Epileptic auras: phenomenology and neurophysiology

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ABSTRACT – This review discusses the phenomenology, neurophysiology, and localization of epileptic auras with particular emphasis on how auras can manifest as part of an epileptic network. Epileptic auras, as the first clinical symptom of a seizure, may lead us to infer the site of seizure onset. At the same time, auras can also be a result of activation or alteration in an epileptic network. They can be highly specific or ill-defined in symptomatology. They occur as a result of limited seizure activation, allowing access of the neural signal to the conscious brain. An understanding of epileptic auras offers a window into understanding fundamental brain functions, and helps the clinician at the bedside to make appropriate diagnostic and therapeutic choices.

Key words: epileptic aura, seizure localization, epileptic network

Phenomenology of auras

Aura, a "breeze" or "soft air" in Latin and Greek etymology, has been recognized since the time of Pelops (Greek, second century CE) and described by Hughlings Jackson as "the so called warning". It is defined in the 1981 International Classification of Epileptic Seizures as "that portion of the seizure which occurs before consciousness is lost and for which memory is retained afterwards" and corresponds to a simple partial sensory seizure when occurring in isolation. In the latest 2010 proposal of the ILAE Commission on Classification and Terminology, an aura would correspond to a focal seizure involving subjective sensory or psychic phenomena only (Berg *et al.*, 2010). Epileptic auras are usually short, lasting for a few seconds to a few minutes. Occasionally, auras can be prolonged or recurrent with short breaks and of fluctuating intensity. At that point, it may be a form of partial status epilepticus or *aura continua*. Auras are easy to recognize when followed by observable changes in behaviour or cognition typical of that in a clinical seizure. In cases where there are no further clinical manifestations, it may be challenging for the clinician to determine whether the reported sensations are epileptic or not,

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particularly if they are non-descript or can be a result of other disturbances, as in the examples of nausea and anxiety.

Aura symptoms are rich in character and diverse in range. They can manifest in each of the major senses, and be divided into elementary or complex phenomena, reflecting activation of the primary receptive or higher order association cortices, respectively. They can further manifest in the perceptual, cognitive, mnesic, or emotional spheres. Others are related to visceral or autonomic systems. Many studies dating back to Jackson, Gowers and Lennox reported on the different types of auras (Jackson, 1879; Gowers, 1880; Lennox and Cobb, 1933). While all studies classified most aura symptoms in commonly understood categories, there also were differences. The easiest to classify are those pertaining to the primary senses: *i.e.* olfactory, visual, auditory, gustatory, and somatosensory. Symptoms related to higher order processes, emotions, and autonomic alterations show greater variation in classification schema. A cold sensation can be placed under autonomic, somatosensory, "other", or its own category of "hot and cold" in different reports. Auras are subjective symptoms that are only revealed in communication and thus influenced by individual choices of words: a dizzy sensation to one may be vertigo or visual instability to another. Auras can be multiple, and the patient may emphasize one or the other components at different times, making an attempt at classification difficult. Furthermore, auras can change over time in the natural course of disease, or as a result of medical or surgical intervention. Treatment at times leads to loss of aura, and less often, an alteration of the experience.

Prodromes and sensations in generalized epilepsy

Auras are different from prodromal symptoms. Prodromes have been reported in generalized as well as focal epilepsies (Scaramelli *et al.*, 2009). They last for hours to days and are often described as a change in demeanour, behaviour, mood or personality perceived by the patient or others. Sometimes they can take the form of more discrete symptoms like headache, restlessness, dizziness, lightheadedness or tiredness (Schulze-Bonhage *et al.*, 2006). The biological basis for prodromal symptoms is unknown. Studies on preictal changes before a clinical seizure have revealed alterations in EEG dynamics that can precede the ictus by minutes to hours (Le Van Quyen *et al.*, 2001; Litt *et al.*, 2001). While these changes have not been directly correlated to prodromal symptoms, they represent a basis for preictal cerebral disturbances.

