epilepsy action

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CURRENT AWARENESS SERVICE

The ketogenic diet and drug-resistant epilepsies

At the International League Against Epilepsy's annual congress in Dublin in September, postdoctoral researcher Natasha Schoeler presented results from University College London's trial into the ketogenic diet (keto) in children.

Based at Great Ormond Street Hospital, London, the Ketogenic Diet in Infants with Epilepsy (KIWE) trial studied children under two years old with drug-resistant epilepsy. Before starting the trial, the team had assessed previous research in this area. It found there had been 13 randomised control trials (RCT) into keto and that it was effective in older children. However, none of the trials concentrated on infants. They conducted a systematic review that found 31 uncontrolled studies and two randomized control trials with evidence of only a very few infants with epilepsy on a keto diet. A meta-analysis showed 60% of individuals had at least a 50% reduction in seizures at three to four months.

The objective of KIWE was to "determine the effectiveness of the keto diet on seizure frequency compared to further ASMs in children under two-years old with apparent drug-resistant epilepsy". Infants were randomized to receive the keto diet or a further ASM. KIWE was a phase-4 randomised control trial, working with patients at 18 sites across the UK. It followed infants in eight-week assessments and ended after 12 months. The demographics of the two groups were similar. Researchers found there was no evidence of keto being superior to a further ASM when it came to seizure frequency, with the two treatments having similar response rates. Of the group taking a further ASM, 40% achieved seizure reduction of more than 50%, with 13% becoming seizure free, and of the keto group, 44% achieved seizure reduction of more than 50%, with 11% becoming seizure free. Similarly, there was no difference in quality of life or impact on parent time. Schoeler said that the researchers were surprised that parents had recorded no difference on the impact of time "considering the perceived difficulty of implementing the ketogenic diet".

When it came to side effects, 43% of the group on ASMs reported at least one serious adverse event compared with 51% in the keto group. The types of event were similar, with seizures, infections and respiratory issues the most common. Schoeler said it was a challenging trial with a predictably slow recruitment, and parents tended to want the ketogenic diet immediately. In conclusion, she said the trial had found the "keto diet was no superior in efficacy to further ASM treatments of drug-resistant epilepsy in infancy". However, it did appear to be safe and should be considered an option in infants after two ASMs.

Schoeler said she expected full results of the trial to be published in the coming months. For more information go to: *www.ucl.ac.uk/child-health/current-trials*

Grace Wood
Co-editor

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Forthcoming courses and conferences

The following are details of forthcoming conferences and courses in epilepsy and general paediatric neurology.

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ilaebritishconference.org.uk

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11
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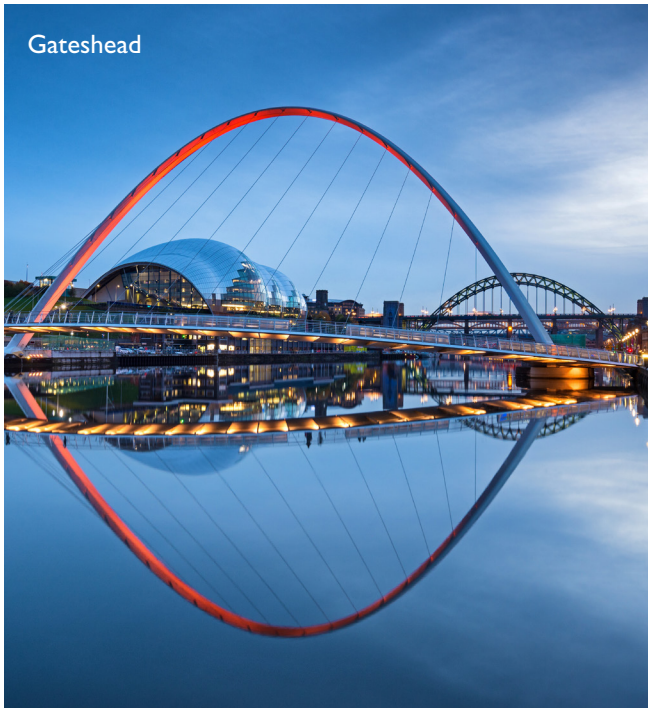
Birmingham, UK
bit.ly/45kfy7E

