Chapter 15

Occipital and parietal lobe epilepsies

JOHN S. DUNCAN

UCL Institute of Neurology, National Hospital for Neurology and Neurosurgery, Queen Square, London

---

Epileptic seizures of parietal and occipital origin are heterogeneous and mainly characterised by the presenting auras, although the most dramatic clinical manifestations may reflect spread, and overshadow the focal origin. The two lobes serve mainly sensory functions, and the characteristic seizure phenomena are therefore subjective sensations. The incidence of these seizures is not well known, but they are generally considered rare. Occipital seizures have been reported to constitute 8% and parietal seizures 1.4% of total seizures in the prevalent population with epilepsy[1,2]. The pattern of seizures is most commonly focal seizures without impairment of awareness, with occasional secondary generalisation. Focal seizures with impairment of awareness are rare and usually indicate spread of the seizure into the temporal lobe.

Seizures with somatosensory symptomatology[1-3]

Somatosensory seizures may arise from any of the three sensory areas of the parietal lobe, but the post-central gyrus is most commonly involved. Seizures present with contralateral, or rarely ipsilateral, or bilateral sensations. All sensory modalities may be represented, most commonly tingling and numbness, alone or together. There may be prickling, tickling or crawling sensations, or a feeling of electric shock in the affected body part. The arms and the face are the most common sites, but any segment or region may be affected. The paraesthesia may spread in a Jacksonian manner, and when this occurs motor activity in the affected body member follows the sensations in about 50% of cases.

Pain is the second most common somatosensory seizure experience, often described as stabbing, intense, torturing, agonising or dull. It may be difficult to distinguish the pain from thermal perception or muscle cramps, which frequently follow the pain. Thermal perceptions are less common than pain or paraesthesia, and rarely occur without other sensory phenomena. A burning sensation is more common than the feeling of cold. Contralateral abdominal pain is also described.

A small subgroup of seizures with sexual phenomenology seems to originate in the paracentral lobule where the primary somatosensory area for the genitalia is thought to reside, usually involving the non-dominant hemisphere. The seizures present with a tactile somatosensory aura affecting the genitalia, but the ensuing seizure may exhibit other features of sexual behaviour.

A feeling of inability to move is thought to involve the secondary sensory area on the suprasylvian border. Such seizures may be preceded by a psychic aura (‘psychoparetic’). Contralateral, ipsilateral, bilateral or midline structures may be affected. Paroxysmal ictal paralysis may spread in a Jacksonian way and be followed by clonic activity in the same body part. Other somatosensory features in epilepsy are body image disturbances, such as feeling of movement or altered posture in a stationary limb, feeling of floating, twisting or even

disintegration of a body part. Rarely the eyes are the only affected body part, and in those cases the discharge is thought to involve the rostral occipital cortex. Illusion of distorted or changed body shape is another phenomenon, in which a body part may be felt to be swollen or shrunken (macro- and microsomatognosia), or elongated or shortened (hyper- and hyposchematica). The peripheral parts of the extremities and tongue are most commonly affected. Other described disturbances are unilateral asomatognosia – where absence of a body part, limb or the hemibody is experienced – and sensation of a supernumerary or phantom limb. Some parietal seizures may resemble panic attacks.

It is important to note that there is also sensory representation in the posterior insula and in the supplementary motor area, so seizures involving these parts may have prominent sensory symptoms[4]. Awareness of this is crucial when surgical treatment is being considered.

Seizures with visual symptomatology[1,3]

Seizures from the occipital lobes and the parieto-occipital junction are characterised by visual phenomena, but visual auras may occur in epilepsy affecting any part of the visual pathways. Elementary visual hallucinations are most common, especially crude sensations of light or colours, which may take various shapes, be continuous, steady or moving, or be interrupted flashes of light. Visual loss, either total or partial, may also occur and is especially common in children. Transient amaurosis as an ictal phenomenon lasts seconds to minutes, but visual loss may also occur as a post-ictal deficit. Amaurosis is usually bilateral and may take the form of a blackout or whiteout.

Formed visual hallucinations are experienced fairly often in epilepsy. Pictures of people, animals or scenes may be perceived, either static or moving. One subtype is epileptic autoscopia, where the subjects see mirror images of themselves, sometimes in long-lived situations. Formed hallucinations are usually brief, and may be associated with slow head and eye turning, with the gaze towards the direction of the moving images perceived. They may be associated with various types of visual illusions. Usually, patients are aware of the unreality of the experience. In comparison with migraine, that is usually associated with sharp lines and fortification spectra, the visual hallucinations of occipital seizures commonly comprise coloured blobs of light. As a further distinction, the visual aura of migraine usually evolves much more slowly, over several minutes.