Although epileptic auras are typically conceived of as a sign of focal seizure onset, similar symptoms are reported in patients with idiopathic generalized epilepsy prior to their seizures. The incidence of aura type sensations in generalized epilepsy ranged from 26% in a series of 176 patients (Gungor-Tuncer *et al.*, 2012) to 70% in a smaller series of 19 patients (Boylan *et al.*, 2006). Another report of 798 patients with generalized epilepsy found that 64% reported at least one form of aura by structured interview (Dugan *et al.*, 2014), a rate much higher than when surveyed by open questions (21%); a possible illustration of recall bias in patients and ascertainment bias in examiners. We share the opinion of Panayiotopoulos (Panayiotopoulos, 2010) that some of the preictal symptoms or auras in generalized epilepsies are most likely related to a build-up of repeated spike and wave discharges prior to a major seizure. Thus, in patients with a form of absence epilepsy, they may experience dizziness, spaciness, loss of time, and altered thoughts, for example. In those with myoclonic epilepsy, they may feel shocks, electricity, tingling, or jitteriness, as fragments of a seizure prior to a major convulsion. Nevertheless, there are also examples of epigastric sensation, elementary visual hallucination, and other sensory auras indistinguishable from those in patients with focal epilepsy. Such focal aura symptoms may be a result of selective focal activation of cortical areas or networks, as supported by other evidence of focal involvement in seizure semiology, EEG, and fMRI studies; an evolving concept to our understanding of the generalized epilepsies.

Neurophysiology and principles of localization

In order to understand the significance of the aura and its potential localizing value, it is important to correlate its neurophysiology to the functional organization of the cerebrum and the networks involved in generating such a phenomenon.

The aura is a seizure, but remains circumscribed in its clinical and neurophysiological extent when occurring in isolation. On scalp EEG recordings, an isolated aura, as with other simple focal seizures, only shows an accompanying ictal EEG signature in 10-15% of cases (Devinsky *et al.*, 1989). With intracranial EEG electrodes, the aura is more frequently recorded but not always (Sperling *et al.*, 1989; Sperling and O'Connor, 1990). By utilizing simultaneous micro and macro intracranial electrode recordings (Babb *et al.*, 1987), it has been shown that during auras only a small percentage of neurons (14%) are recruited in the ictal discharge. Similarly, ictal SPECT in isolated auras failed

to show reliable foci of hyperperfusion, presumably because the neuronal pool recruited is insufficient to increase blood perfusion above the threshold of SPECT detection (Elwan *et al.*, 2014).

The symptoms in an aura are appreciated when the ictal discharge activating the brain has access to consciousness. Because the aura is at the beginning of a seizure, we infer that it arises from or close to the seizure onset zone. When the patient reports a sequence of aura symptoms, the first is more likely to be closest to the seizure onset zone. However, if the seizure starts in a functionally silent area of the brain, there will be no discernible symptom until the seizure discharge propagates to a symptomatogenic area. For this reason, aura symptoms are best interpreted as closest or functionally connected to, but not necessarily arising from, the seizure onset zone. Since an aura is only experienced with intact awareness and recall, an ictal discharge that interrupts consciousness or memory quickly can result in the absence or loss of aura symptoms. This is the likely mechanism for patients who used to have auras early in the course of the disease, but later lose them, and in patients with bilateral foci, as the ictal discharge propagates rapidly to interrupt widespread areas (Schulz *et al.*, 1995; Schulz *et al.*, 2001).

Hughlings Jackson is recognized as a pioneer in correlating the symptoms and signs of seizures to the site of onset. The next advance came from electrical stimulation studies in the awake human brain, as exemplified by the work of Penfield, Jasper and others in Montreal. This permitted a correlation of spontaneous seizure symptoms, including auras, to those elicited by electrical stimulation and has served as the basis of much of our understanding of cortical localization. Bancaud and his school introduced the term "anatomo-electro-clinical correlation" based on the temporal and spatial analysis of the seizure network in intracranial EEG recordings, with appreciation of the sequence and relationships amongst the multiple regions activated in a seizure.

Some auras closely mirror our notions of cerebral functions. Easiest to localize are those arising from or close to primary sensory cortices (*table 1*). Thus, we conceive of an elementary visual aura of bright shapes as a result of activation of the primary visual cortex in the occipital lobe. As the visual stream projects to association areas for higher order processing, aura experiences downstream become more complex in content or manifest an admixture of modalities. In the case of visual input that projects in dorsal and ventral streams to association cortices in the parietal and temporal lobes, respectively, auras from these areas may produce complex visual disturbances with characteristics of illusions (visual disequilibrium, autoscopy) or complex hallucinations with mnesic or emotional qual-