March 2024
3-8
4th International Training Course on Neuropsychology in Epilepsy
Lyon, France
bit.ly/3VvHu2Z



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Seventeenth Eilat Conference on New Antiepileptic Drugs and Devices (EILAT XVII)
Madrid, Spain
bit.ly/3fdKAbT

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ilae.org/congresses/15th-european-epilepsy-congress

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epilepsy.org.uk/yourchild

Your child and epilepsy

Grow your confidence managing epilepsy in your family

Your child and epilepsy is a new online course for parents and carers of children with epilepsy. It's been developed with parents, epilepsy nurses and psychologists.

This course is a helping hand to support families on their epilepsy journey. It's full of advice and stories from parents. It aims to give parents and carers the confidence, skills and knowledge to support their child to manage their epilepsy.

There are eight parts that cover:

- Understanding epilepsy
- Supporting your child with their epilepsy
- Keeping your child safe
- The impact of epilepsy on family life
- Your child's wellbeing
- Learning and behaviour
- Growing up and independence
- Sources of help and support

Free course

The course is free and flexible. It can be accessed at any time on a computer, tablet or smartphone with internet access.

Leaflets about the course to give to families can be requested by emailing **nurseorders@epilepsy.org.uk**

To view the course go to: **epilepsy.org.uk/yourchild**
Get in touch **learning@epilepsy.org.uk**

Epilepsy networks: basic understanding and clinical application

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Introduction

The human brain is essentially an electrical network and disturbance of electrical signals within this network leads to epileptic seizures. Understanding how these networks function is pivotal in learning about seizure onset, propagation and termination; and also improving our understanding about behavioural problems and other comorbidities in epilepsy. Treatment can also be modified depending on the extent of network involvement. Some fundamental questions within epilepsy revolve around: how does a seizure start and spread? Why do generalised spike and wave discharges, and seizures occur when they do? How do we know which area of the brain is involved in which epilepsy? Why do children have cognitive difficulties with epilepsy? Why do seizures persist in some patients following surgical resection?

These networks and their connectivity can be understood on the basis of three main concepts:

1. **Structural (anatomical) connectivity**, which looks at the physical structure of the brain and how it is inter-connected – usually studied by MRI and various advances, including diffusion tensor imaging and cortical thickness measurements.

2. **Functional connectivity**, which is seen by the different EEG patterns in different areas of the brain during wakefulness and sleep, and correlating these with functional imaging techniques.

3. **Effective connectivity**, which relates to the influence of activity between areas of the brain, which is again recorded with intracranial EEG recordings such as stereo-EEG.

This review will simplistically look at the different networks in the brain and how they influence seizures in different types of epilepsy.

Initiation, propagation and termination of seizures

The basic mechanism relating to seizure propagation has been studied predominantly by scalp and intracranial EEG recordings. The seizures in the generalised (genetic) epilepsies usually present with large amplitude spike and wave discharges, which are similar from start to finish. However, the seizures in focal epilepsies show an evolving pattern that initiates with low-voltage fast activity or hypersynchronous epileptiform discharges. This is followed by irregular large amplitude bursts of epileptiform discharges, followed by electrical depression [De Curtis *et al*, 2015]. There is a complex interplay of principal neurons that synchronously fire at the onset of a seizure, and the inhibitory interneurons that are active prior to the seizure and then suddenly deactivate at the onset of the seizure, then reactivate as the seizures end. Along with this, there are various neurotransmitters, predominantly GABA and glutamate, and voltage fluctuations across the sodium and potassium channels, all of which play a role in seizure progression. Inter-ictal epileptiform discharges may not necessarily correlate with seizure occurrence, although there can be an increase or decrease just before the seizure.