Visual illusions also occur as a seizure phenomenon, and visuo-spatial perceptions and topographical sense have been located to the non-dominant parietal lobe. The simplest types mainly involve visual illusion of spatial interpretation, illumination or colouring of vision, or movement in space. Perceived objects may appear diminished or enlarged (micro- or macropsia), altered in shape, squeezed or compressed from above, downwards or sideways, vertical and horizontal components may be oblique and lines wavy. Lines may be defective or fragmented, stationary objects seen as moving, or motion appears too slow or too fast. In some cases, such experiences may be difficult to distinguish from the characteristic illusion of movement in vertigo. More complex forms include inappropriate orientation of objects in space, like teleopsia, where objects appear both small and at a distance, or enhanced stereoscopic vision, in which near subjects seem very close and more distant objects located very far away. Palinopsia, or visual perseveration, in which visual images recur or persist after removal of the stimuli, may also occur as a seizure.

Other seizure phenomena from occipital and parietal regions[1,3]

Ictal anosognosia, apraxia, acalculia, alexia and aphemia may occur in epilepsy from the posterior brain regions, often presenting as confusional states. Gustatory seizures sometimes have their origin on the suprasylvian border close to the sensory region for the mouth and

tongue. Vertiginous sensations are also thought to originate in the suprasylvian and possibly the occipito-parietal region. Various seizure types may occur in a single patient at different times.

The only primary motor seizures from the posterior brain regions are oculotonic and oculoclonic seizures, or epileptic nystagmus, originating in the occipito-parietal cortex. The nystagmus usually has the fast beating component to the site opposite the lesion or EEG focus, i.e. contraversive. The nystagmus may occur as an isolated manifestation, or be associated with head or trunk version, but rarely other motor activity accompanies, and consciousness is usually retained. Eyelid flutter and rapid blinking are other features of occipital epilepsy, often at the very beginning of seizures.

Provoking and associated/accompanying features[1]

Partial occipito-parietal seizures may be provoked by various stimuli involving the receptive, interpretive and connective function of the parietal and occipital lobes. The most common precipitating factor is photic stimulation, but other well-known inducers are tactile stimulation, reading, drawing, calculation and other mental activity. The EEG may show generalised changes, but focal electrical discharges in the posterior regions may occur. These seizures are very rare.

Seizure spread from an occipital or parietal origin may cause a variety of motor activities; some patients may have different patterns of seizure spread in different seizures, misleadingly suggesting multifocal disease.

Post-ictal phenomena associated with parietal and occipital seizures are transient numbness, inability to move despite no loss of power in affected limbs and post-ictal blindness. There is no correlation between duration and severity of seizures and the duration of the post-ictal neurological deficits. Post-ictal numbness and paralysis are usually short lasting, but post-ictal blindness may be prolonged and, in some cases, permanent. Fixed hemianopia may help confirm occipital lobe onset.

Causes

In a large series of patients with parietal lobe epilepsy from the Montreal Neurological Institute[5,6], tumours, gliosis and scarring were the commonest causes. Malformations, vascular lesions and infarction were also described. In occipital lobe epilepsy, three-quarters of patients may have underlying abnormalities shown on MRI. Causes include tumours, trauma, malformations (focal cortical dysplasia, periventricular heterotopia, band heterotopia and polymicrogyria), ischaemia, mitochondrial disease (with migraine, photosensitivity and other neurological manifestations), Sturge-Weber syndrome and coeliac disease with bilateral occipital calcifications. Occipital seizures can occur in hyperglycaemia and pre-eclampsia, and may occur early in the course of Kuf's disease or Lafora body disease. Three subsyndromes of occipital epilepsy have been described in childhood and adolescence[7-10]. Seizure semiology, the occurrence of amaurosis or migraines, and reactive EEG patterns do not differentiate between idiopathic, often benign, and symptomatic occipital epilepsy. MRI will identify most symptomatic cases. The relationship between migraine and occipital epilepsy is complex[7]. The differential diagnosis may be difficult. Further, epileptic seizures may evolve from an attack of migraine, and vice versa.