ities (Bien *et al.*, 2000; Elliott *et al.*, 2009a). Alternatively, the visual experience can be non-descript, no more than a blurring. A similar analogy may be present for auditory aura. Elementary sounds and noises indicate proximity to primary auditory cortex in the transverse gyrus of Heschl, while complex phenomena such as music relate to auditory association areas in contiguous temporal neocortex, and illusions of sound distortion may arise from more distant projections. The case of somatosensory auras is the most complex, as there are multiple brain regions that contribute to this sensory experience: primary sensory cortex in post-central gyrus, the pre-central gyrus to which it is related, the supplementary (sensori-) motor area, the second sensory area in the parietal operculum, and the insula. There may be individual differences to the quality of the somatosensory aura that help to distinguish amongst these areas, but that is often not the case. When the aura is described as tingling numbness or electrical sensation initially localized to an extremity or side of the face, moving along one side of the body in a Jacksonian march, this reliably localizes the aura to the primary sensorimotor cortex as the ictal discharge propagates along the gyrus following the homunculus. Unilateral segmental or widespread somesthetic auras can arise from each of the non-primary sensory areas, more likely from the contralateral hemisphere, but can be at times unilateral, as reported upon stimulation of the second sensory area.

When a patient reports multiple aura symptoms, it can be a result of contiguous cortical propagation causing the auras to change sequentially (Widdess-Walsh *et al.*, 2007). On the other hand, the simultaneous onset of multiple aura symptoms, or variable order or prominence of several auras in different seizures, suggests that a network linking several brain areas is activated (Gloor, 1990). Aura sensations can be secondary to spread beyond the seizure onset zone. A somatosensory aura can be reported in surgically remediated temporal lobe epilepsy even though there is no somatosensory representation in the temporal lobe (Erickson *et al.*, 2006). Auditory illusions of sounds changing can be found in frontal opercular and insular epilepsy, from connectivity to auditory cortex in the temporal lobe (Isnard *et al.*, 2004; Thompson *et al.*, 2015). More recent studies showed that the appearance of semiology may be a consequence of functional coupling or uncoupling of different neuronal structures (Chauvel and McGonigal, 2014). This hypothesis was suggested as the basis for the experiential phenomenon in temporal lobe epilepsy (Bancaud *et al.*, 1994) and an increased synchronization in medial temporal structures was demonstrated as important for the *déjà vu* aura (Bartolomei *et al.*, 2012).

In addition to suggestions on localization of the ictal onset zone, some auras can have a lateralizing value. A

Table 1. Localization of sensory modalities.

	Cerebral location	Supporting literature
Somatosensory		
Localized Jacksonian march	SI	Penfield and Jasper, 1954 Kim *et al.*, 2004
Segmental, unilateral (tingling, numbness)	SI, SII, SMA, insula	Lim *et al.*, 1994; Afif *et al.*, 2010 Ostrowsky *et al.*, 2000
Pain	SII, insula (? SI)	Shibasaki, 2004; Afif *et al.*, 2010 Ostrowsky *et al.*, 2000
Warm	SII, insula	Ostrowsky *et al.*, 2000 Bowsher *et al.*, 2004; Afif *et al.*, 2010
Cold	amygdala, insula, SII, anterior cingulate/mesial frontal	Fish *et al.*, 1993 Ostrowsky *et al.*, 2000 Seifert and Maihofner, 2007
Auditory		
Elementary (tinnitus/buzzing)	Heschl's gyrus, superior temporal	Penfield and Jasper, 1954 Liegeois-Chauvel *et al.*, 1991
Illusions (sound distortions)	lateral temporal, insula	Penfield and Jasper, 1954 Ostrowsky *et al.*, 2000 Florindo *et al.*, 2006
Visual		
Elementary (lights, shapes)	occipital	Penfield and Jasper, 1954 Lee *et al.*, 2000
Illusions (distortion, motion, autoscopy)	parietal, temporo-occipital	Lee *et al.*, 2000; Molholm *et al.*, 2006
Complex hallucinations (multimodal imagery)	temporal (mesial and lateral)	Penfield and Perot, 1963 Gloor *et al.*, 1982; Bancaud *et al.*, 1994
Olfactory		
Pleasant odour	insula	Ostrowsky *et al.*, 2000
Unpleasant odour	olfactory bulb, medial temporal	Penfield and Jasper, 1954 Andy, 1967; Kumar *et al.*, 2012 Rayport *et al.*, 2006
Gustatory		
Pleasant taste	insula	Iannilli *et al.*, 2014
Unpleasant taste	rolandic and parietal operculum, medial temporal, insula	Hausser-Hauw and Bancaud, 1987 Ostrowsky *et al.*, 2000
Vestibular	superior temporal, inferior parietal, and other sites	Kahane *et al.*, 2003
Autonomic	insula, amygdala, hippocampus, cingulate, ventro-medial prefrontal	Beissner *et al.*, 2013

SI: primary sensory cortex; SII: second sensory area; SMA: supplementary motor area.

