause found. Miss M was warfarinised with a target INR of 2.5 for 6 months.[3]

CEREBRAL VENOUS SINUS THROMBOSIS

Katharine Warburton, Victoria Stokes

Some patients may worsen despite anticoagulation. In these circumstances endovascular thrombolysis can be used. This is where a thrombolytic substance is delivered within the occluded sinus by an intraducing catheter. It is also sometimes necessary to insert a shunt to relieve the raised ICP.

Clinical judgement

In this case, Miss M was initially diagnosed with migraine due to the presence of nausea and photophobia. As previously discussed, there were "red flags" to her headache that may have prompted earlier investigation with a more careful history and examination.

Learning points

• Take a careful history in a patient with headache to look for "red flag" signs.

• Be alert to risk factors for cerebral venous sinus thrombosis.

• Always perform a full examination in headache, including fundoscopy and cranial nerve examination.

• Early recognition of cerebral venous sinus thrombosis improves outcome.

Best of two MCQs

MCQ number 1

A 26-year old lady presents to A+E complaining of a headache for about a week. She says she has vomited every morning for the past 5 days and that her eyes have been "going funny" every time she bends down to put on her shoes. The doctor notices that she frequently attends A+E with minor problems, such as pelvic pain, low mood and most recently ear ache.

The most important diagnosis to consider is:

a. Depression
b. Pregnancy
c. Otitis interna
d. Cerebral venous sinus thrombosis
e. Drug misuse

Answer d.

This lady gives a history that is suspicious of raised ICP. Bending over would cause a transient increase in the already raised ICP, further compressing the optic nerve and causing visual disturbance. Vomiting in the morning is characteristic of raised ICP following a period of lying flat. Her recent earache could have been a localised infection predisposing her to cerebral venous sinus thrombosis. (see causes above). It is important not to immediately dismiss patients with frequent minor ailments.

MCQ number 2

A 31-year old lady presents to A+E following a first ever seizure. She is drowsy and confused post-ictally. A CT of brain shows petechial haemorrhages in the right hemisphere. An MRI shows cerebral venous sinus thrombosis. CSF examination is normal.

The best initial treatment would be:

a. Aspirin
b. Vitamin K
c. Antibiotics
d. Supportive management
e. Heparin

Answer e.

The underlying problem is a thrombosis, and the petechial haemorrhages are caused by venous outflow blockage. It is therefore important that anticoagulation is started immediately to relieve the cause. Heparin is the most appropriate anticoagulant to use.

References

1. Lafitte F, Boukobza M, Guichard JP, et al (1997). MRI and MRA for diagnosis and follow-up of cerebral venous thrombosis (CVT). Clin Radiol 52:672.
2. Ameri A, Bousser MG (1992). Cerebral venous sinus thrombosis. Neurol Clin 10:87–111.
3. Einhaupl K, Bousser MG, de Bruijn SF, et al (2006). EFNS guideline on the treatment of cerebral venous and sinus thrombosis. Eur J Neurol 13(6):553–9.

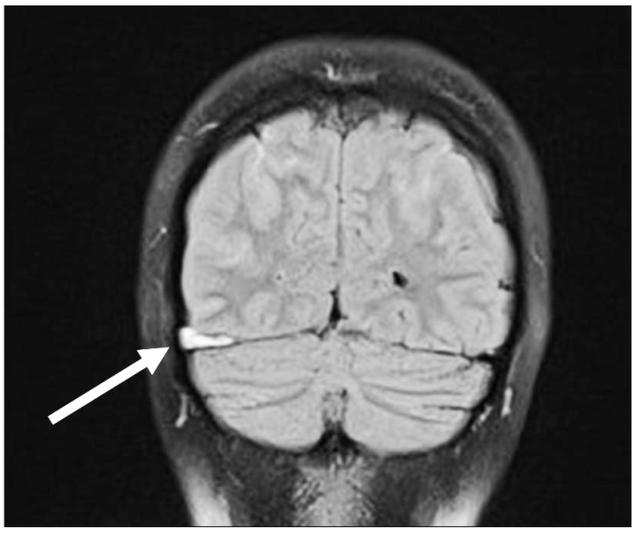


Figure 1:
Coronal MRI showing presence of thrombus in the right transverse sinus.

Authors

1. Dr Katharine Warburton, Year 1 Core Medical Trainee (Neurology), Oxford Radcliffe Hospitals. Email: k.warburton@doctors.org.uk
2. Dr Victoria Stokes, Year 2 Core Medical Trainee (Neurology), Oxford Radcliffe Hospitals. Email: vstokes@doctors.org.uk

CHILDHOOD EPILEPSY IN THE UK - A REVIEW

Erica Tirr, Neel Halder

Childhood Epilepsy in the UK - a review.
Erica Tirr, Neel Halder.

Abstract
One of the most discussed, debated, and studied topics of paediatric neurology is that of epilepsy. It is a challenge to diagnose, treat, and prevent the complications of this condition and much research has gone into improving the outcome of the lives of those affected by different forms of epilepsy.

This review aims to broadly cover the majority of the aspects of childhood epilepsy and to develop an understanding of the difficulties one faces when dealing with these complex neurological conditions. It will be particularly useful for those new to the field, or those who would like a general overview of relevant information in one easy-to-read place.

Introduction
Neurodevelopmental paediatrics is an extremely interesting and complex field within the medical specialties. Paroxysmal events and disorders such as epilepsy have had extremely severe reactions from the general public for centuries. It used to be viewed as a manifestation of demonic Gods, and those who had epilepsy were locked up in quarantine or confined to psychiatric hospitals [1]. People around these patients thought that if they were to touch them, they too would be cursed, thus over time, epilepsy has accumulated a rather heavy social stigma. With advances in the understanding of neurophysiology, and the development of diagnostic machines such as electroencephalograms (EEGs), we have been able to determine the causes, effects, and treatments for epilepsy. Also, the population's awareness of this type of disorder has much increased and fortunately the negative connotations associated with it are disappearing.

The origin of the word "epilepsy" derives from the ancient Greek verb meaning "to seize" or "to attack". Today, we define epilepsy as a recurrent clinical phenomenon resulting from abnormal and excessive excitability of the neurones of the cerebral hemispheres. An epileptic seizure is an intermittent, paroxysmal disturbance of consciousness, behaviour, emotion, motor function, observation, or sensation resulting from cortical neuronal discharge [2].

Aetiology / Causes
Although epilepsy is generally characterised by a series of unprovoked seizures, it is important to mention that a seizure may occur in non-epileptic patients subjected to either direct or systemic cerebral insults (see table 1):

• Fever	⟨ Renal failure
• Hypoxia	⟨ Hepatic failure
• Hypoglycaemia	⟨ Porphyria
• Hypocalcaemia	⟨ Drugs (e.g. Solvents)
• Electrolyte imbalance	⟨ Drug withdrawal
• Inborn errors of metabolism	⟨ Toxins
	⟨ Trauma

Table 1. Cerebral and systemic insults that may result in epileptic seizures.

In the UK, childhood epilepsy is most commonly idiopathic, but can also be caused by malformations of cortical development, cerebral vascular occlusion, hippocampal sclerosis, or small foreign-tissue lesions (vascular malformations, hamartomata and low-grade gliomas).

Also, hypoxia in-utero, intraventricular haemorrhage/ischaemia, hydrocephalus, neurocutaneous syndromes, and in other parts of the world, infectious causes are ordinary. There are also proven genetic links or 'epilepsy susceptibility genes' that predispose a child to developing epilepsy: different mutations in genes that control the excitability of neurons have been described in idiopathic childhood epilepsies. Most commonly, sodium/potassium channelopathies and GABA-receptor mutations are involved. It now is becoming clear that mutations should not only be looked for in familial cases, but also in sporadic cases, especially in infants and young children with unexplained severe epileptic encephalopathies [3].

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Epidemiology

Epilepsy is around twice as common in children as in adults (700 per 100,000 in children under the age of 16 years compared to 330 per 100,000 in adults) [4]. The incidence is approximately 23-190/100,000 per year and about 1/200 children have active epilepsy. Almost one-half of individuals who develop epilepsy do so before the age of 15 years. The prevalence is slightly higher in males and in those in lower socioeconomic groups [5]. Although 5% of the world's population may have a seizure at any time during their life, only one third of these people will develop epilepsy. Thus epilepsy is only diagnosed when an individual has the tendency to experience recurrent seizures; at least two, unprovoked, more than 24 hours apart as stated by The World Health Organization (WHO). Globally about 50 million people are affected with epilepsy [6].

Differential Diagnoses

The basis for the recognition and diagnosis of epileptic seizures is almost entirely dependant on the history given about the paroxysmal event. This is because examination will often be normal, and the results of investigations can only be correctly interpreted with reference to the history. An accurate account from an eyewitness is essential in this situation, especially when dealing with young children [7]. An incorrect diagnosis of epilepsy established as a child could potentially result in a lifetime of unnecessary medications, side-effects, medical follow-up, investigations, social restrictions, and difficulties for the families of those affected.

Therefore, it is important to thoroughly investigate a child with a seizure (or seizures), and rule out any other differential diagnoses.

One of the most common conditions mis-diagnosed, as epilepsy is febrile seizures. These occur in 3-5% of children between the ages of 6 months and 5 years; they present early in a viral infection when temperature is rising rapidly (commonly an upper respiratory tract infection). The seizures are usually brief, generalised tonic-clonic seizures (involving muscle contractions and relaxations). Although simple febrile seizures can be very distressing for parents, they are benign and do not cause brain damage or intellectual decline. They carry only a 1 – 2% chance of developing epilepsy in later life (similar to the risk for all children). Complex febrile seizures (such as those that are prolonged, focal or repeated in the same illness) have a slightly increased risk of subsequent epilepsy: 4 – 12%.

Another common pitfall is that of a syncope leading to an anoxic tonic-clonic seizure. One such example is that of breath-holding attacks. These occur in toddlers when they are upset or angry; the child cries, holds his breath, and goes blue. Sometimes children will briefly lose consciousness but rapidly recover fully. Attacks resolve spontaneously and drug therapy is unhelpful. Reflex anoxic seizures are similar to breath-holding attacks, except that they are precipitated by pain or discomfort, particularly from minor head trauma, cold food or fright. The child becomes very pale and falls to the floor. The hypoxia may induce tonic-clonic seizure. These episodes are due to cardiac asystole from vagal inhibition (and may be reproducible on EEG by ocular compression). Once again the child rapidly recovers.

Other differentials include benign paroxysmal vertigo (thought to be due to viral labyrinthitis), cardiac arrhythmia, tics, daydreaming, night terrors, choreoathetosis, pseudoseizures or fabricated seizures (which are psychological, feigned seizures), and self-gratification [8].

It can be very difficult to separate all these events from epileptic paroxysmal disorders, especially if we rely solely on a history. It is therefore extremely helpful if an eye-witness can video-tape the event or if a particular stimulus is found, so that it can be reproduced in a controlled environment, with an EEG in place.

Classification of Epilepsy

Epileptic seizures are classified into either generalised or focal (partial) seizures. In generalised seizures, the discharge arises from both hemispheres, whereas in partial seizures the discharge arises from one, or part of one hemisphere. Partial seizures are further subdivided into "simple", in which consciousness is retained, and "complex", in which consciousness is impaired or lost. Partial seizures may become secondarily generalised, resulting in tonic-clonic convulsion.

The manifestations of focal seizures depend on the area of the brain where the discharge originates, e.g. temporal lobe seizures may result in auras with smell or taste abnormalities and distortions of sound and shape. Generalised seizures may be:

- **Absence**
- **Myoclonic**
- **Tonic**
- **Tonic-clonic**
- **Atonic**

Absence seizures	Transient loss of consciousness, with an abrupt onset and termination, unaccompanied by motor phenomena
Myoclonic seizures	Brief, repetitive, jerking movements of the limbs, neck or trunk
Tonic Seizures	Generalised increase in tone
Tonic-clonic seizures	Rhythmical contraction of muscle groups following the tonic phase. The limbs jerk, breathing is irregular, cyanosis develops, there may be biting of the tongue and incontinence of urine. After the seizure, unconsciousness, deep sleep or drowsiness may occur.
Atonic	Often combined with a myoclonic jerk followed by a transient loss of muscle tone causing a sudden fall to the floor or drop of the head

Fig.1: Generalised epilepsies [9].

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In addition to these broad categories for the classification of epilepsy, there are some specific syndromes related to seizure pattern to consider. In the generalised seizure category, the commonest are:

· West syndrome (Infantile spasms) occurs between 5 – 12 months of age. Presents as violent spasms of the head, trunk and limbs and a sudden bending forward and stiffening of the body followed by an extension of the arms (salaam spasms). Spasms tend to begin soon after arousal from sleep.

On EEG, abnormal, chaotic brain wave patterns are shown. Typically mental retardation develops [10].

· Lennox-Gastaut syndrome occurs in 1 – 3 year olds (although there are reports of a much later presentation). It is characterised by astatic seizures, tonic seizures, and atypical absences. The prognosis is rather poor: neurodevelopmental arrest, regression, and behaviour disorders often accompany the seizures.

· Typical (petit mal) absence seizures occur later in childhood, usually between ages of 4 and 12 years. The child stares momentarily and stops moving, there may be some hand or eyelid twitching. This lasts only a few seconds and the child has no recall except he/she may realise they have missed something on regaining full awareness. These seizures may interfere with schooling however the majority of cases remain developmentally normal and 95% of them attain remission in adolescence.

· Juvenile myoclonic epilepsy occurs in adolescence or early adulthood. The seizures may be myoclonic only or may also be absence or tonic-clonic in nature. These are most frequent on waking and a typical history is throwing drinks or cornflakes about in the morning. There is a characteristic EEG pattern associated with juvenile myoclonic seizures and a genetic link has been identified. Response to treatment with certain drugs (e.g, sodium valproate) is good and learning remains unimpaired [11].

Within the focal epilepsies, two examples of well-described, documented, and discussed syndromes are:

· Benign rolandic epilepsy / BCECTS is an inherited disorder-affecting children in a 4 – 10 year age range, where tonic-clonic seizures take place in sleep, or when simple partial seizures with awareness of abnormal feelings in the tongue and distortion of the face (supplied by the rolandic area of the brain) occur.

· Benign occipital epilepsy presents with autonomic features such as periods of unresponsiveness, eye deviation, and vomiting. In older children, it presents with headaches, visual image distortion, and hallucinations. It is an uncommon condition and usually has a very good outcome.

Idiopathic with age-related onset	• Benign neonatal familial convulsions • Benign neonatal convulsions • Benign myoclonic epilepsy in infancy • Childhood absence epilepsy • Juvenile myoclonic epilepsy • Epilepsy with generalized tonic-clonic seizures on waking
Symptomatic and cryptogenic	• West syndrome • Lennox-Gastaut syndrome • Epilepsy with myoclonic-astatic seizures • Epilepsy with myoclonic absences
Symptomatic	• Early myoclonic encephalopathy • Early infantile myoclonic encephalopathy with burst suppression • Others
Idiopathic with age-related onset	• Benign childhood epilepsy with centrotemporal spikes (BCECTS) – also known as Benign rolandic epilepsy • Childhood epilepsy with occipital paroxysms • Primary reading epilepsy
Symptomatic	• Epilepsy with simple partial, complex partial, or secondarily generalized seizures arising from the frontal, parietal, temporal, or occipital lobe or from multiple lobes, or of unknown lobe of onset • Epilepsia partialis continua • Syndromes characterized by specific modes of activation
	• Severe myoclonic epilepsy in infancy • Epilepsy with continuous spike-and-wave activity in sleep • Acquired epileptic aphasia
Generalised Epilepsies & Syndromes	
Focal Epilepsies & Syndromes	
Epilepsies & Syndromes Undetermined (focal or generalized)	

Table 2: List of focal and generalised epilepsies.

CHILDHOOD EPILEPSY IN THE UK - A REVIEW

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Associated Comorbidity

Comorbid conditions are common in children with epilepsy; some may even overshadow the epilepsy in their symptomatic effects on the child. These conditions may significantly affect epilepsy and its treatment; similarly, antiepileptic drugs (AEDs) may affect the associated conditions. Therefore it is important to consider each individual separately and try to find a balance in treatment that best suits the child and his/her parents [13].

The main comorbidities associated with epilepsy are cognitive or psychiatric in nature, which poses some difficulties with academic learning and performance, and also with social interaction and behaviour. There are different hypotheses as to why these specific difficulties arise in epileptic children and there are still many un-answered questions on this subject. For instance, is there a learning difficulty to begin with that originates from an insult to the same area of the brain as the one generating seizures, or is it the fact that a child has epilepsy first that then impacts on his/her development? Or why do seizures and learning difficulties go hand in hand in some children while others experience seizures but no cognitive decline? To what extent are antiepileptic drugs to blame for cognitive and behavioural problems? All these questions have been studied and discussed with no real successful answers – all that is certain is that children with epilepsy are at a significantly higher risk of developmental disorders than others of the same age that are seizure-free.

The studies examining the intricate relationship between epilepsy, language, behaviour, and cognition has yielded a list of specific disorders that are commonly seen in children with epilepsy.

- **Autistic Spectrum Disorder (ASD):** refers to a variety of different developmental disorders characterised by impaired socialisation and language skills, a restricted repertoire of interests and activities which has an onset in early childhood. Cognition may range from severe mental retardation to superior intelligence. The aetiology of ASD includes causes such as tuberous sclerosis, fragile X syndrome, and congenital infections e.g. rubella.

- **Verbal Auditory Agnosia (VAA):** defined as a severe receptive and expressive language disorder believed to arise from inadequate auditory or phonologic processing that engages activity in primary or secondary auditory cortices (one of the disorders that make up LKS; see below).

- **Autistic Regression:** when children undergo a regression in language, sociability, or behaviour, following a period of normal development or superimposed on already aberrant development (contrasted with children that present with disability from infancy).

- **Attention Deficit and Hyperactivity Disorder (ADHD):** characterised by excessive activity and learning impairment, which often results in disruptive, or aggressive behaviour.

- **Childhood Disintegrative Disorder (CDD) / Heller's Disease:** presents as regression in language, behaviour and cognition in a previously normally developing child, occurring between 2 and 10 years of age. The long-term prognosis is poorer than in children with ASDs. Paroxysmal EEG abnormalities have been reported in up to one half of children with CDD.

- **Landau Kleffner Syndrome (LKS) / Acquired Epileptic Aphasia:** a disorder that is characterised by an acute deterioration in language skills in children with previously normal expressive and receptive language development in association with seizures or epileptiform abnormalities on EEG. It is more common in boys than girls and presents in the preschool years. The most frequent language disorder in LKS is VAA, but pure expressive dysphasias, and ADHD-type behaviour have also been reported.

- **Electrical Status Epilepticus in Sleep (ESES):** this is an EEG diagnosis in which an epileptiform EEG is most often associated with a behavioural disorder (often psychotic) and global developmental regression. The majority of children with ESES have seizures [14].

- **Depression/Anxiety/Psychosis/Alcohol Dependence:** are the commonest psychiatric comorbidities [15].

Treatment

The treatment of epilepsy has undergone update after update with new and improved antiepileptic drugs on the market to choose from. The key treatment goals remain the same however: achieve seizure control and optimal physical and cognitive function using the simplest possible AED regimen. Apart from medical therapy, there have also been technological advances in this field such as vagus nerve stimulation implants; and also dietary measures such as the Ketogenic diet; surgical treatment also remains an effective alternative in certain types of epilepsies.

Status Epilepticus & Emergency Treatment

Status Epilepticus (SE) is the most common medical neurological emergency and continues to be associated with significant morbidity and mortality. It is defined as a generalised tonic-clonic convulsion lasting 30 minutes or longer, or repeated tonic-clonic convulsions occurring over a 30 minutes period without recovery of consciousness between each convulsion. Estimated incidence is between 10-60 cases/100,000 per year. It recurs in over 13% of patients.

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Table 3 provides guidelines for the management of SE from the UK's The National Institute of Clinical Excellence.

Premonitory stage (pre-hospital) *At home: drugs administered by a healthcare professional or by a trained family member / carer.*	• Diazepam 10□20 mg given rectally, repeated once 15 minutes later if status continues to threaten, or • Midazolam 10 mg given buccally
Early status	• Lorazepam (IV) 0.1 mg/kg (usually a 4 mg bolus, repeated once after 10□20 minutes) • Give usual AED medication if already on treatments
Established status	• Phenytoin infusion at a dose of 15-18 mg/kg at a rate of 50 mg/minute or • Fosphenytoin infusion at a dose of 15□20 mg phenytoin equivalents (PE)/kg at a rate of 50-100 mg PE/minute and/or • Phenobarbitone bolus of 10-15 mg/kg at a rate of 100 mg/minute
Refractory status*	General anaesthesia, with one of: • Propofol (1-2mg/kg bolus, then 2-10mg/kg/hour) titrated to effect. • Midazolam (0.1-0.2mg/kg bolus, then 0.05-0.5mg/kg/hour) titrated to effect. • Thiopentone (3-5mg/kg bolus, then 3-5mg/kg/hour) titrated to effect; after 2-3 days infusion rate needs reduction as fat stores are saturated. Anaesthetic continued for 12□24 hours after the last clinical or electrographic seizure, then dose tapered

Table 3. Emergency AED therapy for convulsive status epilepticus [16]

Medical (Non-Emergency) Treatment

Antiepileptic drugs or AEDs generally act upon neuronal channels that facilitate seizures. They block these transmembranal ion pumps and can therefore lower a seizure threshold by reducing the electrical potential. Gamma-aminobutyric acid (GABA) neurones are involved in the inhibition of electrical activity and there is evidence that they are reduced in epilepsy; so some AEDs are designed to stimulate GABA (see table 4).