From a network perspective, the brain has a set of network connections across different areas, which is referred to as the default mode network (DMN). [Tangwiriyasakul *et al,* 2018]. In children with epilepsy, there are groups of neurons that may be firing in synchrony, which are referred to as 'hubs'. These hubs are related to neighbouring areas of the brain through various networks creating 'microhubs', which help in propagation of the seizure (Figure 1). During a seizure, the deactivation of the DMN, coupled with increased synchrony in the hubs and microhubs, leads to the seizure. This has been proven in rodent models and in-vivo epilepsy models, and can differ in generalised and focal epilepsies [Pinto *et al*, 2005]. In addition, in children who have epilepsy there is dysfunction in the resting state DMN – mainly increased network-synchronisation, which can contribute to cognitive and behavioural difficulties (Figure 2). Increased connectivity between the hubs and unrelated far away microhubs may be one of the reasons why selective epilepsy surgery is not successful [Shih 2019].

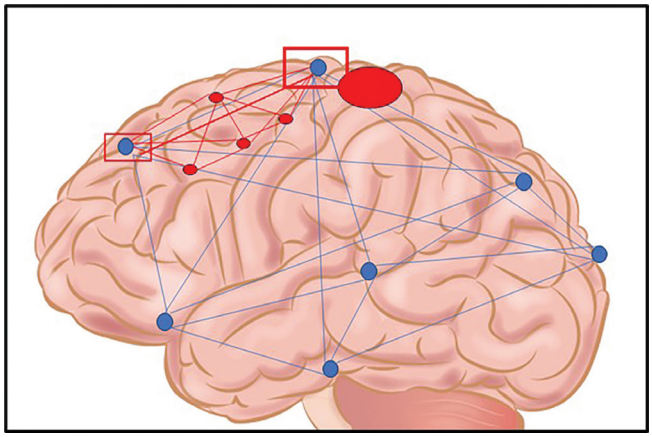

Figure 1: 'Hubs' and 'microhubs' – denoted by red forming an epileptic network within the default network of the brain

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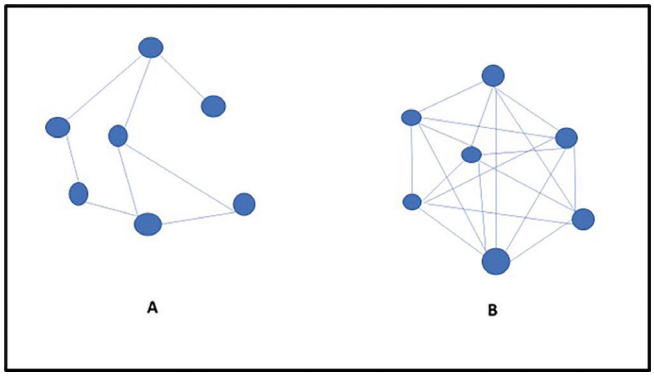

Figure 2: Normal brain (A) and epileptic brain (B), increased network synchronisation in epileptic brain can lead to cognitive and behavioural difficulties

Networks in generalised epilepsy

It is now accepted that generalised seizures typically start in one or several parts of the brain but rapidly spread to involve several networks within the brain. The genetic generalised epilepsies (GGE, previously termed idiopathic generalised epilepsies) are a group of epilepsies where the presenting seizure type is a generalised seizure (tonic, tonic-clonic, absence, myoclonic) and the EEG shows generalised spike and wave discharges. They are presumed to be due to a genetic cause and, although several genes have been identified in some cases, no single gene would contribute to the bulk of the generalised epilepsies. In practical terms, the GGEs comprise four syndromes in childhood – childhood absence epilepsy (CAE), juvenile absence epilepsy (JAE), juvenile myoclonic epilepsy (JME) and epilepsy with generalised tonic-clonic seizures alone (GTCA). The clinical and even the EEG distinction between these syndromes may not always be straightforward with some overlap. Although the majority of children with this group of epilepsies have normal development and cognition, some children do show comorbidities with learning difficulties, hyperactivity and features of autistic spectrum disorder. It is not clearly known why some children are more prone to develop comorbid disorders than others, but increased synchronisation within the networks may well play a role, as discussed earlier.