lateralized elementary visual aura or somatosensory aura with Jacksonian march is contralateral to the seizure focus. More complex and segmental somatosensory sensations, for instance painful discomfort involving one side of the body, are suggestive of involvement of other sensory cortices, such as the second sensory area or insula, and are usually contralateral, but at times ipsilateral to the epileptogenic zone. A lateralized auditory aura is uncommon, but may also lateralize to the contralateral hemisphere. Unilateral chills and piloerection is thought to be ipsilateral to the seizure focus (Loddenkemper *et al.*, 2004). Some auras have been reported to favour the dominant or non-dominant hemisphere. For example, orgasmic aura (Janszky *et al.*, 2002) and urinary urge (Baumgartner *et al.*, 2000; Loddenkemper *et al.*, 2003) were reported from the non-dominant hemisphere. However, as these are rare auras and patient numbers small, the reliability of such observations on cerebral dominance should await further confirmation.

Despite some of these advances, our understanding of auras is far from complete, as in the example of the abdominal aura that can occur in focal epilepsy from all lobes of the brain. Penfield and colleagues elicited abdominal sensations from stimulation of the insula, opercular structures in the Sylvian fissure, and supplementary motor area. Penfield reported that abdominal sensations from insular stimulation could be accompanied by gastric motility changes (Penfield and Faulk, 1955), but this relationship was not reproducible in later studies. Van Buren reported abdominal aura or sensation in eight epileptic and six non-epileptic patients when stimulating the medial temporal structures of the amygdala and hippocampus, and also the basal ganglia (Van Buren, 1961). In addition to all these mentioned sites, a more recent stimulation study found abdominal sensation on stimulation of the anterior cingulate cortex and concluded that there is an extensive cortical network involved in the cortical processing of information from the gastrointestinal tract involving the limbic and paralimbic network (Mulak *et al.*, 2008).

Auras from the temporal lobe

The commonest auras reported in temporal lobe epilepsy include abdominal, emotional, psychic, olfactory, and gustatory auras and are more frequent in medial temporal lobe epilepsy (*table 2*). Historically, the presence of combined olfactory and gustatory aura was the defining characteristic of "uncinate fits". Somatosensory, auditory, vertiginous and cephalic auras are less commonly reported and may correlate more with the lateral neocortical compartments of the temporal lobe (*table 3*). Auras with alterations in higher

processing of visual and other inputs may implicate the association cortices in the more posterior portions of the temporal neocortex. However, as there are rich connections between the medial and lateral compartments such a dichotomization is far from precise, and experiential hallucinations arise from both medial and lateral temporal structures. Multiple auras are common in temporal lobe epilepsy, occurring in 48% to 57% (Taylor and Lochery, 1987; Ferrari-Marinho *et al.*, 2012), while there is complete absence of auras in about 10% of patients with temporal lobe epilepsy (Ferrari-Marinho *et al.*, 2012).

Psychic aura (or "experiential phenomena" or "dreamy state") is a broad category and encompasses *déjà vu*, complex visual or multimodal hallucinations, and memory flashbacks. While not the most common aura (found in 12-16% of TLE series), it may be the most specific. Some authors have included sensory illusions, emotional disturbances and feelings of strangeness in this category. Jackson in 1876 described the dreamy state in a patient with an uncal lesion. Penfield later reported experiential visual hallucinations upon lateral neocortical temporal stimulation. Later studies found medial temporal stimulation to evoke the dreamy state and other experiential phenomena more reliably (Gloor *et al.*, 1982) with the hypothesis that this produced activation of widespread neuronal matrices (Gloor, 1990). Bancaud in 1994 proposed a network involving both neocortical temporal and limbic structures, as responsible for psychic aura. Experiential phenomena were elicited more frequently upon stimulation of the rhinal cortex than the hippocampus or amygdala (Bartolomei *et al.*, 2004). Further work suggested that the experience is dependent on theta band synchronization within mesial temporal structures (amygdala, hippocampus, perirhinal and enterorhinal cortex), leading to activation of a wider neural network that includes the primary visual cortex and other association cortices. (Barbeau *et al.*, 2005; Vignal *et al.*, 2007). Overall, psychic auras suggest networked activation of temporal limbic and neocortical areas and could result from either a medial or neocortical epileptogenic focus.

Emotional auras occur in temporal lobe epilepsy, and also from the medial frontal cortex. Affective auras are reported in 10-35% of focal epilepsy (Feichtinger *et al.*, 2001; Chiesa *et al.*, 2007; Toth *et al.*, 2010). If an aura is painful or unpleasant, it may also set up a secondary anxiety or fear response which can be difficult to distinguish from ictal fear itself. Ictal fear is one of the more commonly reported affective phenomena in patients with medial temporal epilepsy (Gloor, 1990; Feichtinger *et al.*, 2001; Maillard *et al.*, 2004). Other less commonly reported affective auras include happiness, sadness, guilt, and anger. Ictal fear is sudden and may range from mild anxiety to outright

Table 2. Relative incidence of auras in focal epilepsies.