The newer (second line) antiepileptic drugs gabapentin, lamotrigine, oxcarbazepine, tiagabine, topiramate, and vigabatrin (as an adjunctive therapy for partial seizures), are recommended for the management of epilepsy in children who have not benefited from treatment with the older (first line) antiepileptic drugs such as carbamazepine, phenytoin or sodium valproate, or for whom the older antiepileptic drugs are unsuitable because

· there are contraindications to the drugs.
· they could interact with other drugs the child is taking (notably oral contraceptives).
· they are already known to be poorly tolerated by the child in terms of side-effects.
· the child is currently of childbearing potential or is likely to need treatment into her childbearing years.

Drug name	Type of epilepsy drug is used for	Mechanism of action	Side-effects
Sodium Valproate	All forms	Blockade of neuronal transmembrane sodium channels	{ GI upset { Weight gain { Hair loss { Tremor { Thrombocytopenia { Hepatotoxicity
Carbamazepine/ oxcarbazepine	All forms except myoclonic or absence seizures	Blockade of neuronal transmembrane sodium channels	{ Nausea/vomiting { Double vision, dizziness, drowsiness, ataxia { Leucopenia { Hyponatraemia (*These are less frequent with oxcarbazepine.*)
Phenytoin/fosphenytoin	All forms except absences	Blockade of neuronal transmembrane sodium channels	{ Cerebellar function and co-ordination disturbance { Gum hyperplasia { Hirsuitism { Skin rashes, acne { Folate deficiency
Ethosuximide	Absence seizures	T-type calcium channel inhibition	{ Nausea, vomiting, anorexia { Teratogenicity { Drowsiness and headaches
Phenobarbital	All forms	Prolongation of opening time of postsynaptic neuronal chloride channels + potentiation of GABA effects	{ Sedation, fatigue, poor memory, depression { Hyperactivity, aggression { Nausea, vomiting { Withdrawal effects
Vigabatrin	Only used for refractory epilepsy as an adjunct	Inhibition of GABA transaminase which inactivates GABA	{ Sedation, fatigue { Psychosis { Peripheral visual field defects { Weight gain
Lamotrigine	Wide spectrum of efficacy for partial and generalised seizures	Inhibition of neuronal sodium currents	{ Nausea, vomiting { Skin rashes { Headache, dizziness, double vision, ataxia, tremor
Gabapentin	Partial seizures	GABA effect enhancement	{ Drowsiness, dizziness, ataxia, fatigue, diplopia
Topiramate	Add-on treatment for refractory partial or generalised seizures.	Sodium channel blocker	{ Cognitive difficulties, tremor, dizziness, ataxia, headache { GI upset
Tiagabine	Adjunctive therapy for partial seizures	Decreases neuronal and astrocytic uptake of GABA	◆ Dizziness ◆ Lethargy, somnolence
Benzodiazepines (clonazepam, diazepam etc)	Prophylaxis and control of seizures	GABA enhancement	◆ GI disturbance ◆ Drowsiness, headache ◆ Depression, confusion

Table 4. AED effects and side effects [21].

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The children's British National Formulary (BNF) and the NICE guidelines recommend that children should be treated with a single antiepileptic drug (monotherapy) wherever possible. If the initial treatment is unsuccessful, then another drug can be tried, rather than adding to the previous one (caution is needed during the changeover period).

Combination therapy (adjunctive or 'add-on' therapy) should only be considered when attempts at monotherapy with antiepileptic drugs have not resulted in seizure freedom. If combination therapy does not bring about worthwhile benefits, treatment should revert to the regimen (monotherapy or combination therapy) that has proved most acceptable to the child, in terms of the balance between effectiveness in reducing seizure frequency and tolerability of side effects.

Side Effects of Medical Treatment

Because of the harmful effects most of these drugs will eventually have when taken long-term, much debate has taken place amongst paediatric neurologists as to if and when a child should be gradually taken off their medication for a trial period. Some argue that if an epileptic child is weaned off AEDs too soon, they are at risk of developing intractable seizures [17]; and others believe that with the arrival of the new drugs with less and less side-effects, children are at much less risk in taking them [18].

Others still believe that these drugs are more toxic than therapeutic, or that they are not adequate for their patients and that the sooner they can be rid of them, the better [19]. However the general consensus in the literature reviewed was that children with epilepsy should be reviewed regularly but not necessarily weaned off AEDs after a previously standardised 2 years of being seizure-free [20]. Some paediatric neurologists make it a practice to review a child's medical treatment when they are aged 14, two years before they leave school as this is a good time to discuss the pros and cons to continuing AEDs, the social needs of the patient as they become adolescents, and perhaps to approach the topic of adherence to treatment and review appointments.

Drug Interactions

Since many AEDs affect drug-metabolising enzymes in the liver, interactions are frequent. They have major clinical implications for seizure control or toxicity when 2 or more AEDs are used together. Plasma drug concentration monitoring is often advisable in these cases.

- Sodium Valproate inhibits hepatic drug metabolism, often increasing concentrations of phenobarbital, lamotrigine and carbamazepine and reducing plasma phenytoin concentration.
- Carbamazepine induces hepatic enzymes and therefore lowers concentrations of clonazepam, lamotrigine, tiagabine, topiramate, sodium valproate and oxcarbazepine. Also, a very common interaction is with the oral contraceptive pill and doses of oestrogen should be adjusted accordingly to avoid failure of contraception.
- Phenytoin, Phenobarbital same interactions as carbamazepine.
- Vigabatrin reduces phenytoin concentration by unknown mechanisms.

Ketogenic diet

The ketogenic diet is a high fat, adequate protein, low carbohydrate diet designed to mimic many of the biochemical changes associated with prolonged fasting. During starvation, the body first uses its store of glucose and glycogen, and then begins to burn the stored body fat. When there is not sufficient glucose available, the fats cannot be completely burned and ketone bodies are left, as the residue of incompletely burned fat. The ketogenic diet provides exogenous fats for the body to burn, but limits the available carbohydrate so that ketone bodies build up. It is the high level of these ketones, which appear to suppress seizures. First developed in the early 1900s, and successfully used for the treatment of seizures in children during the 1920s and 1930s, the ketogenic diet was then gradually forgotten as anticonvulsant medications were developed. The ketogenic diet has recently been 'rediscovered' and is achieving increasingly widespread use. Its modern day role as alternative management for children with refractory epilepsy is currently being re-defined. When it is considered as an alternative, it is carefully and individually calculated for each child; calories are restricted and depend on the age and activity of the child. If properly calculated and adhered to, the child should neither gain nor lose significant weight, but should grow normally for his/her stature and the weight should remain close to the ideal weight for their height. The diet must be supplemented with vitamins and calcium [22].

Surgical Treatment

Surgery tends to be considered for refractory seizures, and the decision to operate is influenced by the patient's perception of the severity of their condition, and takes into account their future expectations. The goal of epilepsy neurosurgery is to identify an abnormal area of cortex from which the seizures originate and remove it without causing any significant functional impairment. The primary components of the pre surgical evaluation includes a detailed clinical history, and physical examination, advanced neuro-imaging, video-EEG monitoring, neuropsychological testing and assessment of psychosocial functioning. The major surgical questions one hopes to answer with this evaluation are:

- **Are the seizures focal or generalised?**
- **If focal, are they temporal or extratemporal in origin?**
- **Is there a lesion associated with the seizures?**
- **If surgery is undertaken what functional deficits, if any, might be anticipated?**

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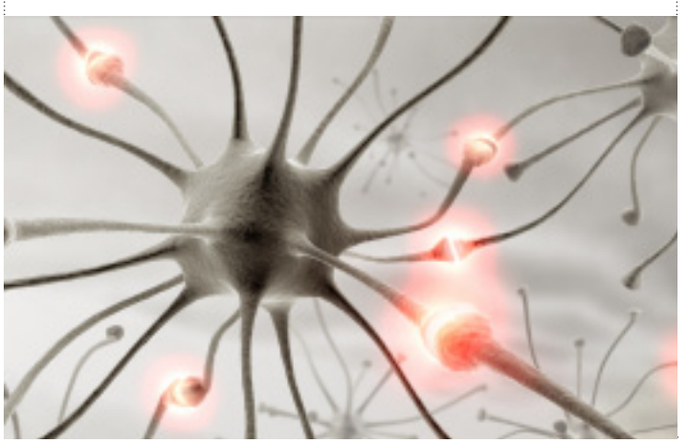
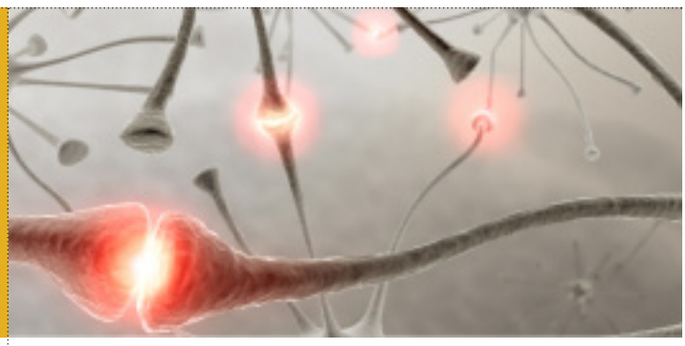
The Wada test or Intracarotid Sodium Amobarbital Procedure (ISAP) is used to establish which cerebral functions are localized to which hemisphere (e.g. the location of the language function). The test is conducted with the patient awake. Essentially, a barbiturate is introduced into one of the internal carotid arteries via a cannula or intra-arterial catheter from the femoral artery. The drug is injected into one hemisphere at a time. The effect is to shut down any language and/or memory function in that hemisphere in order to evaluate the other hemisphere. Then the patient is engaged in a series of language and memory related tests. This is a helpful guide to the surgeon and helps to reduce the risk of damaging these essential structures.

Epilepsy surgery began as removal of gross structural lesions of the brain. With the addition of EEG data from preoperative and intraoperative recordings, areas of removal expanded to include tissue that was grossly normal in appearance but known to give rise to epileptiform activity.

Small areas of resection were soon replaced by partial lobectomies and more extensive cortical resection. While resection techniques (lesionectomy, lobectomy, hemispherectomy, corticectomy) generally, yield the best surgical results, disconnection (callosotomy, subpial transection) and augmentation (cerebellar and vagal stimulation) techniques remain worthwhile considerations.

Studies have shown that although neurosurgery is a drastic measure used to tame seizure, it is very effective and greatly improves the quality of life of children, especially if they are rendered seizure-free. Improvements in level of attention, language, social interactions, self-esteem, physical restrictions, energy, behaviour and general health have been noticed by parents of children post-operatively [23].





Vagus Nerve Stimulation

Vagus Nerve Stimulation (VNS) is a form of treatment that is used in cases of intractable epilepsy. The device is programmed to provide baseline intermittent stimulation to the left vagus nerve of the neck, and it allows the patient or carer to activate the device transcutaneously with an external magnet (e.g. when the patient has an aura or seizure warning). So far this method of seizure control has had very promising reviews and outcomes, and is a safe and effective alternative for children with refractory epilepsy who are not candidates for resective surgery or who are otherwise untreatable [24]. The procedure to implant the device lasts approximately 1 hour, and side effects of the procedure can include voice alteration, coughing, chest discomfort, and nausea. These tend to be transient.

Needs and Concerns of Families

When talking about epilepsy, it is important not to ignore the psychological implications and difficulties that this disorder poses to both child and family. The concerns that children may express include feeling different from their peers, feeling left out of their management plan by healthcare professionals, or fearing the worst for their future, e.g., that they will die or be crippled by epilepsy. Parental concerns are to do with lack of information, lack of support for medical, developmental, emotional, and family issues related to coping [25].

If, these issues are well managed within multidisciplinary teams, the family's quality of life is vastly improved. Schools for children with special needs, with trained staff and a medical team can provide great relief and support for parents and can also improve quality of life for the child simultaneously.

Outcome for Children with Epilepsy

Children with epilepsy, as a group, experience a disproportionate degree of academic underachievement as compared with their epilepsy-free peers. As discussed previously, factors such as AED side effects, underlying brain abnormalities, pre-existing learning disabilities and seizures themselves can contribute to the reasons for poor academic outcome. This in turn may have an effect on their psychological, psychiatric, and also physical outcome in adulthood.

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Psychological Outcome

Depending on the severity of any associated learning difficulties, but also parental income, level of education, and psychosocial problems within the family unit, one can build up a hypothetical prediction for psychological outcomes in children with epilepsy. Studies show that young adults with epilepsy will find it more difficult to seek and sustain employment, and will also encounter troubles with the DVLA (Driving and Vehicle Licensing Agency); these experiences alone can have psychological impacts on them. Add to this a low self-esteem due to being different or restricted, and feelings of social alienation and one can see why almost 20% of adolescents affected with epilepsy are depressed. There are other psychiatric conditions commonly associated with epilepsy such as anxiety, neuroses, and psychoses. However with adequate support and psychotherapy (and pharmacotherapy) these may be avoided [26].

Physical Outcome

People living with epilepsy report a higher frequency of pain, and sleep problems, in fact, their overall health state is comparable to those suffering with other chronic diseases. Treatments can cause considerable physical effects, such osteoporosis and weight changes. Another physical problem is the one associated to injury during seizure, which is a major concern for the patients and their parents, especially when the child grows up and wants to be more independent.

Conclusion

Childhood epilepsy remains a relatively common paediatric disorder, and is undergoing many different trials in terms of treatments, psychological aid, and health awareness programmes. Pharmaceutical and surgical research continues to yield new and improved solutions to offer more advanced treatment options to patients living with epilepsy. Main focuses always being that of striving to ameliorate the patient and their families' quality of life.

References

1. O'Donohoe N. Editor, Epilepsies of Childhood. 1994, 3rd Ed. pp 1-3
2. Appleton R, Gibbs J. Editors. Epilepsy in Childhood and Adolescence. 1998, 2nd Ed. pp 1
3. Lagae L. What's new in: "Genetics in childhood epilepsy". *Eur J Pediatr.* 2008, 1432-1076
4. Patient UK website – Epilepsy in Children and Young people www.patient.co.uk [accessed: 3 May 2008]
5. Sisodiya M, Duncan J. Epilepsy: epidemiology, clinical assessment, investigation and natural history. Medicine. 2004, 30:10
6. Vijayan S. Childhood Epilepsies. Lancet Student. 2008
7. Cross H, Duncan J. The differential Diagnosis of Epilepsy, an Update. Janssen-Cilag DVD. 2005
8. Lissauer T, Clayden G. Editors. Illustrated Textbook of Paediatrics. 2007, 3rd Ed. p. 451-453
9. Lindsay, Bone Editors. Neurology and Neurosurgery Illustrated. 2004, 4th Ed. pp 91- 96
10. National Institute of Neurological Disorders and Stroke. www.ninds.nih.gov [accessed: 4 May 2008]
11. Juvenile Myoclonic Epilepsy of Janz. www.pediatricneurology.com [accessed: 4 May 2008]
12. Berg, Levy, Testa, Shinnar. Classification of Chilhood Epilepsy Syndromes in Newly Diagnosed Epilepsy: Interrater Agreement and Reasons for Disagreement. Epilepsia. 1999, 40(4):439-444
13. Pellock J. Understanding Co-morbidities Affecting Children with Epilepsy. Neurology. 2004, 62(2):17-23
14. Ballaban-Gil, Tuchman. Epilepsy and Epileptiform EEG: Association with Autism and Language Disorders. MRDD Research Reviews. 2000, 6:300-308
15. Gaitatzis, Carroll, Majeed, Sander. The Epidemiology of the Comorbidity of Epilepsy in the General Population. Epilepsia. 2004, 45(12):1613-1622
16. NICE Guidelines on Status Epilepticus. www.nice.org.uk. [accessed 5 May 2008]
17. Camfield P, Camfield C. The frequency of intractable seizures after stopping AEDs in seizure-free children with epilepsy. Neurology. 2005, 64:973-975
18. Pressler, Binnie, Coleshill. Effect of lamotrigine on cognition in children with epilepsy. Neurology. 2006, 66:1495-1499
19. Holland K, Glauser T. Response to carbamazepine in children with newly diagnosed partial onset epilepsy. Neurology. 2007, 69: 596-599
20. Geerts A, Niermeijer J. Four-year outcome after early withdrawal of antiepileptic drugs in childhood epilepsy. Neurology. 2005, 64:2136-2138
21. Waller, Renwick, Hillier Editors. Medical Pharmacology and Therapeutics 2001, 1st Ed. Pp. 264-267
22. Epilepsy Action website. www.epilepsy.org.uk [accessed 5 May 2008]
23. Sabaz, Lawson, Cairns. The impact of epilepsy surgery on quality of life in children. Neurology. 2006,66:557-561
24. Patwardhan, Strong, Bebin. Efficacy of vagal nerve stimulation in Children with Medically Refractory Epilepsy. Neurosurgery. 2000, 47(6):1353-1358
25. McNelis, Buelow. Concerns and Needs of Children with Epilepsy and Their Parents. Clinical Nurse Specialist. 21(4):195-202
26. Bailet, Turk. The Impact of Childhood Epilepsy on Neurocognitive and Behavioural Performance: A Prospective Longitudinal Study. Epilepsia. 2000,41(4):426-431

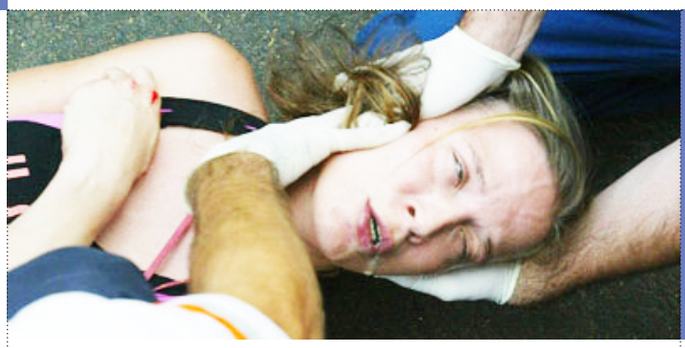
Authors

E Tirr is the lead author and a FY2 doctor currently working at Rochdale Infirmary, OL12 0NB, UK. E-mail: ericatirr@doctors.org.uk

N Halder is a ST6 Psychiatrist, and Medical Education Fellow currently working at Birch Hill Hospital, Rochdale, Union Road, OL12 9QB,UK.
He is the corresponding author. Email: neelh@hotmail.com

EEG IN THE DIAGNOSIS OF EPILEPSY

Gareth Payne



EEG in the diagnosis of epilepsy.
Practical Procedures.

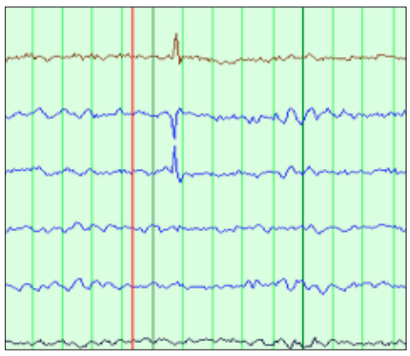
Introduction to the EEG

An electroencephalogram (EEG) is a recording of the electrical activity produced by the brain. The recording represents the activity of millions of neurons all discharging at different times.

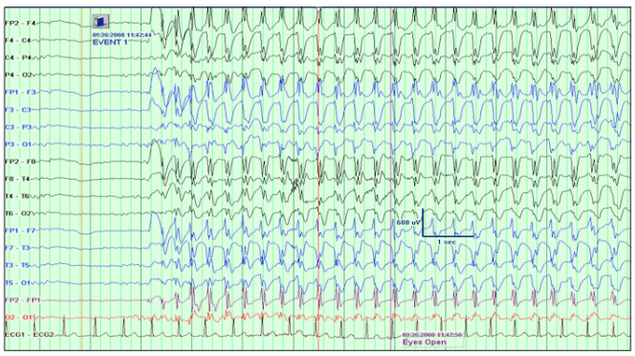
By comparing the responses from two electrodes, we can see distinct patterns and rhythms. Pathological changes result in altered rhythms such as slowing or in spikes caused by the synchronised discharge of electrical activity as in seizures.

The diagnosis of epilepsy is based almost entirely on the history and description of the attack. The aim of the EEG is to be able to confirm the diagnosis and, if possible, to give further information that may aid a doctor, such as whether onset is focal or generalised, whether there is photosensitivity and whether the attacks belong to a particular syndrome.

An example would be showing the difference between a generalised absence seizure and a complex partial seizure, both of which would be characterised by a short episode of staring.

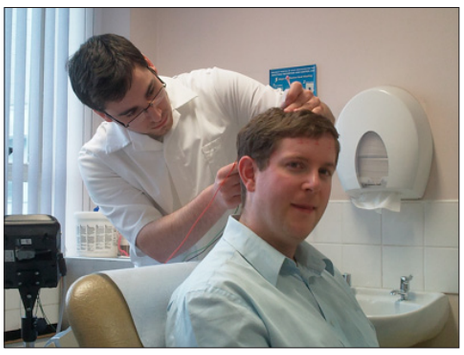


Temporal lobe sharp waves



A routine recording would typically take 30 min to prepare, 30 min to record and then a further 15 min to remove the leads. The preparation starts by explaining the procedure and obtaining consent. Round-disc electrodes are then attached to the head using electrode paste the consistency [Das1] of vasoline to predetermined locations around the scalp (see figure of montage)

Measuring For Electrodes



When the recording starts, a digital video camera is also pointed at the patient so that the EEG can be accurately correlated with the video that may show physical evidence of a fit. The patient will be asked to close their eyes for a period to accentuate certain rhythms in the 8–13 Hz range.