The generalised spike and wave discharges seen on the EEG are conventionally assumed to have an abrupt onset and offset. However, studies with simultaneous EEG and functional MRI show that networks in such a brain may not always discharge abruptly. The generalised epileptiform discharges are driven by a focal cortical region, which engages rapidly with other areas of the brain through thalamocortical circuits. This is seen through the blood oxygenation level dependent (BOLD) functional MRI signal, which increases in the thalamus and reduces in some cortical regions that are thought to represent the default mode network (DMN) [Tangwiriyasakul *et al,* 2018; Lee *et al*, 2020]. The DMN usually lies within the posterior cortical regions and is synchronised at rest. During the generalised discharges, the most active epileptiform network lies around the sensorimotor cortex, with connections with the prefrontal and precuneus regions. The posterior cortical network seems to be not as synchronous in children with such epilepsies. In the few seconds prior to discharge, the low synchrony in the posterior cortical network, followed by the ramping up of the synchrony in the networks between the sensorimotor cortex and frontal areas lead to a pro-ictal state. The prefrontal and precuneus regions become hub nodes and synchronisation between these subsequently leads to generalised spike wave discharges. There is also involvement of these discharges through the thalamocortical circuity in making them generalised.

Another similar study that looked at the network organisation in childhood absence epilepsy, found that the critical epileptic hubs in the network are located in the posterior cingulate cortex, precuneus, angular gyrus, supramarginal gyrus, superior parietal and occipital regions [Kumar *et al*, 2023]. During the interictal discharges, there is weaker connection within these networks – hence the awareness may not always be impaired. However, during ictal discharges, there is stronger connectivity between these networks and the thalamus, leading to impaired awareness.

These networks probably explain why some children experience more than one types of seizure within the same epilepsy syndrome (e.g. juvenile myoclonic epilepsy and juvenile absence epilepsy). Stronger thalamocortical circuitry and increased synchronisation within the networks lead to seizures that may lead to impairment of awareness, such as absence seizures or even generalised tonic-clonic seizures.

Networks in focal epilepsy

Focal epilepsies originate in a select area in the brain and propagate to involve other nearby areas, then spreading to involve wider networks. These have been extensively studied by video-telemetry in presurgical evaluation for epilepsy surgery. Intra-cranial depth electrode recordings have further provided insight into the onset, propagation and termination of the focal seizure, and the networks involved. Luders et al. defined the concept of epileptogenic zone as the area that initiates seizures and whose removal is necessary for complete seizure resolution [De Curtis *et al*, 2015; Liou *et al* 2020]. Other zones related to seizure initiation and propagation were defined as follows (Figure 3):

1. **Irritative zone:** area of cortex that generates inter-ictal epileptiform activity.

2. **Seizure-onset zone**: area where seizures initiate.

3. **Symptomatogenic zone**: area of cortex that produces the initial ictal signs or symptoms.

4. **Epileptogenic lesion**: lesion seen macroscopically or on MRI that is responsible for seizures.

5. **Functional deficit zone**: area of the cortex that is not functioning normally in the inter-ictal period.

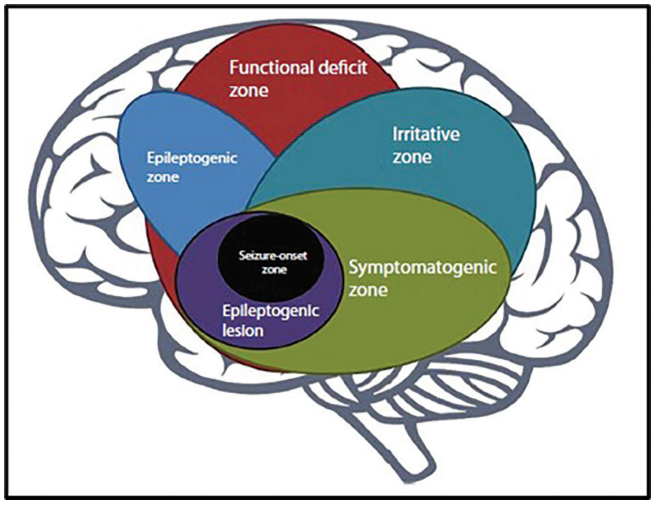

Figure 3: Description of zones related to the epileptogenic area

However, it is important to realise that this is a conceptual exercise based on data-collation from a variety of investigations including neuroimaging, video-telemetry and neuropsychological testing, all of which are clearly integral to pre-operative evaluation of a child undergoing assessment for epilepsy surgery.