	Temporal[a]	Temporal[b]	Frontal[b]	Parietal[b]	Parietal[b]	Occipital[b]	Occipital[b]
	Binder et al., 2009	Ferrari-Marinho et al., 2012	Jobst et al., 2000	Salanova et al., 1995b	Kim et al., 2004	Salanova et al., 1992	Lee et al., 2005
Incidence of auras	114/150 (76%)	188/205 (91%)	18/26 (69%)	77/82 (94%)	27/40 (67.5%)	37/42 (88%)	22/26 (85%)
Somatosensory	12%	9%	12%	63% (11% with disturbance of body image)	32.5%	-	-
Auditory	5%	6%	-	5 %	2.5%	-	-
Visual	6%	14%	-	17 %	10%	73%	77%
Olfactory	11%	2%	-	-	-	-	-
Gustatory		4%	-	-	7.5%	2%	-
Vertiginous	3%	12%	-	11 %	10%	-	15%
Autonomic	1%	30%	12%	-	7.5%	-	-
Abdominal	26%	52%	-	1 %	-	14%	7.7%
Cephalic	-	15%	35%	3 %	-	12%	-
Psychic, Experiential	12%	16%	-	5%	-	9% (complex visual hallucination)	-
Fear	14%	19%	19%	-	15%	2%	-
Affective (not fear)	-	6%	-	-		-	-
Other	-	-	8%	3%	2.5%	-	-
Unclassified	10%	6%	15%	8%	-	2%	-

[a]Percentages refer to the frequency of a particular aura relative to the total auras reported; [b]Percentages refer to the number of patients reporting a particular aura.

Table 3. Auras in temporal lobe epilepsy.

Medial Temporal	Lateral Temporal
Abdominal	Auditory
Psychic/experiential	Vertiginous
Affective	Somatosensory
Gustatory & Olfactory	
Autonomic	
Complex hallucinations	

terror. It may be isolated or accompanied by autonomic features of pallor, mydriasis, rubefaction, piloerection and tachycardia (Biraben et al., 2001), or visceral sensations referred to the chest or abdomen (Gloor, 1990). Fear in temporal lobe epilepsy implicates involvement of the amygdala, the temporal pole with its strong projections to orbitofrontal and medial frontal cortex (Bartolomeil et al., 2002), as well as the anterior cingulate cortex (Biraben et al., 2001).

Abdominal aura, while not specific to temporal lobe epilepsy, is the one most commonly associated with it. Epigastric, visceral, or viscerosensory auras are some other terms used to describe the same phenomenon. The term encompasses a range of sensations in the

abdomen, classically in the epigastrium, sometimes rising, likened to nausea or "butterflies in the stomach", and can be painful. It was reported in 15-52% of patients with TLE (Lennox and Cobb, 1933; Palmini and Gloor, 1992; Henkel *et al.*, 2002; Binder *et al.*, 2009). A series of 491 patients reported a 52% incidence of abdominal aura in temporal lobe epilepsy as compared to 12% in extratemporal epilepsy (Henkel *et al.*, 2002). Abdominal aura in isolation occurred in 42%, but frequently was found combined with other aura types in temporal lobe epilepsy (Kuan *et al.*, 2012). When an abdominal aura evolved to automatisms, this increased the probability of a diagnosis of temporal lobe epilepsy to 98% (Henkel *et al.*, 2002).

Olfactory auras are less common in temporal lobe epilepsy, with a prevalence rate between 0.6% and 16% (Rasmussen, 1982; Acharya *et al.*, 1998; Chen *et al.*, 2003). Overall, olfactory auras were found in only 0.9% of 1,423 patients with intractable focal epilepsy (Acharya *et al.*, 1998). Its relationship is strongest with seizure foci in the medial temporal lobe, in particular, the uncal region that encompasses the entorhinal cortex. There are rare cases of tumours and lesions in the orbitofrontal region with olfactory aura. Most commonly, the olfactory sensation is unpleasant in some way, with a burnt, chemical, or sickening sensation, but not always. The anterior olfactory nucleus, piriform cortex, olfactory tubercle, and lateral entorhinal cortex constitute the primary olfactory cortex. The orbitofrontal cortex is considered a secondary olfactory cortex responsible for odour discrimination (Zatorre *et al.*, 1992). Penfield and Jasper reported unpleasant olfactory sensations upon stimulation of the olfactory bulb consistently, while stimulation of uncus produced the same only occasionally. A recent study reproduced similar findings (Kumar *et al.*, 2012) and found mostly unpleasant odour sensations by stimulating proximal to the olfactory bulb or tract, but not by stimulation of orbitofrontal gyri. There are also some case reports of olfactory aura upon stimulation of the amygdala, but not of the hippocampus (Van Buren, 1961; Andy, 1967; Rayport *et al.*, 2006).