EEG IN THE DIAGNOSIS OF EPILEPSY

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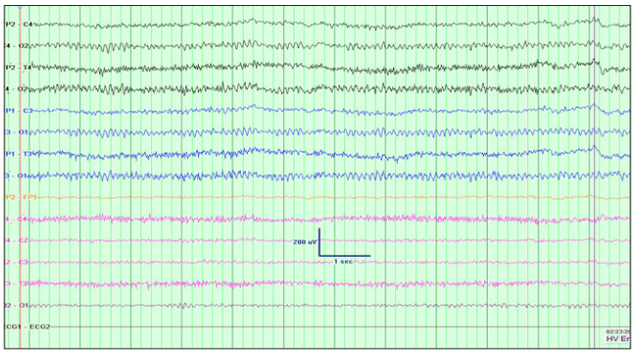
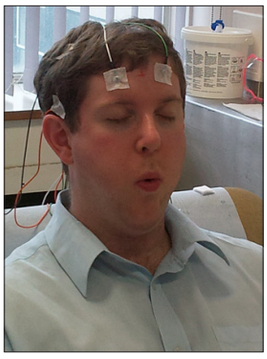


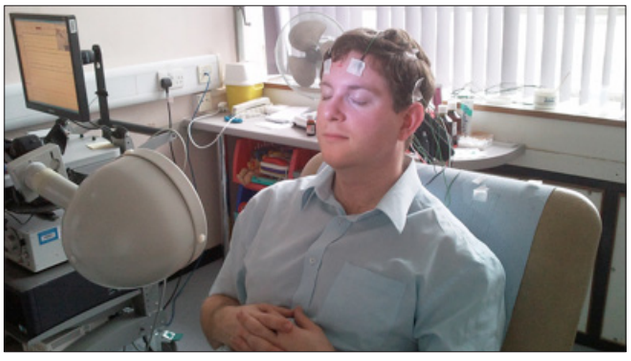
Halfway into the recording, all patients without a history of vascular malformations or cerebrovascular disease will be asked to hyperventilate for 3 min. This accentuates the rhythms of lower frequencies and in the case of some syndromes, e.g., childhood absence epilepsy can bring on an attack.

Hyperventilation

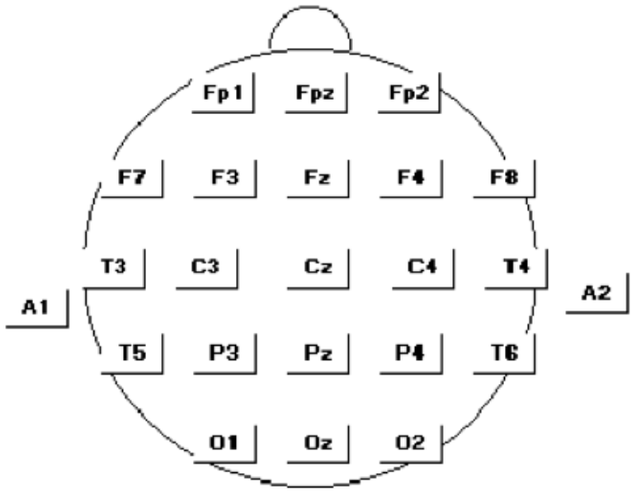


After at least 5 min of recovery, the patient will be shown a photic stimulus. This consists of a strobe light flashing 30 cm away from the patient's head at a range of frequencies between 1 and 60 flashes per second. The patients are asked to have their eyes open for 5 s followed by 5 s with their eyes closed. This can trigger changes in the EEG record in those with photosensitive epilepsy.

Photic stimulation



The 10–20 EEG montage



Learning objective 2

In addition to the routine EEG recording there are a number of variations that can be used to help pick up changes in the EEG rhythm in patients with suspected epilepsy. They are:

- sleep deprivation
- prolonged recordings
- ambulatory recordings
- videotelemetry
- intracranial recordings

Sleep deprivation

Typically the patient will be asked to wake up 5 h before their normal time of rising. The resulting fatigue lowers the seizure threshold and often the patient will sleep during the recording, which is helpful as often there will be interictal activity seen at the transitions with the early sleep stages. It can also allow the recording of seizures that are limited only to sleep.

Prolonged recordings

The recording may be extended for up to 8 h in those who have frequent, daily seizures but for whom no attack has yet been recorded. This has the advantage of increasing the chances of an attack being recorded on video and on EEG without having to admit the patient to the hospital.

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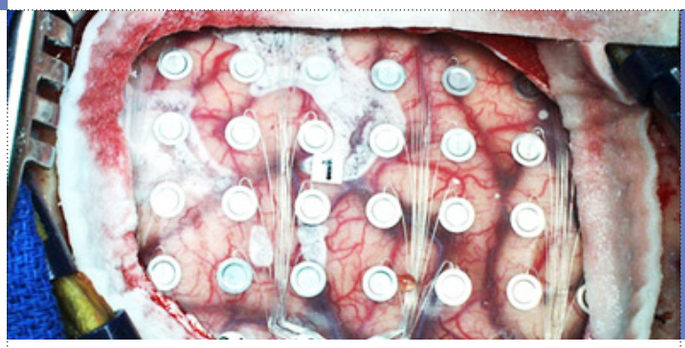

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Ambulatory recordings

The EEG electrodes are attached with collodion glue so that the leads shouldn't fall off. The patient is then allowed to go home and live normally, returning the next day for the equipment to be removed. The investigation can be particularly useful for the investigation of absence seizures or if it is felt that someone with known epilepsy might be having subclinical seizures or more attacks than they realise. It can also allow us to record the activity from a "normal" night's sleep. It does have a number of drawbacks: it is possible for the electrodes to fall off, with no one to reattach them. It is also prone to artefacts such as the electrical noise from a laptop or tooth brushing (which can look very similar to typical seizure activity). The other frustration is that it is not possible to see the patient fitting. So in the more vigorous attacks the EEG may only record muscle artefact so that it is not possible to see whether it is epileptic or non-epileptic in nature. As a result it is an unpopular investigation amongst the neurophysiologists that have to interpret them.

Video telemetry

These are similar to ambulatory recordings, but have the advantage that the patient can be kept under video surveillance. Typically a patient will be kept in an en suite side room that has digital video recording equipment that is linked into the EEG software. The patient can be kept under surveillance for many days, and if there is correct support any complication such as loose leads can be corrected promptly. Some units also reduce anti-epileptic medications in the hope of increasing the chances of patients having an attack. Although this is a labour-intensive and time-consuming investigation, which costs approximately £9,000 per week, it can be very rewarding in providing a diagnosis in carefully selected groups of patients.

Intracranial recordings

Occasionally greater information is required about the exact location of the origin of focal epilepsies within the brain. Intracranial recordings can be made using grids of electrodes under the dura or with electrodes placed into the substance of the brain. The recordings from these can be very clear and very useful, but there is a 4–14% risk of bleeding 5,6 and patients need antibiotic prophylaxis to prevent infective meningitis. The risks are such that the investigation is usually only reserved for patients with intractable epilepsy for whom epilepsy surgery is being considered.

Some reminders when requesting an EEG

• Please include all patient data including a telephone number as this will help to minimise waiting times

• Please include as much data about the attacks as possible. This will aid the neurophysiologist in writing a clinical report

• Please warn us of any known contraindications to parts of the investigation, e.g., vascular malformation, previous stroke, pregnancy, recent abdominal or thoracic surgery that would preclude hyperventilation

Objective 4

The use of EEG

EEG should never be used blindly as there are false-positive results, with typical interictal "epilepsy" changes seen in 0.5% of healthy adults[1] and 2–4% of children.[2] Occasionally changes are found which are unexpected, and it may be necessary to revisit the history before the diagnosis is made. It should also be emphasised that a normal EEG does not mean the patient does not have epilepsy. Only half of those with epil epsy will have interictal EEG activity on their first routine EEG, rising to 80% ii [Das[2]] there are four routine EEGs and recording during sleep.[3] Thus 20% will never have any changes between attacks and 10% will not have changes seen even during a seizure, especially if the seizure is in the frontal lobe, such as in the supplementary motor area which is far from the EEG electrodes.[4] In these cases the video recorded at the same time can be used to verify the diagnosis. Overall the sensitivity in epilepsy is low at 25–56%, but specificity is high at 78–98%.[4]

When to use an EEG

- To look for interictal changes in patients in whom seizures are suspected
- To try to differentiate focal seizures from generalised seizures
- To quantify subclinical seizures frequency
- To look for non-convulsive status epilepticus
- To check for photosensitivity
- To look for interictal activity in patients being considered for drug withdrawal
- To look for temporal lobe epilepsy in patients with acute confusion
- In rapid onset dementia when CJD is suspected
- Metabolic encephalopathies
- To investigate regression in speech development in children (Landau–Kleffner syndrome)
- To investigate cataplexy and other sleep disorders

EEG IN THE DIAGNOSIS OF EPILEPSY

Gareth Payne

When not to use an EEG

- When syncope is suspected – ECG and ECHO would be the first investigations
- Depression
- Schizophrenia
- Migraine and headache
- Chronic dementia

Case 1

A 13-year old boy is noted by his teachers to be having staring episodes and his school grades are dropping. His doctor wondered whether he was having absence seizures. The EEG showed that he was having frequent epileptic discharges in the left temporal lobe. Hence, a diagnosis of complex partial seizures was made. He was started on an appropriate medication for focal seizures.

Case 2

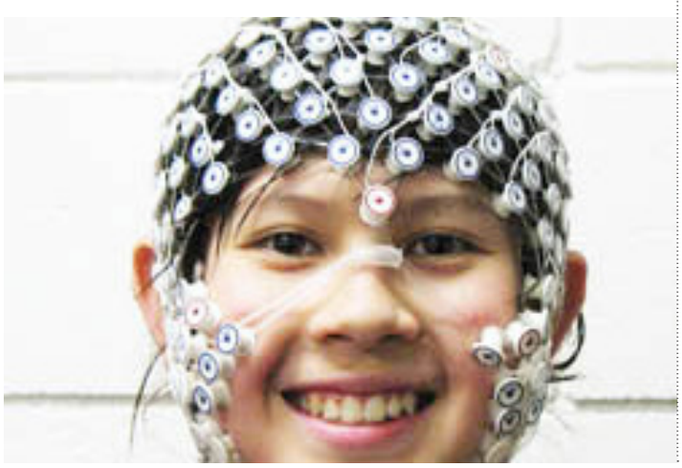
A 20-year old student has a history of morning myoclonic jerks and was labelled as a daydreamer at school. Juvenile Myoclonic epilepsy was suspected by her doctor who requested an EEG. This showed 3 Hz spike and slow wave activity during hyperventilation, and a photoparoxysmal response was seen during photic stimulation, suggesting that she is photosensitive. The doctor was able to give her advice regarding avoiding flickering lights, such as strobe lights in a night club, keeping a background light on whilst watching television and turning her television off with a remote control and she was started on Lamotrigine.

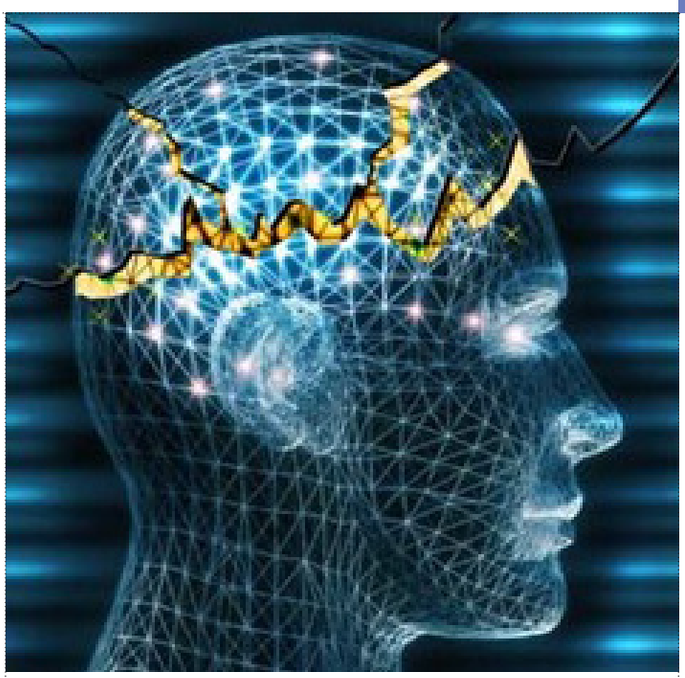
Case 3

A 21-year old trainee RAF fighter pilot attended for his routine EEG as part of his occupational health screening. Unfortunately the EEG showed spikes in the right temporal leads. He had no previous history to suggest a seizure, but the RAF medically downgraded him. Moreover, he was not being allowed to continue his training. Such findings are found in 1 in 200 of applicants.

Conclusion

Epilepsy diagnosis is based almost entirely on the description of the event. EEG can be a useful tool to help characterise and confirm the seizure type, but it should be used appropriately because it has the possibility to create false-positive results that might distract from the correct diagnosis.





Suggested further reading

Smith SJM (2005). EEG in the diagnosis, classification and management of patients with epilepsy. JNNP 76:Sii2–7.

References

1. Gregory RP, Oates T, Merry RG (1993). EEG epileptiform abnormalities in candidates for aircrew training. Electroencephalogr Clin Neurophysiol.86:75–7.
2. Zifkin L, Ajmone Marsan C (1969). Incidence and prognostic significance of "epileptiform" activity in the EEG of non-epileptic subjects. Brain 91:751–78.
3. Binnie CD (1996). Epilepsy in adults: diagnostic EEG investigation. In: Kimura J, Shibasaki H (eds.). Recent advances in clinical neurophysiology. Amsterdam: Elsevier. 217–22.
4. Smith SJM (2005). EEG in the diagnosis, classification and management of patients with epilepsy. JNNP 76:Sii2–7.
5. Van Buren JM (1987). Complications of surgical proceduresnin the diagnosis and treatment of epilepsy. In: Engel J (ed). Surgical Treatment of Epilepsies. New York: Raven Press. 465–475.
6. Wyllie E, Luders H Morris H, et al (1988). Subdural electrodes in the evaluation of epilepsy surgery in children and adults. Neuropaediatrics 19:80–86.

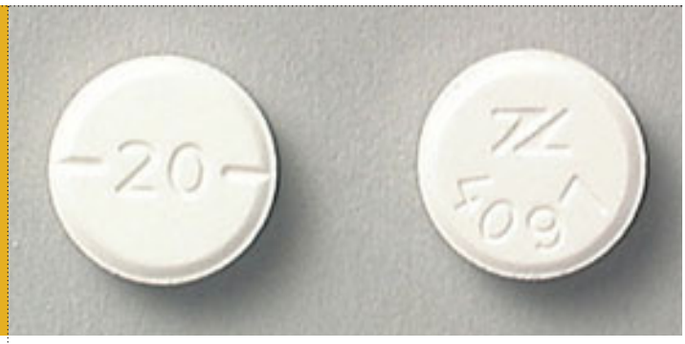
Authors

Dr Gareth Payne, Consultant Clinical Neurophysiologist,
Mr Robert Goulding, Student Clinical Physiologist
University Hospital of Wales, Cardiff

A CASE OF BACLOFEN TOXICITY IN AN ELDERLY PATIENT

Paul Zammit, Rosalie Magro

A Case of Baclofen Toxicity in an Elderly Patient. Good Clinical Care.



Abstract

Baclofen is one of the commonly used drugs in the treatment of spasticity for various conditions. It may cause a wide range of side effects and may cause acute as well as chronic toxicity. This is more likely prevalent in elderly patients and in patients with chronic renal failure.

A case report is being described of a 64-year old lady with multiple medical problems including renal impairment, who presented with episodes of loss of consciousness (LOC) and increasing drowsiness. After a series of medical tests to exclude neurological and cardiological causes for the LOC was done, it was suspected that the baclofen medication was causing these events. On tailing off the baclofen there was a dramatic improvement in the patient's condition with full recovery to her previous medical condition. In this discussion, an overview of the drug balcofen including its uses, side effects, toxicity and management of the latter is explored in more detail.

Key words

Baclofen toxicity, elderly, loss of consciousness.

Introduction

Baclofen is a medication that is mostly used to treat spasticity in neurological disorders. We report a case of an elderly woman in Malta who developed episodes of LOC with increasing drowsiness causing total dependency secondary to baclofen toxicity. On tailing off this medication there was a dramatic improvement in the patient's condition with recovery to her previous state allowing her to be discharged back home. The drug baclofen, its uses, effects, toxicity and other case reports similar to this case are discussed.

Case Report

Mrs. CM was a 64-year old lady admitted to hospital after an episode of LOC lasting a few minutes. A similar episode had occurred before 6 months. On examination her parameters were normal, including pulse, blood pressure and glucose levels. She was found to be neurologically intact and fully oriented. She was a known case of ischaemic heart disease, renal impairment, mild dementia, diabetes and hypertension. Blood tests showed a mild anaemia of 9.6, a creatinine of 170 µmol/L, normal lived function tests, electrolytes (including sodium, calcium and magnesium) and thyroid function tests. A scan of her brain was done which was normal. During her admission she had a further eight episodes of LOC. She was also noted to be more drowsy during her stay. Further tests were done including a repeat CT brain, EEG and 24-h ECG monitoring that were normal. An echo found moderate aortic stenosis, which was managed conservatively by the cardiologists. A coronary angioplasty was done during her stay for stenosis of the left anterior descending artery. A review by a neurologist suspected epilepsy despite the normal EEG, and the patient was started on sodium valproate. The patient progressively got more drowsy with low consciousness levels. She eventually became bed bound and required nasogastric tube feeding.

She was referred after 2 weeks to the geriatricians for further management. On examination her Glasgow coma scale was 12/15, and she had fixed pupils. Reviewing her drug chart she was noted to be on baclofen 25 mg bd. It was suspected that baclofen overdose may be causing the drowsiness, LOC and suspected seizures. In her old medical records, it was noted that they had been given on a prn basis by her general practitioner for muscle cramps, but on admission to hospital these were written on a regular basis on her drug chart. On tailing off the baclofen there was a marked improvement in the patient's general condition. By the next day GCS was 15/15, and she was able to feed independently without swallowing problems. The nasogastric tube was thus removed. Baclofen was slowly tailed off to avoid withdrawal symptoms. On discharge the patient was off this treatment and back to the same condition as before her original admission for LOC. Serum baclofen levels are unavailable in Malta; so the diagnosis was made clinically on the rapid improvement of the patient on tailing off this treatment.

A CASE OF BACLOFEN TOXICITY IN AN ELDERLY PATIENT

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Discussion

Baclofen is a derivative of gamma-aminobutyric acid (GABA) primarily used for the treatment of spasticity in children and adults resulting from multiple sclerosis and spinal cord injuries/diseases. If administered intrathecally it is used for spasticity related to cerebral palsy and spinal cord injury. Baclofen has also been shown to be as effective as diazepam in uncomplicated alcohol withdrawal syndrome .

The drug is rapidly absorbed after oral administration and is widely distributed throughout the body. In a healthy patient, most of the ingested baclofen is excreted in an unchanged form in the urine with a half-life ranging from 2 to 6.8 h. In patients with chronic renal failure, however, the recommended dose or even low doses could cause a rapid baclofen accumulation that may lead to neurological deterioration and coma .

Baclofen can be administered either orally or intrathecally. Intrathecal administration is often preferred in spasticity patients, as very little of the oral dose actually reaches the spinal fluid.

Baclofen therapy is usually started with an initial low dose of about 10 mg daily in divided doses and gradually titrated up in a stepwise fashion until symptomatic relief occurs. The usual maximum dose is 80 mg per day.

Baclofen should be used with caution in psychiatric illness, Parkinson's disease, cerebrovascular disease, elderly, respiratory impairment, epilepsy, history of peptic ulcer, diabetes, hypertonic bladder sphincter, renal impairment and pregnancy.

A double-blind crossover trial of baclofen against placebo in elderly stroke patients was discontinued because the drug produced an unacceptably high level of drowsiness. It was also found that elderly patients took longer to achieve peak plasma baclofen concentrations and eliminated the drug more slowly when compared to healthy controls.

Baclofen may cause acute neurotoxicty particularly in patients with chronic renal impairment due to baclofen accumulation. These patients present with four major clinical manifestations: encephalopathy (disturbance of consciousness and/or seizure), respiratory depression, muscular hypotonia and generalized hyporeflexia.

There are a few reported cases of baclofen neurotoxicity in elderly patients with mild renal impairment. A reported case was that of a 74-year old woman with a history of cerebrovascular disease who developed profound central nervous system plus respiratory depression, generalized hypotonia, sinus bradycardia and urinary retention following an increase in the dose of baclofen . This patient was on baclofen 15 mg three times daily and her serum creatinine was 159 µmol/L. Cessation of baclofen therapy and the relief of the urinary obstruction improved mental status and normalized motor function within 24 h. Thus patients with various forms of CNS disease states may be at risk of serious CNS depression with even small therapeutic doses of baclofen.

There are 21 reported cases of acute baclofen-induced neurotoxicity in elderly patients with renal failure . Serum levels of creatinine ranged from 247 µmol/L to 495 µmol/L. The interval from the start of baclofen therapy to the onset of acute neurotoxicity ranged from 3 h to 4 days. The baclofen dose ranged from 5 mg tds to 20 mg tds. Altered level of consciousness was the major presenting feature of acute neurotoxicity. Other reported symptoms include hyporeflexia, hypotonia, hypotension, myoclonus, muscle weakness, abdominal pain, bradycardia and respiratory depression. Akinetic mutism was another rare symptom[6].

In these cases, the toxic effect of baclofen appeared to be associated with the degree of renal function impairment, the dose and duration of baclofen. Management in all the reported cases involved the discontinuation of baclofen and in some cases haemodialysis or peritoneal dialysis.

Patients with chronic intoxication present with hallucinosis, impaired memory, catatonia or acute mania. Respiratory depression, apnoea, bradycardia, tachycardia, hypotension, hypertension, tremor, weakness, hypotonia, areflexia, urinary retention, sedation, coma seizures, orofacial dyskinesia and hypothermia also have been reported as manifestaions of chronic baclofen toxicity[7].The acute intoxication syndrome has a faster onset, shorter duration, more severe clinical manifestations and higher incidence of seizures than the chronic intoxication syndrome. Baclofen intoxication, although it may cause grave encephalopathic manifestations and electroencephalographic findings, has a benign outcome if actively managed.