Recent studies have shown well-defined networks in different types of focal epilepsy, which have improved our understanding of their semiology and also the impact of network dysfunction on other aspects of cognition. Most of these studies used stereo-EEG or other intracranial recordings [Peng *et al*, 2019; Gupta *et al,* 202].

Temporal lobe epilepsy

This is the most well-studied epilepsy in adults and children, and mesial temporal lobe sclerosis (MTLS) remains the most common cause of epilepsy that is amenable to surgical resection. In children, dysgenetic tumours, focal cortical dysplasia and genetic disorders such as tuberous sclerosis complex are more common than MTLS [Peng *et al*, 2019; González *et al*, 2020]. However, in children the epilepsies usually involve a wider network leading to more varied presentations. The connections to the insula, orbito-frontal and occipital networks are responsible for much of this diversity in seizure semiology.

Behavioural arrest is the most common feature of temporal lobe seizures and implies a spread to the thalamic networks leading to impairment of awareness. In children, motor features such as spasms, dystonic posturing and clonic jerking are all more likely to be seen than in adult temporal lobe epilepsy, and imply a more rapid spread to the frontal networks. Automatisms, oral and motor, are also common and are poorly lateralising. Auras, such as rising epigastric sensation and fear, and various hallucinations and illusions, can also be associated with origin in the mesial temporal area. Vegetative dysfunction, such as hyper-salivation, apnoea, nausea and vomiting, may represent spread to the insular networks. Somatosensory auras often reflect involvement of the frontal networks, whereas visual auras are likely to be due to involvement of occipital networks [Peng *et al*, 2019; González *et al*, 2020]. Sometimes, lesions elsewhere may lead to temporal lobe epilepsy, for example periventricular nodular heterotopias or hypothalamic hamartomas can lead to ictal spread within the temporal networks, leading to temporal lobe seizure semiology. Similarly, seizures arising from the orbitofrontal networks may quickly spread to the hippocampus and amygdala causing non-motor temporal lobe like semiology.

Frontal lobe epilepsies

Extra-temporal seizures are more common in children and the frontal lobes are the second most common origin after the temporal lobes that cause focal seizures. Most of them are short, explosive and motor, and occur in clusters. However, the frontal lobes are large and there is an extensive network within them that can contribute to a myriad of presentations. In addition, many of the epilepsies that originate elsewhere eventually lead to the frontal networks, which result in not only seizures but cognitive and behavioural disturbances [Barot *et al*, 2020]. The frontal lobe is divided into three different regions: motor, premotor and pre-frontal. The mesial aspect of the premotor area is called the supplementary motor area (Figure 3). The prefrontal region not only receives projections from other areas but also the dorsomedial nucleus of thalamus.

Clinically, there are three distinct patterns of seizures arising from the frontal networks. Tonic seizures, with symmetrical or asymmetrical posturing, are likely to originate from the supplementary motor area. Some of these are triggered by sudden and unexpected stimuli, such as a startle. Versive seizures, with head and trunk deviation to one side, forcefully originate from the dorsolateral premotor cortex and occur contralaterally to the seizure focus. Ipsilateral head deviation can occur in seizures from the anterior mesial frontal cortex. Hypermotor seizures consist of either marked agitation with body rocking, kicking and boxing, or milder agitation with movements of trunk and pelvis with tonic posturing. Most of these originate in the mesial frontal or orbitofrontal networks. Autonomic changes, which include tachycardia and bradycardia, may occur due to spread to networks involving the mesial temporal structures, amygdala and hippocampus. Ictal fear implies involvement of the cingulate networks. Ictal laughter, commonly seen in hypothalamic hamartomas, can be seen in the networks involving the pericingulate premotor area.

Posterior cortex epilepsies

Epilepsies that originate from the parietal, occipital and posterior temporal lobes are referred to as posterior cortex epilepsies. The seizures from these regions are less well localised and constitute approximately 10% of all refractory epilepsies [Jacobs, 2020]. The semiology of seizures can be challenging to interpret in view of the fast ictal propagation to the temporal or frontal networks. Most common aetiologies are structural, such as a focal cortical

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dysplasia, developmental tumours or gliosis, secondary to neonatal hypoglycaemia.