Gustatory auras are rare and not restricted to temporal lobe epilepsy but also reported in suprasylvian epilepsies. The sensation is usually unpleasant and has been described as bitter, sharp, metallic, or acidic. Penfield (in 1937) mapped taste in the postcentral gyrus just above the sylvian fissure. Hausser-Hauw and Bancaud reported isolated gustatory hallucinations upon electrical stimulation of Rolandic and parietal operculum, the amygdala, hippocampus, and medial anterior temporal region (Hausser-Hauw and Bancaud, 1987). They proposed that gustatory hallucinations in spontaneous or electrically induced temporal lobe seizures are a result of ictal spread to the operculum by a "functional reorganization of the connections within these epilep-togenic areas". More recent stimulation studies found gustatory hallucinations upon insular stimulation as well (Ostrowsky *et al.*, 2000).

Elementary auditory auras of basic sounds and noises suggest lateral neocortical epilepsy from activation of the auditory cortex in Heschl's gyrus or surrounding cortex. Cortical stimulation studies have confirmed a tonotopic organization of the auditory cortex which shows a transition from high frequency sound, to broad band noise, to auditory illusion, while passing from primary to secondary auditory areas of Heschl's gyrus (De Graaf *et al.*, 2000). Ictal deafness, although rare, has been described in dominant hemispheric epilepsies (Ghosh *et al.*, 2001; Florindo *et al.*, 2006) and suggests ictal involvement of the primary auditory cortex. Auditory illusions characterized by distortion of sounds (louder, softer, altered pitch) may arise in auditory association cortex or may be the result of propagation from connected areas, such as the insula and inferior frontal operculum (Isnard *et al.*, 2004; Thompson *et al.*, 2015). More complex auditory hallucinations and verbal hallucinations are of particular interest. They suggest ictal involvement of the neocortical temporal cortex. Verbal hallucinations most likely arise from altered processing in auditory association cortex in the superior temporal gyrus and adjacent posterior language areas of the dominant hemisphere (Serino *et al.*, 2014).

Somatosensory, vertiginous, and autonomic auras also occur in temporal lobe epilepsy. The amygdala and hippocampus are part of the central autonomic network, but in any given seizure arising from the temporal lobe, it is difficult to separate the contribution of secondary activation of insular and medial frontal regions to the experience of autonomic symptoms, given the strong connectivity amongst the different structures. Somatosensory auras, when encountered in temporal lobe epilepsy, are the presumed result of ictal spread, and are discussed later in the section on auras from the parietal lobe, as are vertiginous auras which implicate a network in posterior temporal and inferior parietal regions.

Auras from the insula

The insula or the "island of Reil" was the least explored cortex due to its difficult accessibility deep in the Sylvain fissure until stereotactic depth electrode exploration was refined and shown to be safe. Seizures arising from the insular cortex can mimic those of temporal, frontal and parietal lobe epilepsy because of its robust connectivity with these other areas. In spite of much recent work, it still remains uncertain how much of the semiology of insular seizures is purely the result of activation of the insular cortex and

how much is due to its propagation to other areas. Resting state fMRI studies (Cauda *et al.*, 2011) showed two major insular networks. The anterior insular network connects with the rostral anterior cingulate, middle and inferior frontal, and temporoparietal cortices. The posterior insula network connects to the dorsal posterior cingulate, sensorimotor, premotor, supplementary motor, temporal cortices and some occipital areas. Cortico-cortical evoked potentials upon insular stimulation in eleven patients studied by stereo EEG electrodes showed that the highest connectivity rate was between the insula and peri-sylvian cortex, while the highest long distance connectivity was with the peri-central regions followed by the amygdala, posterior hippocampus and lateral temporal neocortex (Almashaikhi *et al.*, 2014). Electrical stimulation of primates (Hoffman and Rasmussen, 1953) and humans (Penfield and Faulk, 1955) had long shown the insula to be a part of the autonomic network. Stereo EEG stimulation mapping in 14 patients (Ostrowsky *et al.*, 2000) demonstrated a role of the anterior insula in viscerosensitive and visceromotor functions while the posterior insula was more involved with somatic sensations.