A CASE OF BACLOFEN TOXICITY IN AN ELDERLY PATIENT

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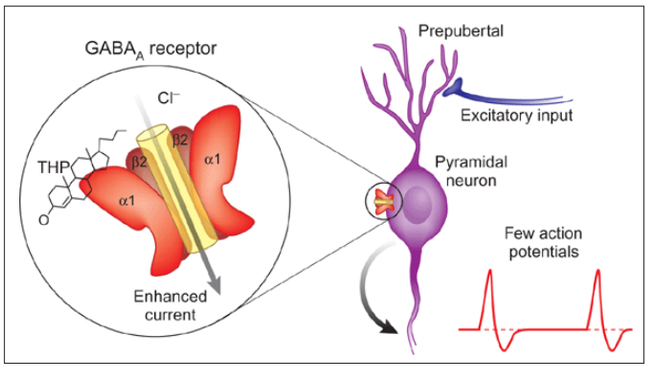


The management of baclofen toxicity involves the discontinuation of baclofen. The dose should be tapered down slowly as abrupt discontinuation can be associated with a withdrawal syndrome. Withdrawal symptoms may include hallucinations, delusions, agitation, delirium, disorientation, fluctuation of consciousness, insomnia, inattention, memory impairments, perceptual disturbances, anxiety, depersonalization, hypertonia, hyperthermia, formal thought disorder, psychosis, mania, mood disturbances, restlessness and behavioral disturbances, tachycardia, seizures, tremors, autonomic dysfunction, hyperpyrexia, extreme muscle rigidity resembling neuroleptic malignant syndrome and rebound spasticity. Vulnerability to baclofen-withdrawal delirium is greater in individuals who received chronic baclofen therapy and in elderly patients[8,9]. There is no specific antidote for baclofen intoxication. Hemodialysis is an effective means of reversing baclofen-induced neurotoxicity[10].

Conclusion

Physicians should be aware of the possibility of baclofen-induced neurotoxicity in elderly patients especially those with chronic renal impairment. Thus baclofen should be avoided in these patients.

MCQs

MCQ 1: Baclofen acts on which of the following receptors?

1- NMDA receptors
2- GABA receptors
3- K-opioid receptors
4- Alpha adregernic receptors
5- Dopamenergic receptors

MCQ 2: Baclofen may cause:

1- Acute renal failure
2- Hypertension
3- A transient increase in gamma-glutamyl transpeptidase (GGT) levels
4- Hypoglycaemia
5- Seizures

Answers

1. The answer is (2).

Baclofen works on the inhibitory GABA receptors.

2. The answer is (5).

Baclofen does not cause renal failure but toxicity is increased with patients having this pathology. Balcofen may cause hyperglycaemia and/or hypotension. Baclofen may increase alkaline phosphatase levels. Baclofen lowers the seizure threshold, thus increasing the risk of seizures.

References

1. Addolorato G, Leggio L, Abenavoli L, et al. (2006). Baclofen in the treatment of alcohol withdrawal syndrome: A comparative study vs diazepam. Am J Med 119:276.e13–276.e18.
2. Chou CL, Chen CA, Lin SH, et al. (2006). Baclofen-induced neurotoxicity in chronic renal failure patients with intractable hiccups. South Med J 99:1308–1309.
3. Hulme A, MacLennan WJ, Ritchie RT, et al. (1985). Baclofen in the elderly stroke patient its side-effects and pharmacokinetics. EJCP 1985; 29: 467–469.
4. White WB (1985). Aggravated CNS depression with urinary retention secondary to baclofen administration. Arch Intern Med 145:1717–1718.
5. Su W, Yegappan C, Carlisle EJF, et al. (2009). Reduced level of consciousness from baclofen in people with low kidney function. BMJ 339:b4559.
6.Rubin DI, So EL (1999). Reversible akinetic mutism possibly induced by baclofen. Pharmacotherapy 19: 468–470.
7. Lee TH, Chen SS, Su SL, et al. (1992). Baclofen intoxication: Report of four cases and review of the literature. Clin Neuropharmacol 15:56–62.
8. Leo RJ, Baer D (2005). Delirium associated with baclofen withdrawal: A review of common presentations and management strategies. Psychosomatics 46:503–507.
9. O'Rourke F (2001). Withdrawal of baclofen may cause acute confusion in elderly patients. Br Med J 323:870.
10. Chen KS, Bullard MJ, Chien YY, et al. (1997). Baclofen toxicity in patients with severely impaired renal function. Ann Pharmacother 31:1315–1320.

Authors

Paul Zammit MD MRCP (UK)
(Corresponding author)
Trust Registrar at the Royal Bournemouth Hospital, Bournemouth
and Higher Specialist trainee in Geriatric Medicine in Malta

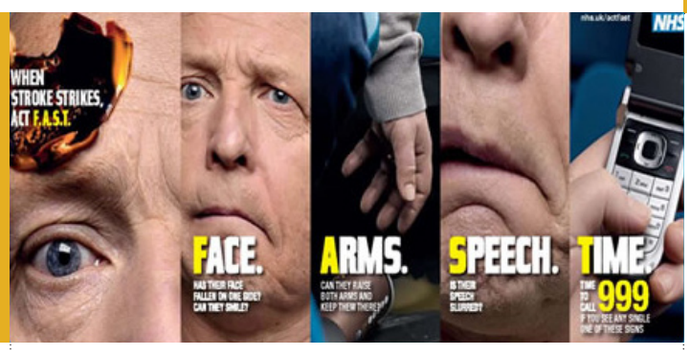
Karen Grech Hospital, Luqa, Malta
Email: pzamm@hotmail.com

Rosalie Magro MD
Basic Specialist trainee in General Medicine in Malta
Mater Dei Hospital, Tal- Qroqq, Malta

MANAGEMENT OF ACUTE STROKE

Kirsty Lawton, Nicola Gainsborough

Management of Acute Stroke.
Good Clinical Care.



Abstract

This article presents a young male with acute stroke. It covers the initial assessment and management of acute stroke, including assessment for thrombolysis. There is also an overview of acute ischaemic changes that can be detected on CT head scans. It is important for foundation year doctors to be aware that stroke is a medical emergency and how they can help facilitate prompt management and treatment.

Case History

A 48-year old gentleman with acute onset left-sided hemiplegia, left facial weakness, expressive dysphasia and left homonymous hemianopia arrived in accident and emergency 40 min after symptom onset. Following a 'stroke team call ' the stroke consultant assessed the patient using Recognition of Stroke in the Emergency Room (ROSIER) acute stroke assessment tool. The clinical diagnosis of stroke was confirmed and thus workup for thrombolysis was initiated by taking the patient immediately for a CT brain scan. The CT scan showed some hyperacute ischaemic changes and importantly no evidence of haemorrhage. The diagnosis of a right total anterior circulation infraction (TACI) was made, and the patient was thrombolysed in the radiology department 1 h and 7 min after the onset of symptoms and transferred directly to the stroke ward.

Clinical background of stroke

Stroke results from ischaemic infarction or haemorrhage into part of the brain, manifested by rapid onset of focal neurological signs and symptoms [1]. During acute stroke, 1.9 million neurons die every minute[2] .Within this ischaemic cerebrovascular bed are two zones of confluent injury: the core of profoundly ischaemic brain and the surrounding penumbra of potentially salvageable brain. Every stage of the assessment until treatment is given is therefore time critical and foundation year doctors are well placed to make this pathway as quick and effective as possible. The recovery potential of the brain is maximised by active management helping to minimising the amount of ischaemic neuronal tissue[3]. Every year in the UK, 110,000 people in England have a stroke and 25% of strokes occur in people who are under 65[4].

In the last decade, our approach to stroke has changed substantially. Previously we took a nihilistic approach where it was felt that little could be changed acutely. Currently, time is brain, and emergency stroke protocols in accident and emergency departments identify patients in the therapeutic time window for thrombolytic therapy. There is plenty of evidence to suggest that active stroke management makes a real difference in terms of reducing mortality, improving functional recovery and minimising residual disability [3,4,5]. The UK Stroke Association's Act FAST campaign has been designed to help the general public, paramedics and other health professionals recognise symptoms of stroke in the community and facilitate immediate admission to hospital [6].

What would you do next?

People who present with acute stroke need immediate clinical assessment and physicians need to rapidly confirm the diagnosis of stroke, exclude haemorrhagic stroke, eliminate stroke mimics and approximate the vascular territory and size of the infarction [7].

Clinical history and presentation:

The diagnosis is rapidly established by taking an accurate history and examination after using a validated tool, such as ROSIER[8]. Patients with acute onset neurological symptoms and signs that persist need a clear diagnosis to separate out acute cerebrovascular causes from other causes, especially those such as hypoglycaemia, syncope and seizures [5,8].

ROSIER stroke assessment tool [8]

The aim of this assessment tool is to enable medical and nursing staff to differentiate between stroke and stroke mimics.

GCS: E_____ M _____ V_____

Blood Pressure: ___/___

Blood Glucose: _____ (if less then 3.5 mmol/l treat urgently and reassess)

Has there been seizure activity	Yes (-1)	No (0)
Was there loss of consciousness of syncope?	Yes (-1)	No (0)

MANAGEMENT OF ACUTE STROKE

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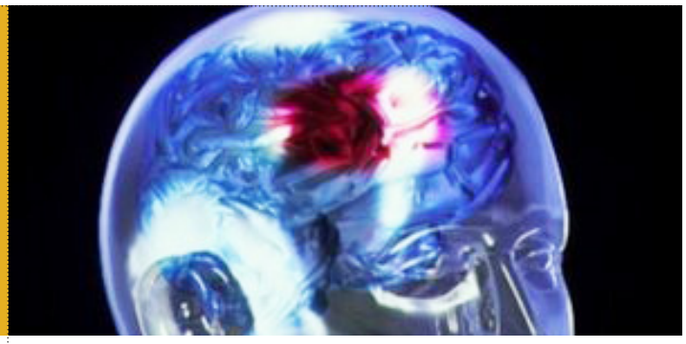

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Is there NEW ACUTE onset (or on awakening from sleep)

1. Asymmetrical facial weakness	Yes (+1)	No (0)
2. Asymmetrical arm weakness	Yes (+1)	No (0)
3. Asymmetrical leg weakness	Yes (+1)	No (0)
4. Speech disturbance	Yes (+1)	No (0)
5. Visual field defect	Yes (+1)	No (0)

Total score (-2 to +5) _____

Interpretation:

Stroke is likely if score is >0

Scores of ≤0 have a low possibility of stroke but it is not totally excluded (8).

Urgent subsequent steps include gaining IV access and sending baseline bloods. Instead of: Gain IV access and send urgent bloods. This is essential in the management of acute stroke, a patient cannot be given thrombolysis without knowing the baseline investigations (FBC, INR, urea and electrolytes) and the alteplase cannot be administered without intravenous access. As a foundation year doctor it is important to ensure these baseline bloods are run to the lab directly and the results chased while the patient is having the CT scan.

Organise an urgent CT brain scan: The CT brain scan is needed to differentiate between those who have had an ischaemic stroke and those who have had haemorrhage as it is not possible to do this by clinical examination alone. As a foundation year doctor you can speed up this process by discussing the case with colleagues in radiology and ensuring the patient gets immediate imaging.

Acute Phase Care: Thrombolysis

If 10% of acute strokes were thrombolysed, over 1,000 people per year would regain independence rather than die or be dependent in the long term [4]. A Cochrane review of intravenous thrombolysis included data from randomised control trials of rt-PA (alteplase 0.9 mg/kg up to maximum of 90 mg) administered within 4 ½ h of stroke onset significantly reduces death and disability at 90 days [9,10]. The odds of a favourable outcome (near or full recovery) are strongly correlated to the time of treatment and are significantly greater the earlier the treatment is delivered [7,9]. Administration later than 4 ½ h is associated with increased risk of mortality and bleeding [9]. Any patient seen within 4 ½ h of symptom onset and who has been shown not to have intracerebral haemorrhage (or other contraindications) should be treated with alteplase by a trained physician [11].

Medical Therapy

Not everyone will benefit from thrombolysis, but all stroke patients benefit from direct admission into an acute stroke specialist unit[12].

Maintaining homeostasis: Oxygen should be administered aiming to have oxygen saturations of 94–98% or 88–92% if the patient is at risk of hypercapnic respiratory failure [13] blood glucose and blood pressure should all be monitored carefully. All ischaemic strokes are given aspirin 300 mg orally if not dysphagic or aspirin 300 mg rectally or by enteral tube if dysphagic. For every 1,000 patients treated with aspirin, 13 patients will avoid death or dependency (number needed to treat to benefit 79). Antiplatelet therapy is associated with a small but definite excess of symptomatic intracranial haemorrhages, but this is more than offset by the reduction of recurrent ischaemic strokes [14]). Aspirin 300 mg is given for 14 days and then reduced to 75 mg thereafter.

Patients should be made nil by mouth until assessment of their swallow has been undertaken by a trained member of the stroke team to prevent aspiration pneumonia therefore maintenance intravenous fluids are also important in the acute stage[7].

Early mobilisation: Stroke patients are often immobile. The deleterious consequences of even brief periods of immobility in any patients can predispose to DVT, PE and pressure sores. People with acute stroke should be mobilised as soon as possible (when their clinical condition permits) as part of the active management programme of a specialist stroke unit [5].

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Imaging

As mentioned early CT brain scanning is essential. Plain CT may detect the presence of haemorrhage, middle cerebral artery (MCA) thrombus, early grey-white matter changes. CT perfusion scan may help to detect the presence and size of a core of irreversibly infarcted tissue and the presence of hypoperfused tissue at risk of subsequent infarction unless adequate perfusion is restored. A significant early sign of cerebral ischaemia within the first few hours after symptom onset is loss of grey-white differentiation, because there is an increase in the relative water concentration of ischaemic tissue. This sign includes loss of distinction and blending of the densities in the cortex and underlying white matter. The subsequent swelling of the gyri produces sulcal effacement. The sooner these signs become evident the more profound the degree of ischaemia. Another important CT sign is that of increased density within the occluded vessel, which represents intrarterial thrombus. When this is the MCA it is called the hyperdense MCA sign, this sign is seen in one-third to one-half of all cases of angiographically proven thrombosis. Hence is an important indicator of thrombus when present, but its absence does not exclude thrombus [15].

Hyperacute changes seen in ischemic stroke on CT head

Low density: Loss of grey and white matter definition

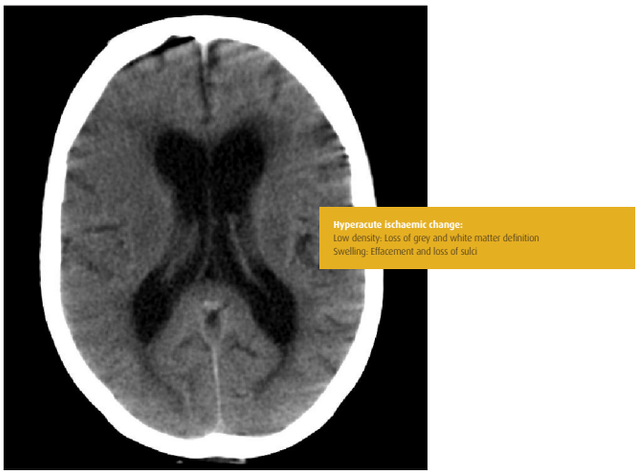
Swelling: Effacement and loss of sulci

Effacement of the ventricles.

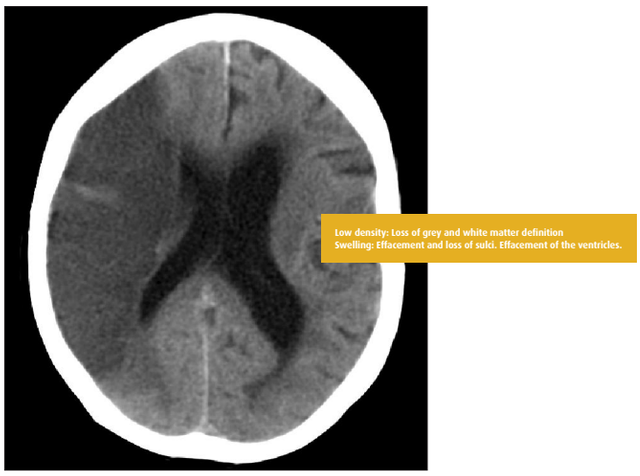
Midline shift (if there is significant swelling)

Arterial changes: Hyperdense/Thrombosed artery

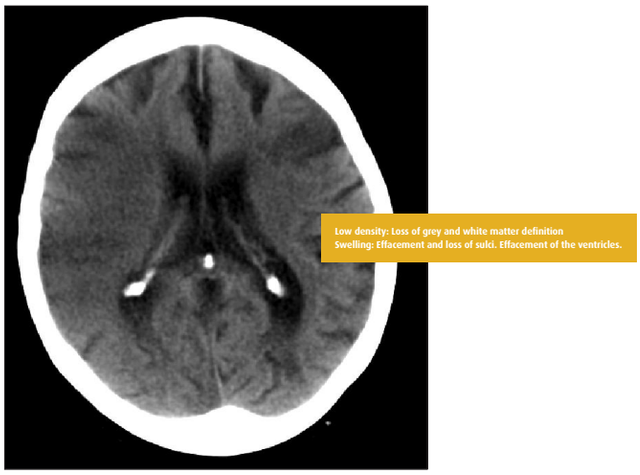
Examples of ischaemic infarction on CT scan



CT brain 1: Patient at 1 h after symptom onset: Loss of grey/white differentiation.



CT brain 2: Same patient as CT brain 1 after 24 h



CT head 3:

CT brain 4 : CT brain demonstrating Hyperdense MCA sign. This is a relatively large mature infarction

MANAGEMENT OF ACUTE STROKE

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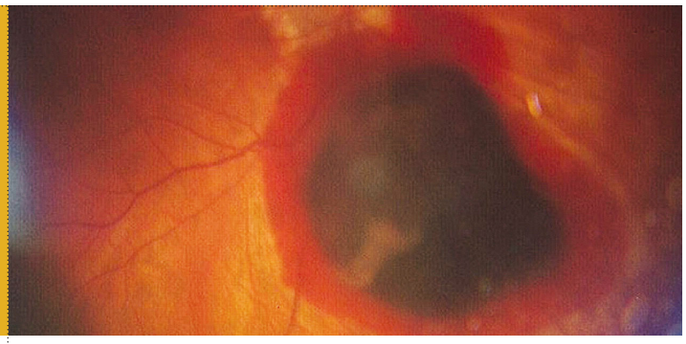

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Final: Case review

The patient's symptoms began resolving immediately after thrombolysis; however, he was left with some residual weakness and sensory deficit in the left arm. He went on to have further investigations that demonstrated a 70% stenosis of the right carotid artery and proceeded to carotid endarterectomy. He was treated with aspirin, dipyridamole, a statin for hypercholesterolaemia, antihypertensives, and given smoking cessation and dietary advice. He was discharged from the acute stroke unit to a rehabilitation hospital and is expected to lead an independent life.

MCQ questions:

(1) Within the first 24 h, which of these is NOT used in the management/treatment of acute ischaemic stroke?

a. Oxygen
b. Aspirin
c. Alteplase
d. Dipyridamole
e. IV fluid

(2) A 72-year old gentleman is admitted with acute onset left hemiparesis, CT scan shows an ischaemic stroke and 300 mg of aspirin is given. In terms of further management in the acute phase which of the following parameters should not be corrected?

(a) Blood pressure: 200/100
(b) Blood glucose: 13
(c) Oxygen saturations: 93%
(d) Temperature: 38.4
(e) Blood glucose: 3.2

Answers

1. Answer : d
Dipyridamole MR 200 mg BD is added after 14 days when aspirin 300 mg is reduced to 75 mg.

2. Answer: a
Elevated blood pressure should not be treated in the acute phase unless complications develop. Other parameters such a blood glucose, temperature and oxygen should be kept within the normal limits.

References

1. Longmore M, Wilkinson I, Turmezei T, Cheung CK (2007). Oxford Handbook of Clinical Medicine. Seventh Edition. Oxford. Oxford University Press.

2. Jeffrey L, Saver MD (2006). Time is brain- quantified. Stroke. 37:263–266.

3. NHS Improvement- Stroke. Why treat stroke and transient ischaemic attacks as emergencies? http://system.improvement.nhs.uk/ImprovementSystem/ViewDocument.aspx?path=Cardiac%2FNational%2FWebsite%2FStroke%2FWhy_Treat_Stroke%5B1%5D.pdf

4. National Stroke strategy. Department of health. 2007. www.dh.gov.uk/en/publicationsandstatistics/publications/publicationspolicyandguidance/dh_081062

5. National clinical guideline for diagnosis and initial management of acute stroke and transient ischaemic attack – NICE guidance published by RCP 21st July 2008. http://guidance.nice.org/CG68/guidance/pdf/English

6. National clinical guideline for stroke. 3rd edition. Royal college of physicians July 2008. http://www.rcplondon.ac.uk/pubs/contents/6ad05aab-8400-494c-8cf4-9772d1d5301b.pdf

7. Scottish Intercollegiate Guidelines Network: Management of patients with stroke or TIA: assessment, investigation, immediate management and secondary prevention. A national clinical guideline. http://www.sign.ac.uk/pdf/sign108.pdf

8. Nor A, Davis J, Sen B, et al. (2005). The Recognition of Stroke in the Emergency Room (ROSIER) scale: Development and validation of a stroke recognition instrument. Lancet Neurology 4(11):727–734.

9. Wardlaw JM, del Zoppo G, Yamaguchi T, et al. (2005). Thrombolysis for acute ischaemic stroke. The Cochrane Library; CD000213.

10. Hacke W, Kaste M, Bluhmki E, et al. (2008). Thrombolysis with alteplase 3 to 4.5 hours after acute ischemic stroke. N Engl J Med 359 (13):1317–29.

11. National institute of clinical excellence: Guidelines: Alteplase for the treatment of acute ischaemic stroke. June 2007.
http://www.nice.org.uk/TA122.

12. Stroke units trialist collaboration: Organised inpatient (stroke unit) care for stroke. The Cochrane Database Syst Rev. 2007; Oct 17(4) CD000197.