The occipital lobe is the main visual processing area, and the primary visual cortex lies on the mesial occipital region. Other aspects constitute the visual association areas responsible for visuo-spatial processing, motion perception and colour discrimination. The parietal lobe is divided into superior and inferior parietal regions. The superior parietal forms the somatosensory association cortex and is responsible for visuo-motor coordination, integrating sensory information from various parts of the body and processes information related to touch and visuo-spatial processing. The inferior parietal region is closely linked with the angular gyrus and supramarginal gyrus.

Visual hallucinations are the hallmarks of seizures originating from the occipital lobe. They can be positive (seeing objects) or negative (not seeing objects) and can be elementary (multicoloured, variable shapes and moving horizontally). Complex visual hallucinations may take the form of persons, animals, objects or figures, which may be familiar, friendly or frightening, and may be in the centre of the visual field and move horizontally. Ictal amaurosis is usually bilateral, but can also be homonymous hemianopsia. Visual illusions occur when the seizure is around the occipito-temporo-parietal area, which consists of distortion of images, changes of size (macropsia or micropsia) and shape (metamorphosia) and can be complex. Ictal or post-ictal headache is common in more than 50% of cases of occipital seizures and can be confused with a migraine [Rajapakse *et al*, 2016; D'Agnan *et al*, 2023]. Ocular motor symptoms, such as forced eye blinking, eyelid flutter, nystagmus and deviation to one side, imply involvement of the frontal networks. Ictal vomiting and autonomic features imply origin in the occipital networks and can be seen in the self-limiting occipital childhood epilepsies.

Somato-sensory auras imply involvement of parietal networks. These include numbness, pins and needles, and unpleasant crawling sensations, usually contralateral to the side of seizure onset. Pain, often described as burning, implies spread to the parietal operculum or insula. An alien hand syndrome (where one hand is not under voluntary control) may also occur. Spread to the temporal networks leads to vestibular sensations and vertigo. Language dysfunction can be seen when the temporal networks of the dominant hemisphere are involved. Focal tonic seizures strongly imply spread to the supplementary motor area networks and automatisms imply involvement of the temporal-limbic networks.

Apart from visual impairment, which is likely in cases of gliosis secondary to neonatal hypoglycaemia, there is often developmental impairment and learning difficulties in children with occipital lobe epilepsies, which implies dysfunction of a wider functional network. The EEG may show a widespread inter-ictal discharge involving the parietal and posterior temporal regions, and can be poorly localising and even lateralising in many cases, in view of the rapid seizure spread to involve the neighbouring networks.

In summary, basic knowledge of various networks involved from epilepsies arising from different areas is essential to interpret the semiology of focal seizures and consequently target investigations more appropriately – particularly when evaluating a child with drug-resistant epilepsy.

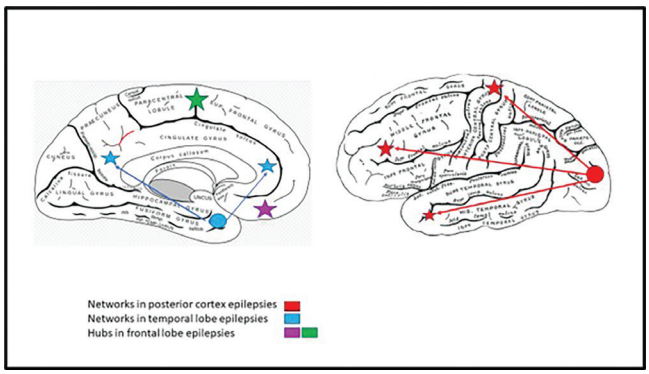

Figure 4: Different epileptogenic networks and hubs in different areas of the brain