Electroencephalographic features

In somatosensory epilepsy, the localisation of electrical discharges on scalp EEG often cannot be correlated with a clinical ictal pattern and the seizures are often electrically silent. EEG changes may be lateralising rather than localising. Sometimes slow activity is most prominent. The two most common EEG features observed are central parietal spike or spike-wave discharges that may be sustained during the ictus, and temporal discharges, with occasionally more posterior spread. During seizures, spread often involves the motor cortex, and the supplementary motor or speech areas of the frontal lobes. Spread to the temporal lobes is said to be rare, but has been described and reproduced by electrical stimulation. Secondary bilateral synchrony may occur.

Changes in the posterior background activity may be helpful in occipital lobe epilepsy. Occipital foci are often widespread and may move between the occipital pole and the anterior temporal lobes. Spread seems to be to the parietal and frontal regions when the discharge originates in the supracalcarine region, but to the ipsilateral temporal lobe when the epileptic activity arises in the infracalcarine cortex. Spread to the contralateral occipital lobe via the corpus callosum seems to occur late in adult cases. Electrical abnormalities may be confined to the temporal lobes, and depth electrode studies in patients with complex partial seizures have in some cases revealed an occipital origin of the epilepsy, although such origin has not been reflected in the clinical picture of the seizures or revealed by scalp EEG. Occipital onset seizures may therefore be more prevalent than previously thought.

Treatment

The medical treatment of occipital and parietal epilepsy is no different to that of other focal epilepsies. Surgical series are less comprehensive than those in temporal lobe epilepsy. Historical series suggest 20% of non-tumoural and 75% of tumoural parietal lobe cases may be rendered seizure-free by resective surgery[5,6]. These figures will probably improve with the application of modern neuroimaging methods and better case selection. Surgical outcome in refractory occipital lobe epilepsy depends largely on the underlying pathology. Outcome is better for tumours than for developmental abnormalities[11].

Surgery to the parietal and occipital lobes carries the likelihood of resulting in a fixed deficit, particularly a visual field defect, somatosensory or higher cognitive impairment. This must explained carefully to the patient in the discussion of the risk-benefit ratio[9,12,13].

References

1. SVEINBJÖRNSDÒTTIR S, DUNCAN JS. Parietal and occipital epilepsy. Epilepsia 1993; 34: 493-521.
2. SALANOVA V. Parietal lobe epilepsy. J Clin Neurophysiol 2012; 29(5): 392-6.
3. ADCOCK JE, PANAYIOTOPOULOS CP. Occipital lobe seizures and epilepsies. J Clin Neurophysiol 2012; 29(5): 397-407.
4. NGUYEN DK, NGUYEN DB, MALAK R, BOUTHILLIER A. Insular cortex epilepsy: an overview. Can J Neurol Sci 2009; 36 Suppl 2: S58-62.
5. SALANOVA V, ANDERMANN F, RASMUSSEN T et al. Clinical manifestations and outcome in 82 patients treated surgically between 1929 and 1988. Brain 1995; 118: 607-627.
6. SALANOVA V, ANDERMANN F, RASMUSSEN T et al. Tumoural parietal lobe epilepsy. Brain 1995; 118: 1289-1304.
7. CARABALLO, R., KOUTROUMANIDIS, M., PANAYIOTOPOULOS, C.P., FEJERMAN, N. Idiopathic childhood occipital epilepsy of Gastaut: a review and differentiation from migraine and other epilepsies. J Child Neurol 2009; 24: 1536-42.
8. MICHAEL M, TSATSOU K, FERRIE CD. Panayiotopoulos syndrome: an important childhood autonomic epilepsy to be differentiated from occipital epilepsy and acute non-epileptic disorders. Brain Dev 2010; 32: 4-9.
9. PANAYIOTOPOULOS CP, MICHAEL M, SANDERS S, VALETA T, KOUTROUMANIDIS M. Benign childhood focal epilepsies: assessment of established and newly recognised syndromes. Brain 2008; 131: 2264-86.
10. KOUTROUMANIDIS M. Panayiotopoulos syndrome: and important electroclinical example of benign childhood system epilepsy. Epilepsia 2007; 48: 1044-53.

11. AYKUT-BINGOL C, BRONEN RA, KIM JH et al. Surgical outcome in occipital lobe epilepsy: implications for pathophysiology. Ann Neurol 1998; 44: 60-69.
12. OLIVIER A, BOLING W. Surgery of parietal and occipital epilepsy. Adv Neurol 2000; 84: 533-575.
13. PONDEL-SORDO M, DIOSY D, TELLEZ-ZENTENO JF, GIRVIN JP, WIEBE S. Epilepsy surgery involving the sensory motor cortex. Brain 2006; 129: 3307-3314.