13. BTS Guideline for Emergency Oxygen Use in Adult Patients. Thorax. 2008: 63: vi1–vi68.

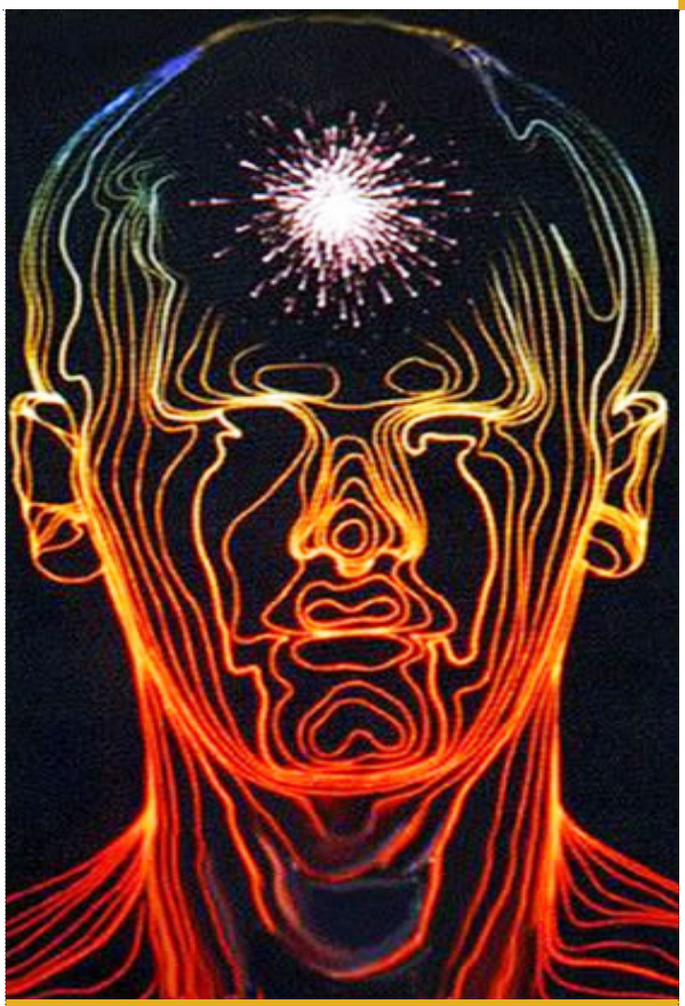
14. Sandercock PA, Counsell C, Gubitz GJ, et al. (2008). Antiplatelet therapy for acute ischaemic stroke. Cochrane Database Syst Rev. (3):CD000029.

15. Latchaw RE, et al. Recommendations for imaging of acute ischaemic stroke: A Scientific statement from the American heart Association. Stroke: Journal of American heart association. 2009; 40;3646-3678

Authors

Kirsty Lawton: Foundation year two doctor.
Brighton and Sussex
NHS hospital Trust.

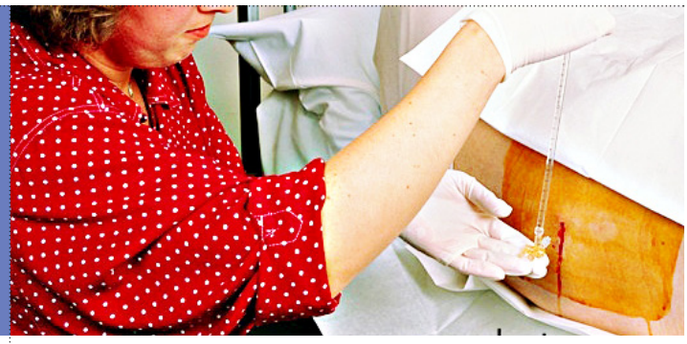
Nicola Gainsborough: Consultant Stroke Physician.
Brighton and Sussex
NHS hospital Trust



Management of Acute Stroke.
Good Clinical Care.

PERFORMING A LUMBAR PUNCTURE

Lawson Aimee



Performing a Lumbar Puncture.
Practical Procedures.

Presenting complaint: Sudden onset occipital headache.

History presenting complaint: Felt like someone had 'hit him across the back of the head with a spade' even as lifting weights at the gym the morning before. He curled up into a ball unable to be in motion for 10 minutes due to pain. The headache then eased from severe to moderate but did not resolve. He experienced no aura, no loss of consciousness, no visual disturbance and no sensory or motor symptoms. He went to his GP the next day and was referred to the on call neurology registrar.

Past medical history: Migraines and diet controlled diabetes.

Drug history: Nil (no allergies).

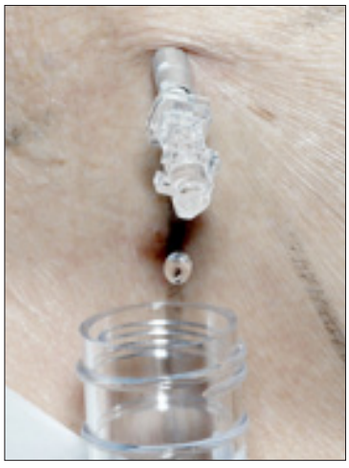
Family history: Type 2 diabetes.

Social history: Lives with wife. The patient is able to perform all activities of daily living. The patient is a non-smoker and drinks alcohol occasionally.

On examination: Blood pressure, pulse, respiratory rate, temperature and saturations were within normal parameters. No abnormalities found in the cardiovascular, respiratory or abdominal system. Examination of the cranial nerves and peripheral neurological system showed no focal neurology. No meningism was elicited. Fundoscopy was normal.

Performing a Lumbar Puncture

Aimee Lawson, Mr Michael Mr S presented with a 'thunderclap headache' (Fig 1), which has a broad differential. The most common life threatening cause is a subarachnoid haemorrhage and it is imperative that this is ruled out. Other diagnosis included in the differential includes benign disorders such as migraine, primary exertion headache & coital headache. There are also more serious conditions such as an un-ruptured cerebral aneurysm, intracranial infection, cerebral venous sinus thrombosis, cervical artery dissection and pituitary apoplexy [1-5]. Posterior fossa stroke or primary intracerebral bleed may present with acute headache but should have other associated neurology apart from the headache.



Mr S had a head CT that showed 'no evidence of haemorrhage or infarct'. Although a CT head is excellent at imaging haemorrhage within the first 12 hours, after this time period a SAH may be missed [6]. To increase diagnostic accuracy the CSF should be examined for the presence of xanthochromia (yellow tinge from bilirubin) [7] . When red blood cells escape into the CSF from a SAH they get broken down into heme and subsequently bilirubin. Therefore xanthochromia is not present from the fresh bloods cells that may come from the process of performing the lumbar puncture (LP). Testing for xanthochromia should not be done less than 12 hours or over 2 weeks from the beginning of the headache [6]. Also the number of red bloods cells can be counted in the first pot of CSF and subsequent pots of CSF collected. If the red cells are present due to the procedure there should be fewer red blood cells (RBC) in the later pots. The house officer who clerked Mr Smith had never seen a Lumbar Puncture before and so the SHO showed him how to perform the procedure.

PERFORMING A LUMBAR PUNCTURE

Lawson Aimee

The Lumbar Puncture

1. It must be ensured that the brain has been imaged and that there are no signs of raised intracranial pressure (ICP). It is not necessary to image everyone prior to LP (i.e., if suspected meningitis and no focal neurology, no LP is needed). Clearly this is different, if there is suspicion of SAH. If ICP is raised the LP could cause herniation of the brain stem.

2. Check the patient's coagulation (particularly important in older patients with co-morbidities).

3. Next the patient is to be consented. Explain to the patient that a thin needle will be inserted into the back to obtain some fluid that can be analyzed in the laboratory. The area will be numbed with local anesthetic before the needle is placed. See below as explanation:

- Benefits of the procedure: Diagnostic
- Risks of the procedure: Headache, pain, bleeding, infection, nerve damage. Warn patient that it is common to feel shooting pain going down either leg (may brush a dorsal root if slightly off centre)

4. Prepare the required equipment (fig 2):

a) Sterile trolley
b) Sharps bin
c) Apron, sterile gloves, eye protection
d) Aseptic solution (chlorhexidine)
e) Local anaesthetic (lidocaine 2%)
f) Needle (small & medium) and syringe
g) Sterile dressing pack
h) Plaster
i) Atraumatic spinal needle and introducer
j) Manometer
k) Four sterile specimen bottle (labeled 1-4)

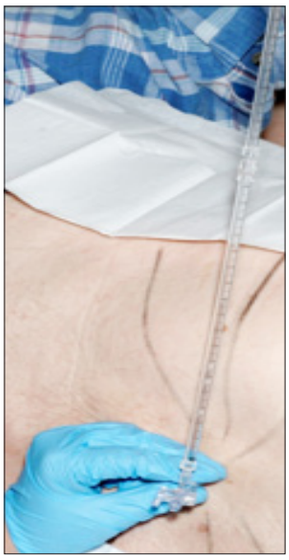
5. Position the patient a lateral position with the bed flat. Encourage the patient to move so that their lumbosacral region is as close to the edge of the bed as possible. Ask the patient to curl up, so that they bend the knees up towards their chest and bend the back forward. This widens the intervertebral spaces making insertion of the spinal needle easier.

6. Palpate the patients back. The gap between L3/L4 is located approximately in line with the iliac crest. Feel between the spinous processes in this plane to find the intervertebral space. Once the gap has been located it is sensible to mark the gap. If a straight line is drawn from the top of the iliac crest to the space and then two more lines transecting this line (see fig 3), when most of the pen is washed away during cleaning, it is still possible to locate the intervertebral space.

7. Next the hands must be washed and sterile gloves applied.

8. Draw up the lidocaine (about 4mls of 2% lidocaine should be sufficient).

9. Prepare the sterile field. Place sterile tissue on top of the patient so that the iliac crest can be palpated (fig 4).



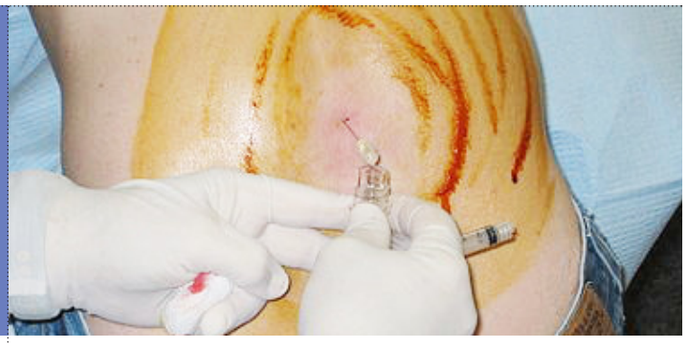
10. Clean the patients back with chlorhexidine (or other appropriate antiseptic solution).

11. Assemble the manometer. This piece of equipment is usually very stiff so turn the tap a few times to ensure it moves freely.

12. Make a bleb in the skin with the lidocaine using the small needle. Using the larger needle inject further into the back. Ensure that you draw back before injecting to ensure that you are not in a vessel. Give the lidocaine a few minutes to work.

PERFORMING A LUMBAR PUNCTURE

Lawson Aimee



Performing a Lumbar Puncture.
Good Clinical Care.

13. Insert the introducer into the intervertebral space. Aim the atraumatic spinal needle with a slight angle towards the umbilicus. Once the introducer is in place insert the spinal needle. If you feel that you have hit bone, the needle will need to be removed and repositioned. You should feel the needle going through the ligaments and then a 'give'. Withdraw the inner core of the spinal needle and watch to see if the CSF is coming out. If not you will need to try adjusting the position of the needle. You should wait for several seconds as the needle is very fine and it can take some time for the fluid to appear. It is considered good practice to use a blunt atraumatic (pencil point) spinal needle, as there is a lower risk of causing CSF leakage [8]. It is acceptable to use the sharper cutting spinal needles as a second line option.

14. Attach the manometer and wait for the CSF to stop ascending up the tube. This is the opening pressure. Then take off the manometer and collect the CSF into 4 pots (about 16 drops in each). When testing for xanthochromia, (this sample must be placed immediately into a black bag and should not be exposed to sunlight). When you have finished collecting the CSF reattach the manometer and find the closing pressure.

15. Gently pull out the spinal needle, followed by the introducer. Put some pressure on the back and apply a plaster.

16. Take some venous blood from the patient to check the glucose level.

17. Encourage the patient to continue lying on their back for at least 20 minutes. They should try to increase their oral intake of liquid for the rest of the day.

18. Safely dispose equipment and sharps. Count the needles that are used and ensure that all have gone into the sharps bin.

19. Send the samples off to the laboratory (fig 5)

Samples: (for this case scenario)

Pot1: micro: cell count

Pot 2: chemistry: protein and glucose

Pot 3: micro: cell count

Pot4: xanthochromia

20. Document the procedure in the notes. Include the colour of the CSF, opening and closing pressure, the intervertebral space used and how many times you had to re-insert the needle.

21. Be aware that the patient may develop a headache hours to days later. The headaches normally settle over a couple of weeks but the best treatment if it does not resolve is a blood patch.

The CSF for Mr S was collected successfully and with no complications. The patient's opening pressure on the manometer was 16cmCSF (10cmCSF-20cmCSF is normal). The closing pressure was 12cmCSF. The CSF was clear. As is often the case, CSF analysis was normal (fig 6). By the time the results had been processed the patient's headache had resolved. The patient was reassured that it was likely that he had experienced a variant of his migraine or a headache related to his exertion. He was advised to be careful with exercise for a few weeks and to go to the GP for treatment if a repeat episode was experienced.

Result:

WCC<2

RCC=10(pot1)

RCC<2 (pot3)

Protein: 40 mg/dl

Glucose: 6mmol/L

Venous Glucose: 9mmol/L

No xanthochromia noted in CSF

Due to the broad differential of a thunderclap headache careful history and examination of the patient is crucial. The history is all-important. All new episodes of thunderclap headaches should get a full examination including blood pressure reading, fundoscopy and thorough examination of the cranial nerves and a peripheral neurological exam. If the CT head and lumbar puncture are negative the need for routine further investigation appears to be controversial in the literature [9,10]. An MRI scan, MR or CT angiography would be the next option. These are often utilized if the patient has a concerning clinical picture (e.g. a cranial nerve palsy, polycystic kidney disease) [11].

PERFORMING A LUMBAR PUNCTURE

Lawson Aimee

Test Yourself - Multiple Choice Questions:

1. What risks should you make the patient aware of when consenting for a lumbar puncture?

a. Weakness of legs
b. Dehydration
c. Headache
d. Sciatica

2. What layers of tissue do you pierce in order to access the CSF (superficial to deep)

a. Skin, subcutaneous fat, external spinal muscle, internal spinal muscle, epidural space, dura mata, arachnoid mata
b. Skin, subcutaneous fat, supra-spinous muscle, infra-spinous muscle, epidural space, dura mata, arachnoid mata
c. Skin, subcutaneous tissue, supra-spinous ligament, inter-spinous ligament, ligamentum flavum, epidural space, dura mater, arachnoid mater

3. How should the CSF be collected if you want to test for a SAH?

a. Use a heparinised collection pot
b. Prevent the sample from being exposed to light
c. Send the collection pots labelled 1 and 3 together for comparison
d. Keep shaking the collection pot to avoid sedimentation

4. What can be done to treat an unresolving headache after a lumbar puncture?

a. Blood patch
b. CSF patch
c. Spinal surgery to repair the dura mata
d. A single suture to close the hole left by the spinal needle

5. When after a thunderclap headache (Subarachnoid haemorrhage), should you perform a lumbar puncture for CSF analysis?

a. As soon as possible
b. Before 12 hours
c. Between 2 days and 1 month
d. Between 12 hours and 2 weeks

Multiple Choice Answers:

1. C The most common complaint after a lumbar puncture is a headache due to loss of CSF.

2. C Skin, superficial fascia, supra-spinous ligament, inter-spinous ligament, ligamentum flavum, epidural space, dura mater, arachnoid mater [12]

3. B It is important to prevent the sample from being exposed to light. The light causes degredation of bilirubin leading to a false negative result [13].

4. A The headaches normally settle over a couple of weeks but the best treatment if it fails to resolve is an epidural blood patch. The patient's own blood is injected into the space where the LP was performed. This will clot and prevent the CSF from leaking.

5. D The test should be done after 12 hours (giving the red blood cells from the SAH sufficient time to be broken down into bilirubin). After 2 weeks it is unlikely that the bilirubin will still be present in the CSF. It is the bilirubin that gives the CSF the yellow tinge known as xanthochromia [6].

References

1. Garza, I. and J. Kirsch, Pituitary apoplexy and thunderclap headache. Headache, 2007. 47(3): p. 431-2.
2. Arnold, M., et al., Pain as the only symptom of cervical artery dissection. J Neurol Neurosurg Psychiatry, 2006. 77(9): p. 1021-4.
3. Wasay, M., et al., Headache in Cerebral Venous Thrombosis: incidence, pattern and location in 200 consecutive patients. J Headache Pain, 2010. 11(2): p. 137-9.
4. Schwedt, T.J. and D.W. Dodick, Thunderclap stroke: embolic cerebellar infarcts presenting as thunderclap headache. Headache, 2006. 46(3): p. 520-2.
5. Jaiser, S.R., A. Raman, and P. Maddison, Cerebral venous sinus thrombosis as a rare cause of thunderclap headache and nonaneurysmal subarachnoid haemorrhage. J Neurol, 2008. 255(3): p. 448-9.
6. Cruickshank, A., et al., Revised national guidelines for analysis of cerebrospinal fluid for bilirubin in suspected subarachnoid haemorrhage. Ann Clin Biochem, 2008. 45(Pt 3): p. 238-44.
7. Dupont, S.A., et al., Thunderclap headache and normal computed tomographic results: value of cerebrospinal fluid analysis. Mayo Clin Proc, 2008. 83(12): p. 1326-31.
9. Moussouttas, M. and S.A. Mayer, Thunderclap headache with normal CT and lumbar puncture: further investigations are unnecessary: against. Stroke, 2008. 39(4): p. 1394-5.
8. Tsen, L.C. and D.L. Hepner, Needles used for spinal anesthesia. Expert Rev Med Devices, 2006.3(4): p. 499-508.
10. Savitz, S.I. and J. Edlow, Thunderclap headache with normal CT and lumbar puncture: further investigations are unnecessary: for. Stroke, 2008. 39(4): p. 1392-3.
11. Davis, S.M. and G.A. Donnan, Thunderclap headache: CT and lumbar puncture but occasionally more! Stroke 2008. 39(4): p. 1396.
12. Boon, J.M., et al., Lumbar puncture: anatomical review of a clinical skill. Clin Anat, 2004. 17(7): p. 544-53.
13. Foroughi, M., et al., Influence of light and time on bilirubin degradation in CSF spectrophotometry for subarachnoid haemorrhage. Br J Neurosurg, 2010. 24(4): p. 401-4.

Authors

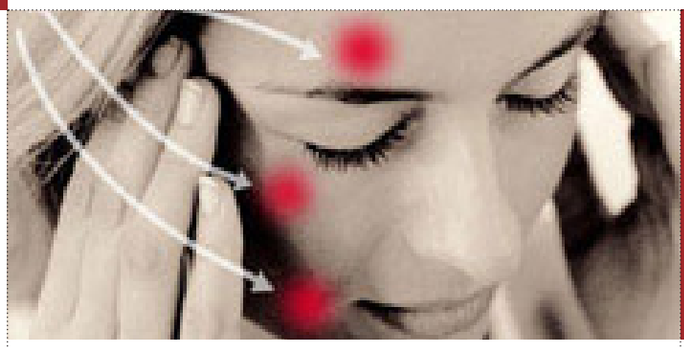
Dr. Aimee Lawson
(F2: Sheffield Teaching Hospital),

Mr A.L.R Michael
(Consultant Spinal Surgeon: Sheffield Teaching Hospital),

Dr. Daniel Blackburn
(SPR in Neurology: Sheffield Teaching Hospital).

TRIGEMINAL NEUROPATHY

Geoff A. Chiu Suresh Shetty, Andrew J. Baldwin



Trigeminal Neuropathy.
Patient Management.

Abstract

We present a case where a patient presented with a trigeminal neuropathy (numbness to the lower right lip and tongue). Initially, the intracranial course was investigated only. The patient returned 14 months later with further symptoms. The extracranial course was then investigated and a lesion was found invading the nerve, by which time the patient had developed multiple metastasis to the lungs and brain. We discuss the course of the cranial nerve and the most common abnormalities leading to a trigeminal neuropathy. When patients present with a cranial nerve neuropathy it is essential to investigate both the intra cranial and extracranial courses of the cranial nerves.

Introduction

The trigeminal nerve (Cranial Nerve V) is the largest of the cranial nerves supplying both motor and sensory function to the scalp and face. Neuropathy can occur anywhere along its pathway both intracranial and extracranial. Most cases are idiopathic and should be classed as idiopathic once all identifiable causes have been eliminated. This article discusses a case where the only the intracranial course of the trigeminal nerve was investigated resulting a missed diagnoses of an extracranial cause. We review the normal anatomy of the trigeminal nerve and the common pathological conditions affecting the trigeminal nerve along its course.

Case Report

In August 2008, a 55 years old gentleman was referred to the Medical Admissions Unit by the Emergency Department with a "pre-syncopal" episode. The patient also noticed "numbness" to the right side of his tongue and lip. Also, there was altered taste sensation to the right side of his tongue. Medically he was a known hypertensive. The medical team examined him: on examination, his blood pressure was 128/87, there were no heart murmurs and an ECG demonstrated sinus rhythm. On cranial nerve examination there was a reduced sensation to light touch over the right lower lip and tongue. The jaw- jerk reflex was reduced. There was no tongue deviation and speech and swallowing were normal. There was normal tone, power, reflexes, co-ordination, and sensation to all four limbs. He was discharged and a magnetic resonance (MR) imaging scan of the brain and stem was organised as an outpatient. The MR only went as far as the skull base, excluding the mandible. This was normal and patient was discharged.