Epileptic network(s) and cognition

Difficulties in learning as well as developmental disorders are well-recognised comorbidities in children with epilepsy. Despite adequate control of seizures, some children continue to experience learning difficulties and studies have attempted to better understand the role of specific networks in this situation. The concept of a 'default mode network' involving functional connectivity between specific areas of the brain, such as the precuneus, medial prefrontal cortex and lateral parietal cortex, is important to understand [Bear *et al*, 2019]. This is a resting state network that becomes engaged in tasks such as social and adaptive behaviours, auditory attention, word list tasks and working memory. It typically attenuates during tasks that require higher cognitive functioning, such as language learning or reading. Multiple studies have showed less deactivation of the DMN during such learning tasks in children with epilepsy. In children with childhood epilepsy with centro-temporal spikes, altered connectivity in the inferior frontal gyrus, temporal lobe, supramarginal and angular gyrus is associated with language dysfunction [Bear *et al*, 2019]. Similarly, network dysfunction in the dorsolateral prefrontal cortex, orbitofrontal cortex and anterior cingulate cortex has been associated with executive dysfunction, particularly with relation to attention, processing speed, working memory and inhibitory control, and is a prominent feature in frontal lobe epilepsies [Barot *et al*, 2020].

The role of networks in epilepsy surgery

It is important to understand the influence of networks when assessing and planning a specific resection for drug-resistant epilepsy. The semiology of the seizures (as seen above in epilepsies originating from various parts of the brain) and the results of video-telemetry recordings,

neuroimaging including functional imaging studies and detailed neuropsychology testing, must be collated in assessing each child for potential surgery. The identification of the extent of network involvement and the removal of all relevant hubs is essential for success of a particular surgery.

Children with MTLS may also have network hubs that involve the lateral neocortical temporal lobe [Amorim-Leite *et al*, 2020; Foit *et al*, 2020; McGonigall 2020]. Evaluating these in detail and using intracranial recordings through stereo-EEG is important when deciding whether selective removal of mesial structures or a more radical removal of the lateral temporal lobe will achieve the best outcome. This applies to both seizure freedom and cognitive outcome. The lack of detailed network analysis may well be one of the most important factors when explaining why some children experience breakthrough seizures after initial surgery.

Understanding the networks in treatment of epilepsy

The aim of epilepsy treatment is complete seizure control, which can be achieved in about 60-65% of children with anti-seizure medications (ASMs) alone [Kwan *et al*, 2010]. The medications have different mechanisms of action that involve the various channels (sodium, potassium, calcium etc) and neurotransmitters in the brain. The influence of ASMs on the networks is not understood. In the remaining 35-40%, drug-resistant epilepsy ensues, and other approaches to treatment need to be considered including surgery. Simplistically, the treatment can be divided into 'vertex-centric', which means removal of the vertex or hub of the epilepsy; or 'network-centric' which focuses on the treatment of the aberrant network; or a combination of the two [Kwan *et al*, 2010]. In children who are being evaluated for surgery, lesionectomy (vertex-centric) offers the best option for seizure control and discontinuing ASMs. Minimally invasive surgical treatments, such as gamma knife, radiofrequency thermocoagulation and laser interstitial thermotherapy (LITT), are other options where lesions are localised but difficult to safely access surgically, for example in the case of hypothalamic hamartoma. In some cases, where the seizures involve larger regions, disconnection of the network by means of corpus callosotomy or hemispherotomy; or large network disconnections such as temporo-parieto-occipital resection might be beneficial.

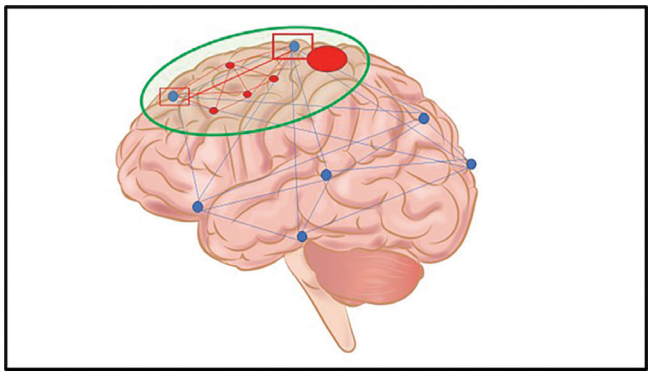

Figure 5: Understanding the network in the lesional epilepsy is essential to do the amount of resection for optimal outcome