From stereo-EEG explorations, a picture of the semiology of focal seizures originating from the insula has emerged. In five such patients (Isnard *et al.*, 2004), the most distinctive auras reported were laryngeal discomfort associated with retrosternal sensation or dyspnoea, unpleasant paresthesias, and painful or warm sensations, either perioral or over large cutaneous areas. It was possible to reproduce these sensations and also pain upon insular stimulation (Ostrowsky *et al.*, 2000; Mazzola *et al.*, 2012). Another series of four patients reported similar somatosensory sensations, with throat and laryngeal paresthesia as part of their seizure semiology. One out of the four patients also had fear, anxiety and *déjà vu* (Nguyen *et al.*, 2009a, 2009b). Other single case studies also found fear, throat tightening, bad taste and vibrating limb sensation as auras in insular epilepsy (Fiol *et al.*, 1988; Kriegel *et al.*, 2012).

Auras from the frontal lobe

Auras in frontal lobe epilepsy are variable, and may not be strongly localizing. In fact, of all lobar epilepsies, frontal lobe epilepsies may stand out for the lack of an aura. Palmini reported that 61% of the patients experienced an aura in their retrospective group while auras were present in 43% of the prospective group (Palmini and Gloor, 1992). More commonly reported were cephalic and somatosensory auras while viscerosensory, experiential and vertiginous auras were less often found. This section discusses auras from the

anterior and premotor frontal cortex, as the Rolandic cortex is grouped with the parietal lobe.

Auras in premotor frontal lobe epilepsy are highly variable. Patients with seizures arising from the supplementary motor area (SMA) and superior frontal sulcus reported somatosensory, visual, cephalic auras and intellectual changes (Beauvais *et al.*, 2005). Of interest was a patient who experienced a visual illusion from ictal discharge in dysplastic cortex in the superior frontal sulcus (Beauvais *et al.*, 2005). Somatosensory aura was found in 45% of patients with SMA seizures (Salanova *et al.*, 1995a), and relates to sensory representation in the SMA, as revealed by electrical stimulation (Lim *et al.*, 1994).

Cephalic aura can be any sensation referred to the head including tingling, pressure, dizziness, fullness, or other indescribable sensation. It is reported more often in frontal lobe epilepsy as compared to the other lobes, but cannot be considered to have localizing value. The sensation can be difficult to separate from non-specific lightheadedness or dizzy feelings. It was found in 24-35 % of frontal epilepsy patients (Palmini and Gloor, 1992; Jobst *et al.*, 2000). Attempts have been made to further localize these sensations. A patient studied by MEG was found to have a midline frontocentral source corresponding to the location of the SMA during a cephalic aura (Canuet *et al.*, 2008). Another case with simultaneous EEG and MEG recording during a cephalic aura showed ictal changes in the right frontal lobe (Kakisaka *et al.*, 2014). However, how the neurophysiological changes produce this sensation remains unclear.

Ictal fear and emotional change are seen in medial temporal lobe epilepsy involving the amygdala, but also in medial frontal epilepsy (Jobst *et al.*, 2000). Ictal fear occurred in 50% of six patients with anterior cingulate epilepsy and one patient had concurrent somatosensory aura and a sensation of freezing (Alkawadri *et al.*, 2013). SEEG and SPECT studies showed that fearful ictal behaviour is the result of imbalances in a network that includes the amygdala, orbitofrontal and anterior cingulate cortex (Biraben *et al.*, 2001; Bartolomeil *et al.*, 2002).

Auras from the parietal lobe

In seizures arising from the postcentral primary sensory cortex (SI), a somatosensory aura that is contralateral and localized at onset (Beleza and Pinho, 2011; Bonini *et al.*, 2013) with or without Jacksonian march (Manford *et al.*, 1996) is almost invariable. The sensation is usually elementary: tingling, numbness, electrical shock-like. Sometimes a sensation of localized tightness is reported which may be due to increased motor tone in that particular region. It may

occur before or simultaneous with motor manifestations. It should be noted that not only seizures from the postcentral gyrus produces an elementary somatosensory aura, but also those from the pre-central gyrus.

As a large part of the parietal lobe is non-eloquent, the symptomatogenic zone responsible for auras may be remote from the seizure onset zone and activated as a result of ictal propagation (Salanova, 2012). The most consistent parietal lobe auras are somatosensory and vertiginous auras. Other reported auras are visual illusions, affective changes and somatic illusions.