In October 2009, patient was referred to the Oral & Maxillofacial unit, by his dentist with increased pain to the right side of his mandible and complete paraesthesia to the lower right lip. He now reports he has limited jaw movement and suffered with persistent agonizing headaches over the last few months. On examination, the patient had trismus, but there was no apparent intra oral pathology noted. An orthopantogram (OPG) (figure 1) showed an unerupted lower right 3rd molar with 1cm diameter radiolucency around the crown of the tooth. The radiolucency was overlying the inferior dental canal and had irregular ill-defined borders. An urgent biopsy of the deep and superficial tissues from the right retromolar region, a MR scan of head and neck, a chest x-ray and a computer tomography (CT) scan of the thorax abdomen and pelvis was performed. The chest x-ray (figure 3) and CT scan demonstrated multiple "cannon ball" pulmonary metastases. The MR scan of the right mandibular area demonstrated normal overlying mucosa but a high signal in the mandible on T2 weighted images extending from the anterior body up to the angle and into the ascending ramus. This also extended out into the masseteric space. This would account for the trismus, loss of sensation to the lip and taste to the tongue. There were a number of suspicious nodes in the neck. A repeat MR of the brain showed at least 6 new ring-enhanced lesions scattered throughout the brain noted on T2 weighted images (figure 4). They appeared to look like cystic lesions highly suspicious of metastases.

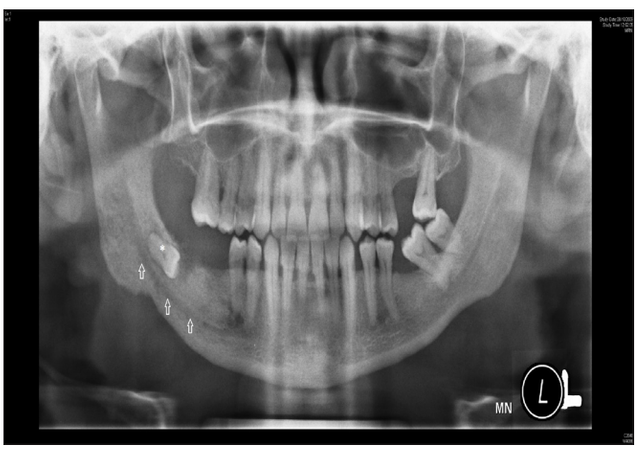


Figure 1
Orthopantogram (OPT) of the patient's jaw. The lower right third molar (*) is lying horizontally. There is an ill defined radiolucency around the crown of the tooth. The lower right third molar is overlying the inferior alveolar nerve (arrows)

TRIGEMINAL NEUROPATHY

Geoff A. Chiu Suresh Shetty, Andrew J. Baldwin

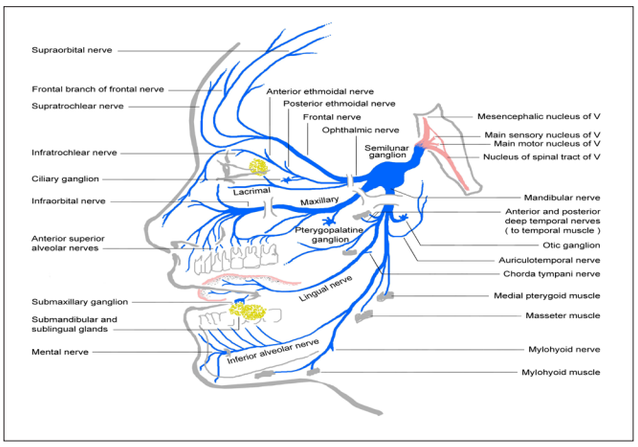


Figure 2

Diagrammatic illustration of the trigeminal nerve.

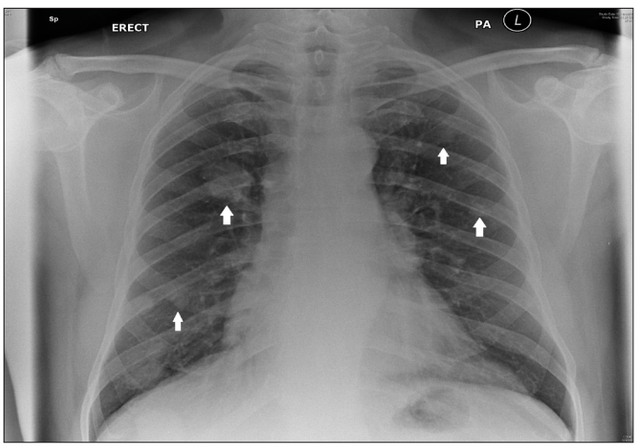


Figure 3

Chest x-ray demonstrating bilateral "cannon ball" metastasis (arrows).

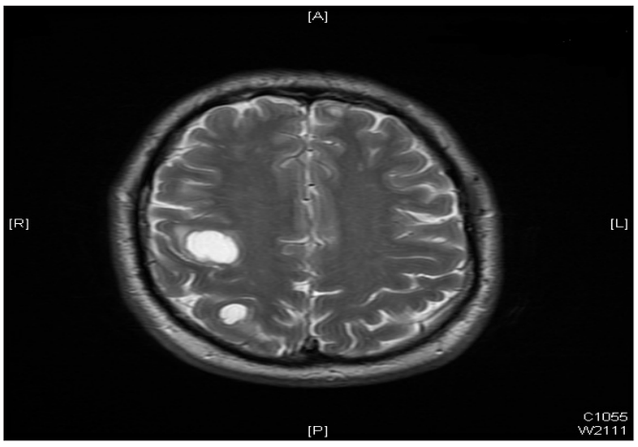


Figure 4

T2 weighted MR (1.5 Teslar) scan of the brain demonstrating two cystic lesions in the right parietal and occipital lobes representing metastasis.

Histology from the right angle of the mandible confirmed normal superficial oral mucosa but the deep tissue of the mandible revealed the lesion as a mucoepidermoid carcinoma. The patient is currently receiving palliative radiotherapy to the brain, followed by palliative chemotherapy.

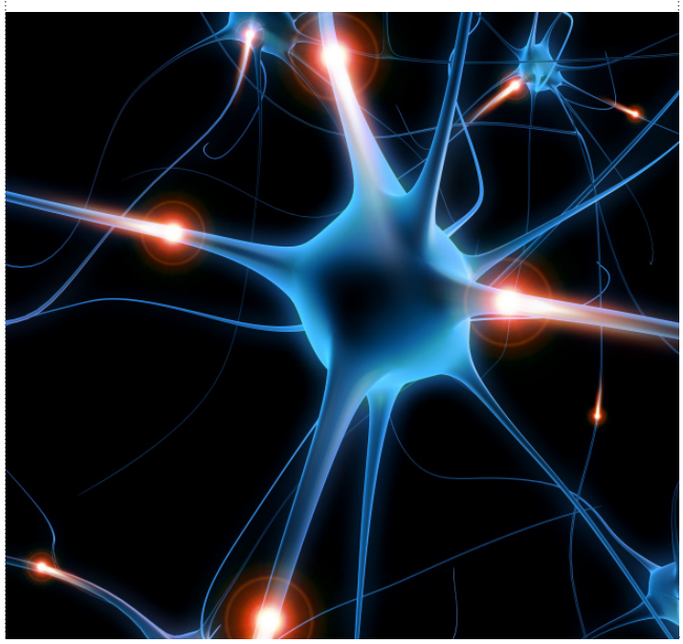
Discussion

Mucoepidermoid carcinoma is the second most common malignant salivary neoplasm. It accounts for 15% of all salivary gland tumours 1-3. It is commonly seen in the parotid gland. The above case is a rare subtype known as a central mucoepidermoid carcinoma being of bony origin, which represents 2-4% of mucoepidermoid carcinomas. The exact pathogenesis is unclear but there are several possibilities including entrapment of submandibular/minor salivary gland tissue during embryonic development, metaplasia of odontogenic cyst epithelium and submucosal gland with intraosseous extension[4].

Mucoepidermoid carcinomas are relatively low-grade tumours and tend to run an indolent course. Treatment of choice is mainly surgical resection. Regional metastasis has been reported in 10-15% of cases 5-7. These tumours rarely show distant metastasis. In this case it is extremely unusual to develop rapid metastatic spread to the brain especially within the 14 months from the first MR scan to the current one.

Anatomy of the Trigeminal Nerve

The trigeminal nerve is the largest of the cranial nerves (Figure 2) with both sensory and motor function. Embryonically, it is associated with and innervates structures from the first brachial arch. The main sensory supply is from the face, scalp, mucus membranes of the sinuses, oral, and nasal cavities. The motor component accompanies the third branch of the nerve and innervates the muscles of mastication (temporalis, masseter, medial and lateral pterygoid), tensor tympani and tensor veli patatini of the soft palate, anterior belly of diagastic muscle and the mylohyoid muscle of the anterior floor of mouth.



TRIGEMINAL NEUROPATHY

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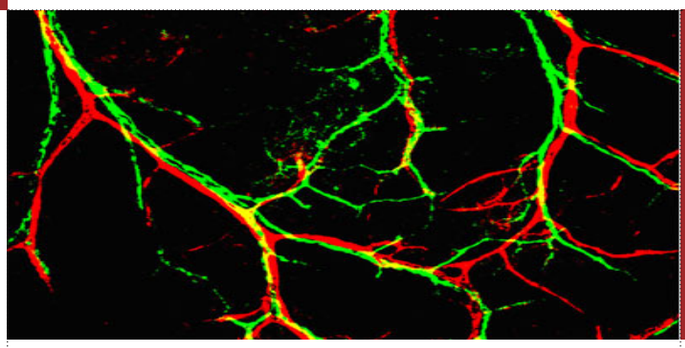


Trigeminal Neuropathy.
Patient Management.

The trigeminal nerve has four central brain stem nuclei: 1) the main sensory nuclei, which mediates tactile sensation, 2) the mesencephalic nucleus which mediates proprioception, 3) the spinal nucleus, which mediates pain and temperature, and 4) the motor nucleus, which provides motor innervation. These nuclei lie in the tegmentum of the lateral pons, along the anterolateral aspect of the fourth ventricle. At the level of the lateral pons, the large sensory and smaller motor root runs through the prepontine cistern and exits as a common truck to enter the Meckel cave. The trigeminal (gasserian) ganglion lies in Meckel cave and contains the cell bodies of the sensory fibres, except those that mediate proprioception. Distal to the trigeminal ganglion, the common root truck trifurcates into its three principal branches, the ophthalmic (v1), maxillary (v2), and mandibular (v3) nerves.

The ophthalmic nerve travels in the lateral wall of the cavernous sinus and exits the skull through the superior orbital fissure. It carries sensory information from the scalp and forehead, the upper eyelid, the cornea of the eye, the nose (including the tip of the nose), the nasal mucosa, and the frontal sinuses. The ophthalmic nerve also mediates the afferent aspect of the corneal reflex.

The maxillary nerve also travels in the lateral wall of the cavernous sinus and exits the skull via foramen rotundum. It enters the pterygopalatine fossa, and its main branch, the infra orbital nerve continues anteriorly through the infraorbital canal to the infraorbital foramen. It carries sensory information from the lower eyelid and cheeks, the nares and upper lip, the upper teeth and gingival, the nasal mucosa, the palate, the pharynx, maxillary, ethmoid and sphenoid sinuses.

The mandibular nerve, contains both sensory and motor fibres, exits the skull through foramen ovale to enter the infratemperol fossa. The main branch, the inferior dental alveolar nerves, enters the medial aspect of the mandible at the level of the lingula (1cm above the occlusal plane) and travels in the body of the mandible to the mental foramen. It carries sensory information from the lower lip, oral mucosa, the lower teeth and gingival, the chin and skin overlying body of the mandible. The lingual nerve, which is a branch of the mandibular nerve, "piggy backs" taste fibres from the tongue which travel to the facial (VII) nerve via the chorda tympani. The motor innervation has already been discussed.

Symptoms of trigeminal neuropathy include facial pain, numbness, masticator muscle weakness, trismus, and neuralgia. On examination there maybe: decreased pain, touch and temperature sensation, and atrophy of the masticatory muscles. One side should always be compared with the other side. Patients with an upper motor neurone disease commonly have a brisk jaw jerk reflex, where as normally it would be absent or slight.

The course of the trigeminal nerve can be divided into four segments: brain stem, cistern, the meckel cave and cavernous sinus and extracranial. Pathology can occur at any point along these segments. Table 1 summarises a list of possible causes of trigeminal neuropathy.

Investigations

As clinical findings of trigeminal neuropathy do not always permit accurate localisation, it is essential to obtain radiographic imaging throughout its entire course. The gold standard is a MR Scan[8,9] T2 weighted images of the brain stem and T1 weighted images should extend from the skull base to the inferior mandible. A high Teslar (T) scan should be used where possible. Currently the standard is 1.5T but 3T is becoming more increasingly available. If bone pathology is suspected as the cause then a CT scan of the facial bones can be done in addition.

Initial single plane views can be obtained to investigate extracranial causes. To look at the upper and middle branches occipital-mental (OM) views can be requested. An OPG demonstrates the course of the mandibular branch, though inferior dental alveolar canal in the mandible.

Conclusion

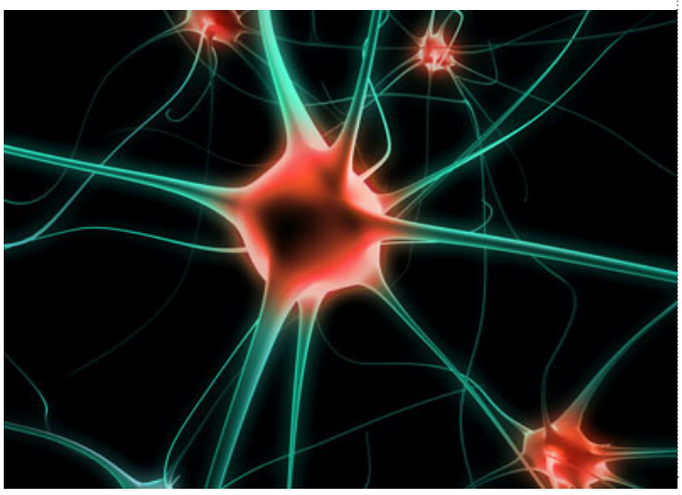
Clinical findings of trigeminal neuropathy patients are difficult to localise[10] When, investigating causes of cranial nerve neuropathy, it is essential to investigate both the intracranial and extracranial courses. In this case, taste and sensation to the tongue were lost, which are carried in the lingual nerve by two fibres of differing origins, facial and trigeminal respectively. This may have pointed to an extracranial cause for the neuropathy. Extending the initial MR scan down from the skull base into the neck may have picked up the lesion compressing the right inferior dental alveolar nerve, third branch of the trigeminal nerve during its extracranial course.

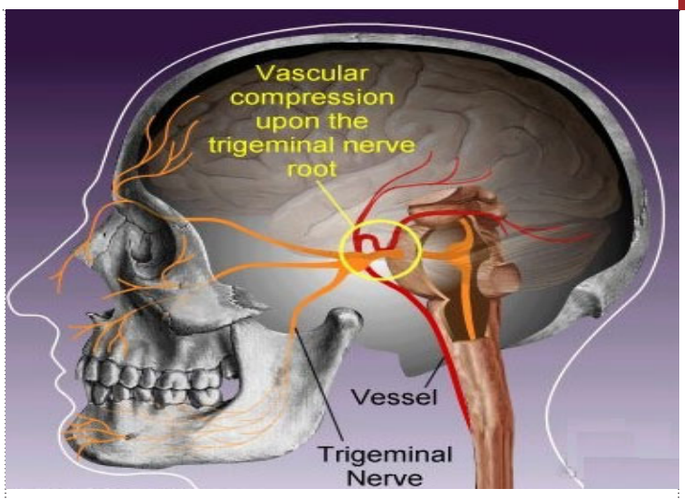
TRIGEMINAL NEUROPATHY

Geoff A. Chiu Suresh Shetty, Andrew J. Baldwin

Segment	Differential Diagnosis
Brain Stem	Multiple sclerosis
	Cerebral vascular accident
	Glioma
	Syringobulbia
	Metastasis
Cistern	Vascular compression
	Arterial-vascular malformation
	Meningioma
	Epidermoid cyst
	Acoustic Neuroma
	Metastasis
	Surgical sectioning
Meckel cave/	Carotid artery Aneurysm
Cavernous sinus	Trigeminal Schwannoma
	Pituitary adenoma
	Base of skull neoplasms
	Metastasis
	Perineural spread of distal tumour
Extracranial	Primary tumour of the head and neck
	Metastasis
	Sinusitis
	Infratemperol fossa space abscess
	Facial trauma
	Iatrogenic

Table 1: List of possible causes that can produce a trigeminal neuropathy.





MCQ's

1: Mucoepidermoid carcinomas of the salivary gland are:
A. most common benign salivary gland tumours
B. most common malignant salivary gland tumours
C. second most common benign salivary gland tumours
D. second most common malignant salivary gland tumours

2: From embryology development which branchial arch does the trigeminal nerve originates from?
A. first
B. second
C. third
D. fourth

3: Which of the following muscles is NOT supplied by the motor branch of the trigeminal nerve?
A. mylohyoid muscle
B. posterior belly of diagastic muscle
C. tensor tympani
D. tensor veli patatini

4: Which foramen does the mandibular branch of the trigeminal nerve travel through to exit the skull?
A. foramen ovale
B. foramen rotundum
C. jugular foramen
D. stylomastoid foramen

5: Which of the trigeminal nerve brain stem nuclei mediates proprioception?
A. main sensory nuclei
B. mesencephalic nucleus
C. motor nucleus
D. spinal nucleus

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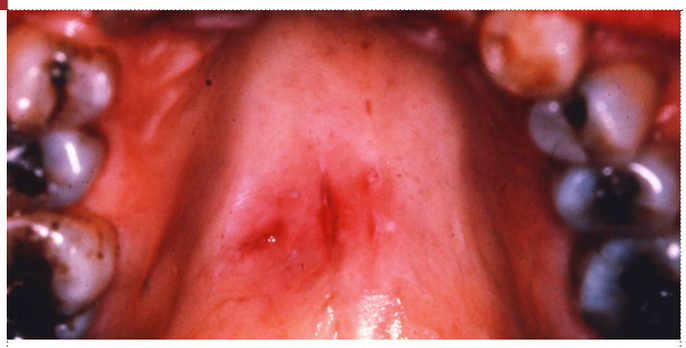

Answers

1. Answer: D.
The most common malignant salivary gland tumour is the adenoid cystic carcinoma which accounts for 13% of salivary gland tumours. This followed by the mucoepidermoid carcinoma. The most common benign salivary gland tumour is a pleomorphic adenoma, which accounts for 40%. This is followed by adenolymphoma benign tumour (10%).

2. Answer: A.
First brachial arch. The facial nerve originates from the second arch. The glossopharyngeal originates from the third arch. The vagus nerve is from the fourth arch.

3. Answer: B.
The posterior belly of diagastric muscle is supplied by the facial nerve. It is the anterior belly that is supplied by the trigeminal nerve.

4. Answer: A.
The maxillary branch of trigeminal nerve exits though foramen rotundum; the glossopharyngeal nerve exits through the jugular foramen and the facial nerve through the stylomastoid foramen.

5. Answer: B.
The main sensory nuclei mediates tactile sensation .The spinal nucleus mediates pain and temperature. The motor nucleus provides motor innervation.

Acknowledgements

I wish to thank the medical illustration department at North Manchester General Hospital for reproducing figure 2 which was initially taken from: **http://www.medicallecturenotes.com/2010/01/cranial-nerves-v.html**

References

1. Lebsack JP, Marrogi AJ, Martin SC. Central Mucoepidermoid Carcinoma of the Jaw with Distant Metastasis. A case report and review of the literature. J Oral Maxillofac Surg. 1990; 48:518-22.
2. Wladron CA, Koh ML. Central Mucoepidermoid Carcinoma of the Jaws: Report of four case with analysis of the literature and discussion of the relationship to Mucoepidermoid, Sialodontogenic, and Glandular Odontogenic Cysts. J Oral Maxillofac Surg. 1990; 48:871-7
3. Baj A, Bertolini, Ferrari S, Sesenna E. Central Mucoepidermoid Carcinoma of the Jaw in a Teenager: A case report. J Oral Maxillofac Surg. 2002; 60:207-11
4. Martinez-Madrigal F, Pineda-Daboin K, Cairaghi O, Luna MA. Salivary Gland Tumours of the Mandible. Annals Diag Pathol. 2000; 4(6):347-353
5. Pires FR, Paes De Almeida O, Lopes MA, Elias da Cruz Perez D, Kowalski LP. Central mucoepidermoid carcinoma of the mandible: report of four cases with long-term follow-up. Int J Oral Maxillofac Surg. 2003; 32:378-382
6. Maremonit P, Califano L, Mangone GM, Zupi A, Guida C. Intraosseous mucoepidermoid carcinoma. Repost of a long-term evolution case. Oral Oncology. 2001; 37:110-113
7. Li Y, Long-Jiang L, Huang J, Han B, Pan J. Central Malignant Salivary Gland Tumors of the Jaw: Retrospective Clinical Analysis of 22 cases. J Oral Maxillofac Surg. 2008; 66:2247-2253
8. Zacest AC, Magill ST, Miller J, Burchiel KJ. Preoperative magnetic resonance imaging in Type 2 trigeminal neuralgia. J Neurosurg 2010; 113:511-515.
9. Goru SJ, Pemberton MN. Trigeminal neuralgia: the role of magnetic resonance imaging. Br J Oral Maxillofac Surg. 2009; 47:228-9.
10. Bennetto L, Patel NK, Fuller G. Trigeminal neuralgia and its management. BMJ. 2007; 334:201-5

Authors

Geoff A. Chiu
Specialty Registrar, Oral and Maxillofacial Surgery

Suresh Shetty
Consultant, Oral and Maxillofacial Surgery
Department of Oral and Maxillofacial Surgery
North Manchester General Hospital
Delaunays Road, Crumpsall, Manchester M8 5RB

Andrew J. Baldwin
Consultant, Oral and Maxillofacial Surgery
Department of Oral and Maxillofacial Surgery,
North Manchester General Hospital, Delaunays Road,
Crumpsall, Manchester M8 5RB

Corresponding Author

Mr Geoff Chiu
Department of Oral and Maxillofacial Surgery
North Manchester General Hospital
Delaunays Road, Crumpsall, Manchester M8 5RB.
Email: geoffchiu@btinternet.com. Tel: 0161 795 2143

EXPANDING THE ROLE OF FOUNDATION YEAR DOCTORS AS EDUCATORS

Jennifer Howes, Kathryn Staley, Natasha Behl and Andrew J Batchelder

Expanding the Role of Foundation Year Doctors as Educators.
Training & Teaching.