Neuromodulation is gaining prominence in the treatment of drug-resistant epilepsy and particularly where surgery is impossible or has previously failed. Neuromodulation can be invasive, such as vagus nerve stimulation (VNS), deep brain stimulation (DBS), or responsive neurostimulation (RNS), or non-invasive, such as transcutaneous auricular vagus nerve stimulation or transcranial direct current stimulation [Lehnertz *et al*, 2023]. The latter yields to modulate cortical excitability by subthreshold membrane depolarization or hyperpolarization. Similarly, RNS delivers neuromodulation locally. It is a closed-loop system with subdural or depth electrodes (placed within or very close to the seizure-onset network) that records neurophysiological ictal signs and stimulates locally to terminate the ictal discharges. This is promising for cases where the epilepsy may be arising from eloquent areas of the brain that may not be amenable to a safe and complication-free resection.

Another closed-loop neuromodulation device does not use cerebral input, but a cardiac-based seizure detection algorithm (CBSD-VNS). A pre-defined increase of the heart rate is supposed to be seizure-related and triggers additional vagus nerve stimulation, thereby the closed-loop system is a brain-heart loop. The exact mechanisms of action of CBSD-VNS and of the classic off-loop VNS are not fully understood but due to the widespread projections of the vagal nerve, by now a rather unspecific global (i.e, network-centric) brain activation is presumed. The transcutaneous auricular vagus nerve stimulation device (taVNS), stimulating the auricular branch of the vagus nerve, seems to target similar projection areas as the VNS device, so an unspecific and rather global modulation is to be supposed. For DBS in epilepsy, the exact mechanism of action is not fully understood. In drug-resistant epilepsies arising from both temporal lobes, electrodes are implanted bilaterally into the anterior nuclei of the thalamus. The anterior nuclei of the thalamus have widespread interaction, so an unspecific and rather global modulation has to be assumed. To sum this up, VNS, taVNS and DBS conceptually act as 'network-centric' seizure control.

Summary

In summary, it is exciting but also challenging to review concepts in epilepsy as a network disease. Although there is often a discrete localised region to resect, ablate or neuromodulate, in many patients with drug-resistant focal epilepsy, functional and structural connectivity studies have convincingly shown that multiple regions of the brain are involved in the genesis and/or propagation of seizures or in the maintenance of epileptogenicity. Although one may be able to eliminate seizures by removing a small part of the brain, other networks that may be involved can rekindle epileptogenicity or contribute to comorbid problems, or both. It is possible that multiple interventions, including ASMs, resection and neuromodulation may all be needed in a few patients to significantly improve seizure outcome and, consequently, quality of life.

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One promising clinical application is the use of network studies in seizure detection, which is an area of increasing research. A sensitive and consistently reliable method to predict seizures will significantly impact clinical practice and mitigate disability for our patients, who usually have no idea when the next seizure will occur. If patients can know with a high degree of accuracy that a seizure will start within minutes of its occurrence, personal safety measures and notification algorithms can be developed and then implemented. Theoretically, this could lead to greater independence, reduced anxiety, fewer injuries and potentially a lower risk for sudden death in epilepsy. This is a really exciting area for future research.

Finally, the increasing knowledge and clinical significance of networks further emphasises the need to always obtain a detailed account, supported by mobile phone footage, of seizures when diagnosing epilepsy and the specific epilepsy syndrome.

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The Epilepsy Space

The mobile friendly website is a helping hand for 16-25 year olds to live their best life with epilepsy

The Epilepsy Space will help young people to:

- Manage their epilepsy
- Feel less alone
- Increase their confidence
- Get the support they need

There's lots of epilepsy facts, tips and stories from young people sharing their experience.

The content is short and interactive. It's not all reading, there's video and young people can share their own quotes, stories and videos too.

It's been created with young people and reviewed by epilepsy nurses.

Take a look at:
epilepsyspace.org.uk

Leaflets about The Epilepsy Space to give to young people can be requested by emailing:
nurseorders@epilepsy.org.uk

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Find out more
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Recently published papers

This section highlights recently published papers. Hopefully this will be very useful to all, helping to keep everyone up to date with the latest developments. It will certainly save you research and reading time, not having to search so many journals.

There are many (often more than 300) epilepsy papers published every three months, so what follows has been edited. All animal papers have been excluded. We hope you find the papers of interest in your pursuit to keep abreast of the very latest knowledge.

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