Abstract

Peer and near-peer teaching offers benefits to both tutor and student, in terms of acquisition of skills and knowledge. Foundation Year (FY) doctors are well placed to provide academic and pastoral support for final year medical students, however there are multiple demands on their time and little provision is made for an educational role. Here we describe a "near-peer"-led initiative, comprising bedside teaching and speciality-based lectures. The programme was conceived and developed by FY doctors to assist their undergraduate colleagues in preparation for their final examinations. This framework serves to provide junior doctors with opportunities to teach as part of a structured programme that extends across the East Midlands region. In this way, we hope it may act as a guide to others who consider establishing a similar initiative in their region.

Introduction

Peer-led and peer-assisted learning have been shown to be effective educational methodologies, serving to create a 'comfortable environment in which students can absorb clinical knowledge, whilst fostering the development of confidence'.[1] Foundation Year (FY) doctors, with the memory of their own final examinations still fresh in their minds, are ideally placed to provide teaching for senior medical students and can also empathise with the demanding and often stressful period prior to final examinations. In addition, reciprocal benefits have also been described; peer teaching serves to enhance cognitive, psychomotor and affective development of tutors and increases collegial behaviour.[2]

The GMC's Good Medical Practice: Teaching and training, appraising and assessing states that, 'If you are involved in teaching, you must develop the skills, attitudes and practices of a competent teacher' and also 'be honest and objective when assessing colleagues'.[3] Opportunities to present and direct small group teaching sessions enable FY doctors to develop and explore teaching styles and evolve into effective educators through regular self-appraisal and feedback from students.

Based upon these principles and drawing on previous experience of peer teaching as undergraduates, a group of FY doctors from Leicester University Medical School established a revision programme for students preparing for their medical title fight. The aim of the programme was to create a formalised framework for regional near-peer teaching, which would accommodate the demands of FY doctors, who is keen to teach with the benefits of providing tailored teaching to meet the needs of individual students. The programme has continued to develop in response to feedback from participating doctors and students. Here, we describe the structure about programme and discuss the difficulties which we have encountered. In this way, we hope this article acts as a guide to other FY doctors with an interest in medical education, which may consider implementing a similar framework within their region.

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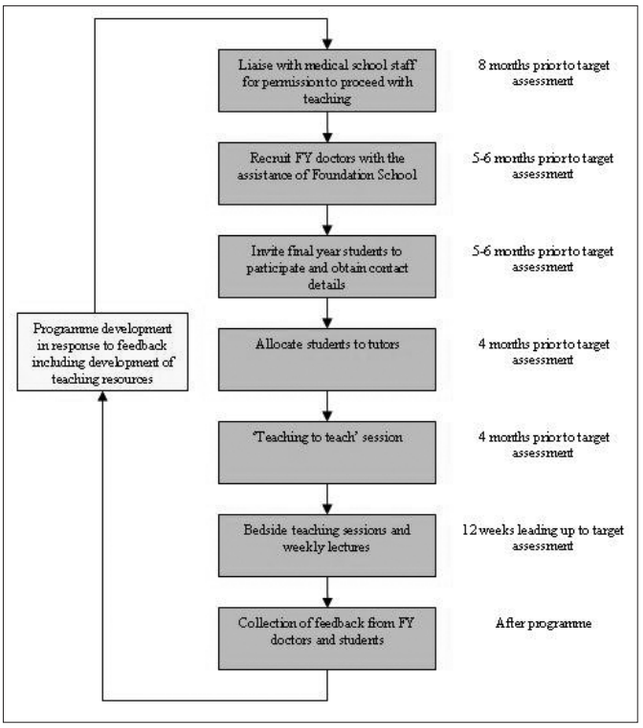
The Programme

All final year medical students were invited to take part and responders were paired in a 2:1 ratio with FY doctors, who had expressed an interest in teaching. The student-tutor pairings continued for a three months period from January to March prior to the university's final examinations. The allocations included students on clinical placements at local university teaching hospitals and students rotating through three regional district general hospitals. The framework for near-peer teaching sessions sought to achieve a balance between autonomy of tutors and quality assurance of the teaching delivered. A short "Teaching to Teach" course was held prior to the start of the bedside teaching. The course was delivered by consultants involved in undergraduate assessment and included sessions on: The organisation and assessment criteria of the final examinations; a review of the programme and its aims; and an introduction to learning styles and teaching techniques. A timeline for the programme is included in Figure 1.



Basic structure for establishing a revision programme.

FY doctors were encouraged to meet their students on a weekly basis but scheduling was flexible to allow for clinical commitments and other programmed activities. The content of each session was determined by needs identified by participating students, with emphasis on consultation skills and problems solving in the context of investigation and management. Weekly speciality-based revision lectures served as an adjunct to the clinical teaching. These were delivered by FY doctors, who had either completed a placement or expressed a personal interest in the subject areas. Lecture materials, including audio recordings and power point presentations, were made available on the university's virtual learning environment to allow access for students who were unable to attend.

Summary of Results

The programme is now entering its third successive year. Last year, 125 students and 59 doctors participated at three university teaching hospitals and one district general hospital. This year, we have already received responses from 98 FY doctors who are keen to be allocated students and this serves to demonstrate the willingness of junior doctors to develop their teaching skills. Each year qualitative feedback is collected from both students and FY doctors through online survey comprising items rated on Likert scales and free text comments. Previously, 100% of FY doctors who completed the questionnaire stated that they found teaching a useful experience which aided their own learning; one junior doctor commented, "A lot of my time is spent doing clerical rather than clinical jobs and therefore the chance to give clinical teaching improved my teaching skills, reinforced my clinical knowledge and was a refreshing and rewarding experience". 82.6% of students who completed the questionnaire reported they felt more confident approaching the final professional examinations as a result of the teaching initiative. Additionally, 52.2% of students also reported that their tutors were a source of pastoral support.

EXPANDING THE ROLE OF FOUNDATION YEAR DOCTORS AS EDUCATORS

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Problems Encountered and Areas for Future Development

Inevitably, co-ordinating an accessible revision programme of a large scale is a demanding and challenging endeavour. Whilst every effort was made to pair FY doctors with students on placements at their base hospital, discrepancies of numbers meant that this was not always possible. This meant some students had to travel across sites to attend teaching sessions. Anecdotal feedback suggested that students considered the benefits of additional teaching outweighed this inconvenience. Many FY doctors reported that clinical commitments made it difficult to meet students for bedside sessions within their working day. Nevertheless, the vast majority of tutors were willing to undertake teaching activities during their personal time. In recognition of the demands on time, future participating FY doctors have been advised of this when committing to the programme.

We encountered difficulty setting up near-peer teaching in regional district general hospitals. FY doctors working in many of these localities are training in other deaneries and rotate to different hospitals regularly. Consequently, establishing contact with enthusiastic FY doctors in these centres can be difficult and satisfying the rigours of quality assurance can be challenging. This year, we have appointed co-ordinators at each district general hospital, which has largely overcome this problem.

Objective Structured Clinical Examinations (OSCE's) commonly include stations on management of acute scenarios and counselling patients. Feedback from participants has identified this as an area where students would appreciate more support in preparing for their assessments. Consequently, we are currently creating a library of electronic teaching aids for tutors, including case scenarios, electrocardiographs and imaging studies

Summary

Over the last three years, our programme has continued to develop and expand within the region and we have endeavoured to ensure that we maintain the quality of the teaching as this occurs. We have also noted that a large volume of the student cohort who then go on to work in the region are now actively seeking instructor positions within the scheme, creating a sustainable source of enthusiastic teachers.

References

1. Hudson J.N., Tonkin A.L. Clinical skills education: Outcomes of relationships between junior medical students, senior peers and simulated patients. Medical Education 2008; 42 (9): 901-8.
2. Secomb J. A systematic review of peer teaching and learning in clinical education. Journal of Clinical Nursing 2008; 17 (6): 703-16.
3. Tomorrows Doctors. General Medical Council. London, 2003.

Authors

Dr JA Howes, MBChB.
FY2 Doctor in Emergency Medicine, Kettering General Hospital, Rothwell Road, Kettering, Northamptonshire, NN16 8UZ.

Dr KG Staley, MBChB, BSc, BA. ST1
Doctor in Paediatrics. Department of Child Health, Robert Kilpatrick Clinical Sciences Building, Leicester Royal Infirmary, Infirmary Square, Leicester, LE1 5WW

Dr N Behl, MBChB. FY2 Doctor in Surgery.
Leicester Royal Infirmary, Infirmary Square, Leicester, LE1 5WW

Dr A Batchelder, MBChB (hons) BSc (hons).
CT1 Doctor in Surgery. Leicester Royal Infirmary, Infirmary Square, Leicester, LE1 5WW

Address for correspondence

Kettering General Hospital
Rothwell Road, Kettering,
Northamptonshire, NN16 8UZ.

CASE BASED DISCUSSION – ACUTE NEUROMUSCULAR WEAKNESS

Dariush Saeedi, Vijay Bangar

Case Based Discussion – Acute Neuromuscular Weakness. Patient Management.

Abstract

A 37 years old lady presented to the medical admissions unit (MAU) with a seven-day history of increasing pain, limb weakness and altered sensation in her hands and feet. This case based discussion focuses on the assessment of acute neuromuscular weakness, and the relevant differential diagnosis, investigations and management.

Case History

A 37 years old person normally fit and well midwife presented to the medical admission unit (MAU) with generalised pain, throbbing in character, with a severity of 7 out of 10. This was particularly severe in her lower back and was exacerbated by slight movement or touch. This had been getting worse over the last 24 hours prior to admission, and now was so severe she was unable to mobilise without significant difficulty. Over the past 7 days she reported altered sensation starting in her hands, specifically numbness pins-and-needles and pain. These sensations later developed in her feet and gradually progressed to the symptoms described above. At the time of admission felt that her feet were particularly painful and felt 'heavy'.

The patient also reported a frontal headache of similar character, which had been getting worse over the last 24 hours, and complained of photophobia and pain on eye movements.

What Do You Want To Do Next?

• As with the assessment of any patient, a full and thorough history and examination is essential to aid early diagnosis and guide management.

• In this case, the information given thus far suggests the patient is presenting with an acute motor and sensory neuropathy. What salient points should be elicited from the history in such a patient? Below are some important factors to consider:

1. The presenting complaint/history of the presenting complaint

Finding out more about the character and nature of the pain may provide clues to the underlying pathology. For example, a neuropathic-type shooting pain may suggest nerve root compression. The location, distribution, and radiation of the pain and any altered sensation, i.e., sensory level, should be carefully documented as this can highlight the location of a lesion. It is also important to ascertain if there are any associated symptoms such as fevers, rigors, rashes, bladder or bowel dysfunction, visual disturbance, or vomiting.

These factors may help differentiate infective causes from mechanical, such acute cord compression for example. It is also important to document any confusion or unusual behaviour if present.

2. Past medical history

It is important to establish whether there has been any preceding illness or any co-existing disease. It may also be useful to ascertain whether the patient has had a similar episode before.

3. Drug history

Carefully document any medications and adverse reactions.

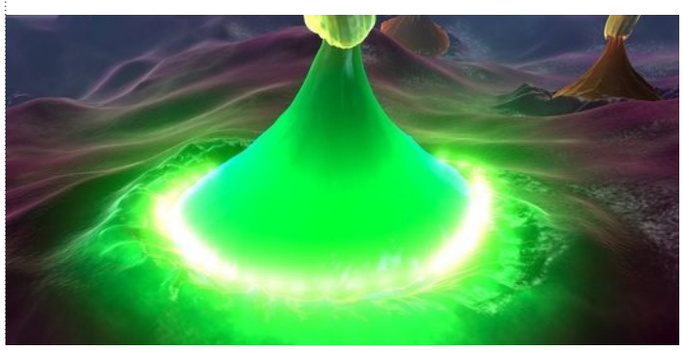
4. Social history

A good occupational history is important, enquire whether there has been any exposure to heavy metals or aerosolised neural tissue (i.e., abattoir workers). What is her normal alcohol consumption and is there any drug use? Recent travel should also be documented, including camping or stays in wooded areas.

5. Family history

Are there any inherited neurological or metabolic disorders? Are there any family members with similar symptoms?

Prior to this episode the patient stated, she was relatively well, although had not felt completely back to normal after suffering a vomiting and diarrhoeal illness 2 weeks previously. Some members of her family had similar symptoms. She was on no regular medication and her past medical history included irritable bowel syndromes and carpal tunnel decompression several years ago. She normally worked as a midwife, did not smoke, and had only moderate alcohol intake. There was no relevant family history.



CASE BASED DISCUSSION – ACUTE NEUROMUSCULAR WEAKNESS

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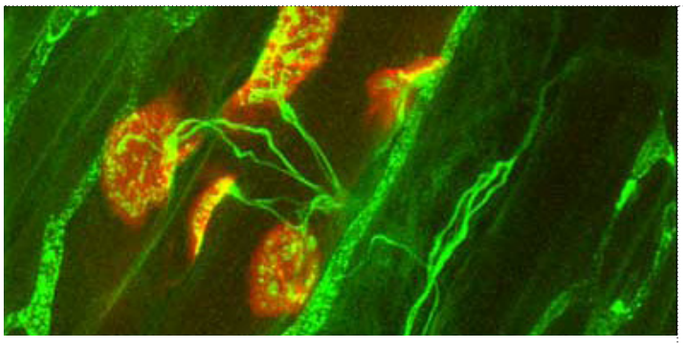

What do you want to do next?

· Careful systematic examination is crucial in determining neurological disease; a full examination including cranial and peripheral nerves should be carried out, as well as a general examination of the main systems.
In particular...

1. Try to ascertain, if any deficit is peripheral or proximal e.g., asking the patient to rise from sitting to standing without using his/her arms may help differentiate proximal and distal weakness.

2. What is the distribution of weakness? Is it symmetrical?

3. Carefully assess the deep tendon reflexes; absence or abnormality may be a hallmark of the underlying disease.

4. Are the cranial nerves involved?

5. Is there any obvious evidence of autonomic dysfunction?

6. Is the bladder and bowel functioning normally?

7. Is there any sensory deficit? What is the distribution?

8. Are there any cerebellar signs?

9. Are there signs of other system involvement?

10. Are there signs of infection?

Examination

The patient was alert, orientated, and a febrile. Neurological examination revealed power 3/5 in the upper and lower limbs, sensation was reduced (to light touch) over both feet. There was also marked tenderness generally, especially over the face. There were no other sensory abnormalities. Tone was normal. Reflexes were brisk in the ankles and knees, with down-going plantars. There were no cranial nerve deficits. The examination was otherwise unremarkable. Routine observations were within normal ranges.

What are the differential diagnosis for this patient?

Please see table 1 for an overview of differential diagnoses relevant to the presenting complaint:

Muscle disorders	*Hypokalaemia*
	Hyperkalaemia
	Inflammatory myopathy
Myelopathic disorders	*Acute transverse myelitis*
	Focal compression
	Poliomyleitis
	Rabies
	Enterovirus
Peripheral neuropathies	*Guillain-Barre Syndrome*
	Heavy metals (arsenic, lead)
	Vasculitic neuropathy
	Porphyria
	Diptheria
	Lyme disease
Neuromuscular junction disorders	*Myasthenia gravis*
	Snake venom
	Botulism

Table 1: Differential diagnosis of acute neuromuscular weakness

What is the likely cause of the patient's symptoms?

After taking a history and examination, the likely diagnosis of this patient was thought to be an acute motor and sensory neuropathy, initially Guillain-Barre syndrome (GBS). The case was discussed with a neurology consultant. Although demonstrable weakness in the peripheries was evident on examination, the combination of brisk knee and ankle reflexes (absent reflexes are usually a characteristic of GBS) with severe back pain meant that the suspicion of cord compression was raised. This then became the primary differential diagnosis after the post-take ward round.

Factors from the history and examination, which were in keeping with GBS are highlighted below:

· Weakness
· Sensory involvement
· History of suspicious preceding illness (gastroenteritis)

CASE BASED DISCUSSION – ACUTE NEUROMUSCULAR WEAKNESS

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Case Based Discussion – Acute Neuromuscular Weakness. Patient Management.

However, several factors from the case are less frequently encountered and not in keeping with 'classical' GBS. Therefore this raised the index of suspicion for other causes:

- Brisk knee and ankle reflexes (usually absent)
- Severe generalised pain, worse lower back region
- Rapid onset (sudden deterioration last 24 hours)

What do you want to do next?

Investigations:

1. Bloods

- Full blood count, C-reactive protein (CRP) – useful for excluding infection.
- Urea and electrolytes – some patient with GBS may produce inappropriate levels of anti-diuretic hormone1. Also check the potassium level, which may lead to the root cause (i.e., in hyperkalaemic periodic paralysis).
- Liver function tests - elevated in up to one third of patients[2]
- Erythrocyte Sedimentation Rate (ESR)
- Clotting screen
- Vasculitis screen
- Antibodies (i.e., GD1a antibody) – may be of limited use in diagnosis but may have a prognostic role[3]

It should be noted that in GBS routine blood tests may all be normal4 and the majority of tests are useful in largely in excluding other diagnoses (i.e., infection).

2. Cerebrospinal Fluid (CSF)

Patients with GBS may have a raised protein level in the absence of a raised cell count. CSF studies are also useful to check for oligoclonal bands (i.e., in transverse myelitis), and again for any evidence of infection.

3. Nerve Conduction Studies (NCS)

Nerve conduction studies can be very useful in confirming the diagnosis of GBS, as they are abnormal in approximately 85% of patients5. Focal conduction block is characteristic, and F- waves may be prolonged3.

4. Cardiac Monitoring/ Electrocardiogram (ECG)

In certain subtypes of Guillain-Barre syndrome there may be autonomic dysfunction, with cardiac conduction block and arrythmias.

5. Magnetic Resonance Imaging (MRI)

If there is clinical suspicion of spinal cord compression, MRI is the preferred modality. Selective anterior nerve root enhancement may be strongly suggestive of GBS2.

Results

The patient's blood results were within normal ranges. The only abnormality found in the CSF studies was a protein level of 1.5 (normal range 0.15-0.45) with an absent white cell count, consistent with GBS. MRI spine and head were grossly normal with no obvious focal pathology. Nerve conduction studies showed results suggesting a demyelinating polyneuropathy, consistent with Guillain-Barre syndrome.

What do you want to do next?

Management of Guillain-Barre Syndrome

This can be divided into general supportive measures and definitive treatment:

1. General supportive measures

- Inform the anaesthetist and the Intensive Care Unit (ICU) - approximately 30% of patients with GBS will require treatment in ICU4.
- Early involvement of specialists (i.e., neurology specialists, physiotherapists, occupational therapists).
- If suspected GBS, start regularly measuring spirometry – monitor for signs of respiratory compromise, if vital capacity falls below 20ml/kg transfer to ICU.
- Cardiac monitoring – telemetry may be used.
- Pain management – this is often difficult and may require analgesics such as gabapentin or carbamazepine6.
- Thromboprophylaxis.

CASE BASED DISCUSSION – ACUTE NEUROMUSCULAR WEAKNESS

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2. Definitive treatment

- Intravenous immunoglobulins. These are thought to neutralise circulating antibodies and down regulate pro-inflammatory cytokines[2]. This method is relatively easy to administer and has fewer complications than plasma exchange[1].
- Plasma exchange. This involves the exchange of the patients' plasma for a substitute, such as albumin solution. This is thought to remove auto antibodies and immune complexes from the serum.
- Both methods have been shown to reduce recovery time by as much as 50%[2]. A Cochrane analysis of three trials involving IV immunoglobulin therapy showed that the efficacy of treatment was equivalent to that of plasma exchange[7].

Progress Report

On day 5 of the patient's admission, IV immunoglobulins were administered. This was continued for the duration of 5 days. By day 9 her symptoms were significantly improved with less reliance on opiate analgesia. On day 10 post-admission power had improved in all four limbs with sensation returning and she was beginning to mobilise freely. On day 12, the patient was mobilising as per normal, and not on any analgesia and was discharged with neurology follow up. FVC had remained stable throughout the admission and the patient was managed on a general medical ward.

The patient was followed up in the neurology out patient clinic 2 months after discharge. She felt that she was '90% better'. On examination her power was 4+ out of 5 lower limbs, and 5 out of 5 upper limbs. Her tendon jerks were present but slightly more pronounced on her left side. Sensation was normal. The patient was receiving regular physiotherapy and was undergoing occupational therapy at home. She is due to be seen again in clinic to monitor progress.

Discussion

In this case, we have discussed, the assessment, differential diagnosis, investigations, and management of patient presenting with acute neuromuscular weakness, specifically in this case GBS.

What is GBS?

GBS is a peripheral polyneuropathy, which causes acute neuromuscular weakness. It is the common cause of acute neuromuscular paralysis3, and has a mortality of approximately 10%[8]. GBS was thought initially to describe a single disease, however, it is now recognised to be a spectrum of at least 3 different disorders1. The underlying pathology common to the group is the mistreated immune driven response to host nerve cells, usually directed to the myelin sheath as in the acute inflammatory demyelinating polyneuropathy (AIDP) or less commonly, the axon itself, acute motor axonal neuropathy (AMAN). Two thirds of GBS cases occur following a gastrointestinal tract or upper respiratory tract infection[3]. Commonly associated pathogens include Campylobacter jejuni and cytomegalovirus.

Beware uncommon presentations

Misdiagnosis is common and can be fatal1 due to rapidly ensuing respiratory failure. It is therefore important to be aware that GBS may present, as in this instance, with uncommonly encountered symptoms and signs. Classically GBS presents with progressive symmetrical ascending limb weakness, paralysis, and areflexia. Most patients reach a nadir in clinical functioning within 2-3 weeks, somewhat dissimilar to the timescale of our patient. Sensory symptoms may be present, and the autonomic system may be affected. Cranial nerve and pure motor nerve involvement is not uncommon[2].

It is important to be aware therefore that initial neurological symptoms in GBS can vary from patient to patient[4], and some of the symptoms and signs demonstrated in our patient were arguably more vague and non-specific than the classical picture. Findings that were in keeping included the identification of a vomiting and diarrhoeal illness 2 weeks prior to admission. Such a finding may raise the clinical suspicion of GBS above other potential diagnoses. Pain is another fairly common feature, it may be pronounced but it is often overlooked[9]. It can be most severe in the shoulder girdle, back and posterior thighs[4]. This was consistent with our patient, who was heavily reliant on opiates for symptom control.

Features less frequently encountered in GBS include the deep tendon reflexes demonstrated in our patient. Normal reflexes or hyper-reflexia in GBS is unusual[10], however, it seems to be a feature in certain less common subtypes of GBS (such as the AMAN type). In the case of our patient, the presence of brisk reflexes and back pain initially raised the suspicion of cord compression as the underlying pathology. This demonstrates that even despite early specialist involvement, the diagnosis may still be equivocal. However, the patient was still duly investigated for other causes with a lumbar puncture and bloods, and had her FVC monitored regularly. Because of the high risk of respiratory compromise in cases of acute neuromuscular weakness, any suspicion however slight should prompt close monitoring until the disorder can be reasonably excluded.

CASE BASED DISCUSSION – ACUTE NEUROMUSCULAR WEAKNESS

Dariush Saeedi, Vijay Bangar

Summary

The most important features in the management of this patient's case included a thorough history and examination, which were essential in aiding the early recognition of GBS, and excluding other relevant causes (i.e. cord compression and infection). This case also highlights the necessity of a good understanding of the differential diagnosis and awareness of the relevant investigations. The importance of recognising acute neuromuscular weakness at an early stage is paramount, because of the risk of rapidly ensuing serious complications. The prompt involvement of seniors, specialists and the multi-disciplinary team is vital and close monitoring of the patient is required if there is any level of suspicion. Doctors should be aware of the ease of misdiagnosis and the less common presentations of acute neuromuscular weakness. As in this case, follow up plays a crucial role in checking progress and identifying any residual deficit and again, relies on the involvement of the multi-disciplinary team in the community to provide rehabilitation and support post discharge.

Self-Assessment Best Of Five Questions:

1. A 42 years old man presents with a 10 days history of altered sensation in his fingers and feet. He describes heaviness of his feet over the last 2 days, and reports some double vision and blurring. He was otherwise well with no significant medical history, although he describes an episode of coryza, fevers, and cough 3 weeks ago, which has now largely resolved. What is the most useful investigation in confirming the underlying diagnosis?

a. Magnetic resonance imaging studies of the spinal cord
b. Nerve conduction studies
c. Auto-antibody screen
d. Stool sample for c jejuni
e. Lumbar puncture

2. The 42 years old man described above is admitted to a general medical ward. What is the single most important factor in the ongoing management of the patient?

a. Intravenous immunoglobulin therapy
b. Plasma exchange therapy
c. Regular review of urea and electrolytes
d. Cardiac monitoring
e. FVC monitoring

Answers

1) b

This gentleman's symptoms are strongly suggestive of Guillain-Barre Syndrome (GBS), and subsequently all the listed investigations may have a role in excluding other causes and supporting a diagnosis. However the most useful test is the nerve conduction studies. These are rarely normal in patients with GBS and features such as a delay in F-waves, decreased action potentials and conduction block are highly suggestive[11].

2) e

Although cardiac monitoring is necessary, and both IV immunoglobulins and plasma exchange are two potential methods of treatment, by far the most important aspect of management is the regular monitoring of the patients forced vital capacity. Mortality in GBS can be as high as 10%1 with rapidly ensuing respiratory failure being a major contributing factor. Early anaesthetic involvement is crucial, if the vital capacity falls below 20mls/kg transfer to the intensive care unit is necessary3.

References

1. Winer JB. Clinical review. Guillain-Barre Syndrome. BMJ 2008;337:a671
2. Miller AC, Rashid RM, Sinert DO. Guillain-Barre Syndrome. Emedicine online article 2010; http://emedicine.medscape.com/article/792008-overview
3. Manji H, Connolly S, Dorward N, et al. Oxford Handbook of Neurology. New York: Oxford University Press 2007;202-204.
4. Shields RW, Wilbourn AJ. Demyelinating Disorders of the Peripheral Nervous System. In: Goetz CG ed. Textbook of Clinical Neurology, 2nd edn. Elsevier Science (USA) 2003;1083-1101.
5. Hadden RD, Cornblath DR, Hughes RA, et al. Electrophysiological classification of Guillain-Barré syndrome: clinical associations and outcome. Plasma Exchange/
Sandoglobulin Guillain-Barré Syndrome Trial Group. Ann Neurol 1998;44:780-8.
6. Hughes RA, Wijdicks EF, Benson E, et al. Multidisciplinary Consensus Group. Supportive cares for patients with Guillain-Barré syndrome. Arch Neurol 2005;62:1194-8.
7. Hughes RA, Raphael JC, Swan AV, van Doorn PA. Intravenous immunoglobulin for Guillain-Barré syndrome. Cochrane Database Syst Rev 2001;(2):CD002063.
8. Rees JH, Thompson RD, Smeeton NC, Hughes RA. Epidemiological study of Guillain-Barré syndrome in south east England. J Neurol Neurosurg Psychiatry 1998;64:74-7.
9. Ruts L, Drenthen J, Jongen JLM, et al. Pain in Guillain-Barré syndrome, A long-term follow-up study Neurology October 19, 2010;75:439-1447.
10. Somarajan A. Guillain-Barre syndrome with brisk reflexes-another variant. Neurol India 2006;54:215-216.
11. Winer JB. Guillan-Barre Syndrome. Mol Pathol. Dec 2001;54(6):381-5

Authors

Dr Dariush Saeedi
Foundation Year 2, General Practice, Halifax

Dr Vijay Bangar
Consultant Physician, Diabetes and Endocrinology,
Calderdale Royal Hospital, Halifax

CLINIC LETTERS – PARENTS PERSPECTIVE

Sethuraman S, Ahmed M

Clinic Letters – Parents Perspective.
Professionalism In Practice.

Abstract

Background
Department of Health has published numerous documents reiterating the benefits of copying clinic letters to patients. The National Health Service plan recommends that patient must receive a copy of the clinic letters. This practice is believed to improve the communication between the doctor and patient.

Aim
To determine the opinion of parents/carers on the value of clinic letters received following their children's outpatient clinic consultation.

Setting
A paediatric outpatient clinic in a district general hospital.

Methods
200 parents whose children had at least one previous outpatient clinic visit and had subsequently received a clinic letter were asked to complete a questionnaire.

Results
94% of the parents wanted to continue to receive a copy of clinic letters. 96% always read the letters. 92% felt that the whole of the letter was useful. 81% felt that the all the information in the letter was accurate. Internet was the most common source of information, if parents did not understand parts in the letter. 90% saved and filed a copy of the clinic letter for future reference.

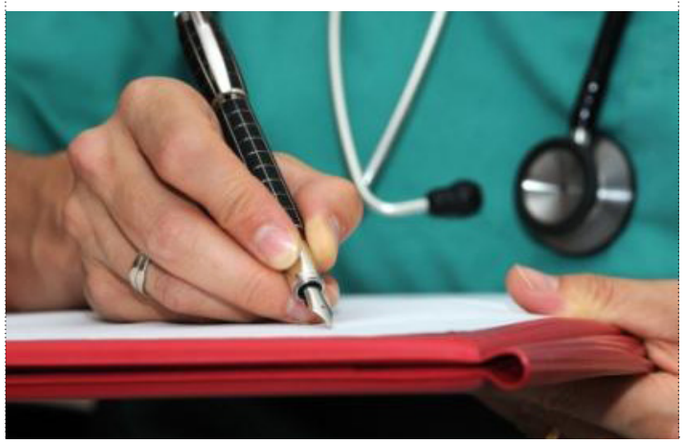
Conclusion
Nearly all the parents appreciate receiving clinic letters. Most of parents read and preserve a copy of clinic letter. Internet is the most common source of information even after seeing a specialist. Further studies are required to evaluate its financial implications and alternative ways of communication.

Background

As part of National Health Service (NHS) Plan 2000, the Department of Health for the first time, as an endeavour to give patients more information over their care, published a directive to copy all letters about patients between health professionals to patients. Since April 2004, patients should automatically receive copies of correspondence between health professionals. There has been overwhelming evidence that this practice improves the communication between the doctor and the patient. This is widely welcome among adult patients in different specialities.

Information sharing between the health professional and the patient/carer in a paediatric clinic setting can be affected by multitude of factors like time restriction, parental anxiety, complexity of the condition or simply because of an unsettled baby or a disruptive toddler running around during clinic consultation. Parents are the source of information to other carers of the child like nursery, school, and child minder. This information includes: diagnosis of a medical condition, investigation plan/results, medication and/or changes to the existing treatment.

The role of clinic letter multiplies in these situations and becomes crucial for parents/carers. It has been shown that patients are more satisfied over their clinic appointment, if they receive copy of letter and a high percentage of them read the letters and are in favour of receiving the letters . There is paucity of data on the work done in paediatrics in connection with parents' reflection on the content of clinic letters and their use of the letters.



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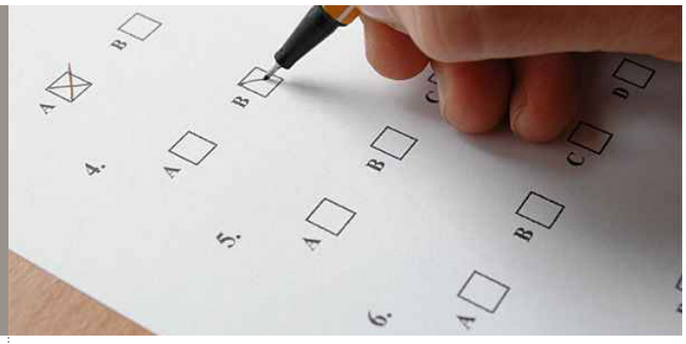

Clinic Letters – Parents Perspective.
Professionalism In Practice.

Aims

We sought to determine the opinion of parents/carers on the value of clinic letters received following their children's outpatient clinic consultation in a district general hospital setting.

Methods

Specifically designed questionnaires were distributed to 200 willing parents/carers who attended paediatric outpatient clinic. The questions were aimed towards achieving parent's opinion on receiving clinic letter, its content, ease of language used, and its subsequent use. We asked them about their choice of alternate source of information. The inclusion criteria were all children who had received a clinic letter following at least one previous clinic appointment. Since 2004, our practice involves copying General Practitioner's (GP) letter to parents automatically following clinic appointment.

Results

The mean age of children in the survey was 6.6 years (79 months). 33% responders had more than 10 appointments, 17% had 6 – 10 appointments, 28% had 3 – 5 appointments, 13% had 2 appointments and 9% had 1 appointment before the survey.

The questions had variable response rate with text responses scoring considerably lower than fixed responses. 94% always wanted a copy of the clinic letter while remainder preferred to leave it optional. Most common reason in favour of receiving letters was to use it as a reminder of information given to them in the clinic. Other reasons included keeping a record for future reference and better understanding of information.

Half of the parents favoured to receive the letter within a week and ~1/3 wanted within 1–2 weeks. 96% always read the letters. 92% felt that whole letter was useful. The specific parts of the letter, which parents/carers found useful were doctor's impression, treatment plan, and follow-up.

81% strongly agreed that information in the letter was accurate and 84% concurred that they could understand all of the information in the letter. Ringing the consultant's secretary was the most common action that parents/carers would like to do, if they found inaccurate information in the letter. Internet (64%) was the most common source of information for parents, if they could not understand information, words, or terminology in the letter. This was followed by enquiring from GP, friends, family and the paediatric team.

93% parents/carers felt that receiving letters was useful and 90% would file or preserve the letters for future reference. The most common reason to share the clinic letter with others was in connection with school/educational matters. Other individuals included family, friends, health visitors, and other professional involved in the child's care. However, one third had never shown the letter to others.

Discussion

This survey has revealed parent's perspective of clinic letters and its utilization. A vast majority of the parents/carers read the clinic letters, felt that these were useful, and wished to receive it soon after the consultation. Our Trust policy is to send all clinic letters within 5 days. Parents/carers admit that not all of the information shared in the clinic is retained later and hence find the contents of the letter as a useful reminder of clinic consultation. Although parents/carers focussed on treatment plan and follow up, they found the whole letter useful. The survey has also reassured us in terms of content, simplicity, and accuracy in our clinic letters. Our practice is to ask parents/carers to ring the consultant's secretaries if they have any queries or concerns.

It is known that internet is an important source of information to patients. In our group, we see that internet has established itself as the principal source of medical information even after parents have had the opportunity to get in contact with their GP or specialist doctor. It is interesting to note that parents prefer to seek the help of internet, GP or even family members over paediatric team. Random unverified searching on the internet could end up in biased and inaccurate information. Clinicians and other health professionals should take efforts in guiding their patients towards safe and accurate information on the internet. Helping parents/carers finding right information on the internet is an important way forward. This can be achieved, by adding information and recommending authentic websites within the clinic letter. Another field to explore is the use of emails to send the clinic letter as it is likely to save time and money in the current financial climate.

CLINIC LETTERS – PARENTS PERSPECTIVE

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However, internet safety and potential breach of confidentiality remains a serious risk precluding its widespread implementation. This survey illustrates that parents focus on record keeping of the child's medical condition and preserve the clinic letters. This record is especially useful if the family moves to a different strategic health authority or visits/immigrates to another country. However, it is not common practice for parents to bring previous clinic letters to various medical consultations. Having a national NHS patient database with access of medical health records to all relevant health professionals may obviate its need.

Relatively little empirical research has been performed in this area but what exists is generally supportive. This initiative is one of many that the NHS has introduced to enhance openness, honesty, and the quality of information provided to young persons and parents/carers. In order to minimise incorrect information and to diminish the odds of subsequent complaints by parents, a compromise might be to dictate the letter in their presence during clinic consultation and to provide a speciality-specific glossary of medical terminology. Further studies are required to evaluate the financial implications and safety around alternative ways of communication like emailing letters.

References

1. Harris C., Boaden R. Copying letters to patients: the view of patients and health professionals. J Health Serv Res Policy. 2006; 11: 133-40

2. Krishna Y., Damato BE. Patient attitudes to receiving copies of outpatient clinic letter from the ocular oncologist to the referring ophthalmologist and GP. Eye 2005; 19: 1200-4

3. Saunders NC., Georgalas C., Blaney SP., Dixon H., Topham JH. Does receiving a copy of correspondence improve patients' satisfaction with their out patient consultation? J Laryngol Otol 2003; 117: 126-9

4. Pothier D., Nakivell P., Hall C. What do patients think about being copied into their GP letters? Ann R Coll Surg Engl 2007; 89: 718–721

5. Tuffrey C., Finlay F. Use of the internet by parents of paediatric outpatients. Arch Dis Child 2002; 87: 534-536

6. Wainstein BK., Sterling-Levis K., Baker SA., Taitz J., Brydon M. Use of the Internet by parents of paediatric patients. Paediatr Child Health 2006; 42: 528-32

Authors

Sethuraman S Department of Paediatrics
Queen's Hospital, Burton Upon Trent UK.

Ahmed M Department of Paediatrics
Queen's Hospital, Burton Upon Trent UK

Address for Correspondence

Dr M Ahmed
Consultant Paediatrician, Queen's Hospital, Belvedere Road,
Burton-on-Trent, Staffordshire, UK, DE13 0RB

Tel: 01283 511511 ext 4081
Email: mansoor.ahmed@burtonh-tr.wmids.nhs.uk

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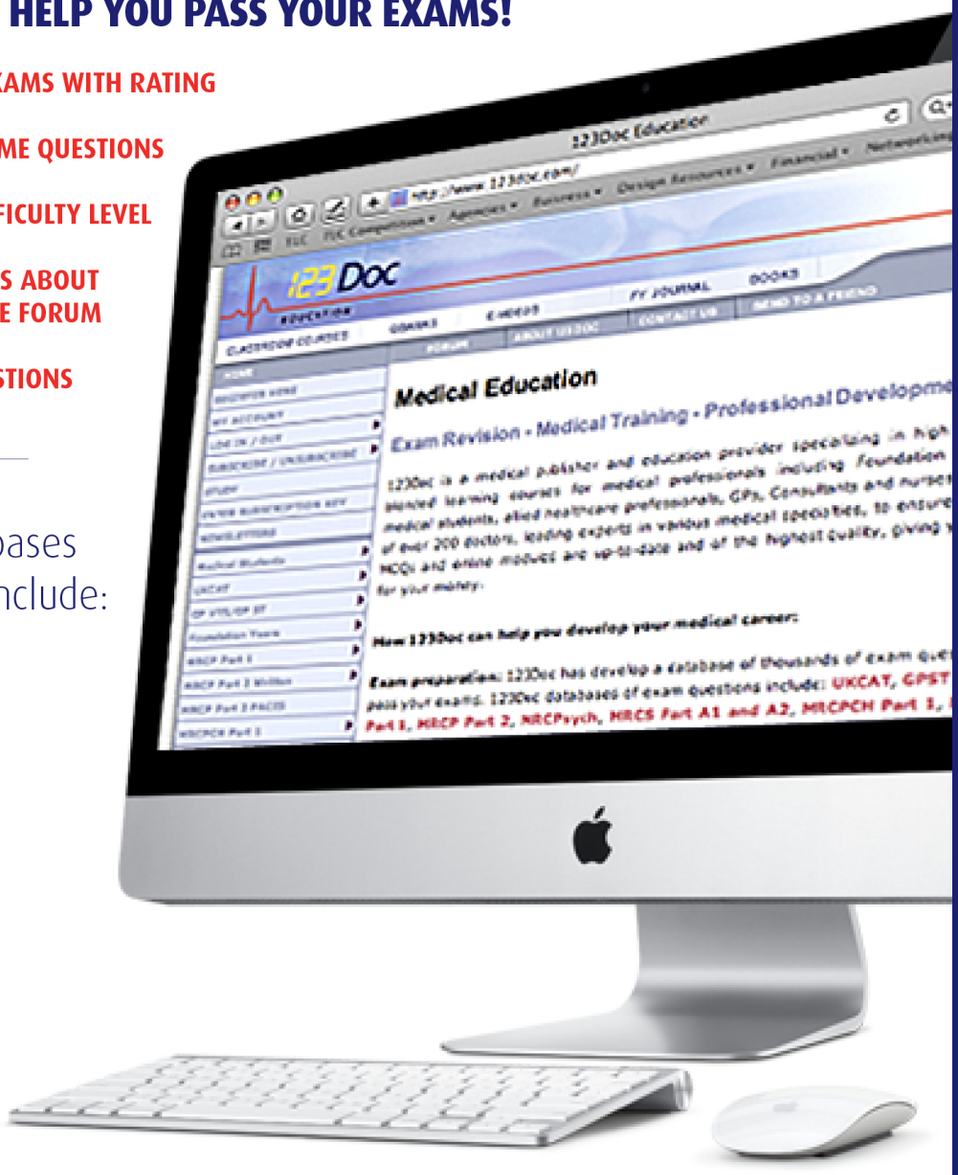

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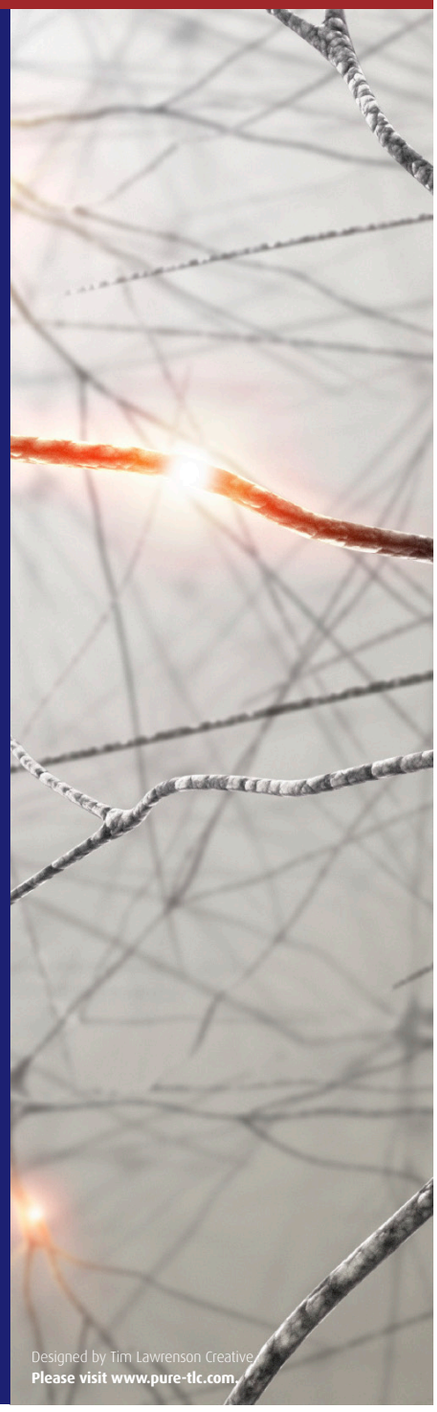

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