Somatosensory auras, as the most frequently reported auras in parietal lobe epilepsy, occur in 30-63% of patients studied (Salanova *et al.*, 1995b; Kim *et al.*, 2004; Bartolomei *et al.*, 2011). The sensation is usually unilateral and contralateral to the epileptogenic zone and found in seizures from each of the sub-compartments of the parietal lobe (superior parietal lobule -BA 7, superior parietal lobule -BA 5, inferior parietal lobule, and parietal operculum) (Bartolomei *et al.*, 2011). Somatic sensations also arise in the second sensory area (SII) in the parietal operculum, and the posterior insula. SII has topographic representation for somatic sensations (Mazzola *et al.*, 2006; Mazzola *et al.*, 2012) including those of pain and temperature. Painful somatosensory auras suggest involvement of SII and the posterior insula, with some involvement of the opercular portion of SI (Montavont *et al.*, 2015). Whether restricted epileptic activity in SI also produces pain is a matter of debate. Connectivity of the insula with parietal operculum and mesial and lateral parietal cortex has been demonstrated by cortico-cortical evoked potentials (Almashaikhi *et al.*, 2014). This knowledge is fundamental to the understanding of various types of somatosensory symptoms in parietal lobe epilepsy.

Vestibular auras were first ascribed to temporo-perisylvian epilepsy (Behrman, 1955; Smith, 1960) but the network for vestibular representation may be much more widespread. Vestibular auras have been described also from the frontal lobe (Kluge *et al.*, 2000) and superior parietal lobule (Bartolomei *et al.*, 2011). Other reports suggested the temporo-parieto-occipital junction as an area of interest for epileptic vestibular disturbances (Erbayat Altay *et al.*, 2005; Hewett *et al.*, 2011; Hewett and Bartolomei, 2013). Electrical stimulation revealed a widespread multisensory cortical network for vestibular processing with the "temporo-perisylvian vestibular cortex" located in the superior temporal neocortex and lateral aspect of the inferior parietal cortex (Kahane *et al.*, 2003). Other sites that produced vestibular symptoms in this study of 28 patients were the inferior frontal premotor cortex, SMA, anterior cingulate gyrus, hippocampus, amygdala, posterior insula, medial occipital cortex, and the antero-inferior part of the precuneus. A case report of

a patient with a right paramedian precuneus tumour and vertiginous seizures reproduced the symptoms on electrical stimulation of the precuneus (Wiest *et al.*, 2004). Blanke *et al.* reported uni- (contralateral rotational) and bi-directional (side to side) vestibular sensations by stimulating the inferior parietal lobule near the anterior part of the intraparietal sulcus (Blanke *et al.*, 2000a).

Various types of visual and somatic illusions have been reported from the parietal lobe. They include kinetopsia, autoscopic phenomena, visual hallucinations of complex scenes, teleopsia, tunnel vision, and macropsia. Evidence for the superior parietal lobule in multi-sensory integration of visual and auditory information and visual and somatosensory information is derived from intracranial recordings (Molholm *et al.*, 2006) and imaging studies (Sereno and Huang, 2014). One proposal suggests autoscopic phenomena are a result of failure to integrate proprioceptive, tactile and visual information along with vestibular dysfunction (Blanke *et al.*, 2004; Blanke *et al.*, 2005). Other proposals hypothesize that autoscopic phenomena during seizures are due to a functional disconnection between prefrontal cortex and the temporo-parietal junction (Easton *et al.*, 2009), or ictal disturbance of the normal integration process of body representation within the parieto-occipital network (Anzellotti *et al.*, 2011).

Auras from the occipital lobe

Auras in occipital lobe epilepsy, as would be expected, are mostly visual. They may be in the form of elementary visual hallucination or more complex visual illusions. They may also be in the form of negative symptoms like blindness or visual field defects (Panayiotopoulos, 1999). Less commonly, patients reported dizziness, psychic symptoms and abdominal sensations in occipital seizures secondary to ictal spread (Salanova *et al.*, 1992; Lee *et al.*, 2005; Yang *et al.*, 2015).

Elementary visual auras, usually in the form of spots, lights, or other geometric shapes, which can be white, black, or coloured; stationary or moving, indicate ictal onset or early ictal spread to the calcarine cortex (Palmini *et al.*, 1993). Complex visual hallucinations of fully formed scenes and images suggest involvement of a wider neural network in visual association cortices with a more diffuse anatomical substrate (Elliott *et al.*, 2009b). In a series of 878 surgically treated patients with epilepsy, 20 were shown to have visual aura (Bien *et al.*, 2000) which was reported not only for patients with occipital lobe epilepsy but also with occipitotemporal and anteromedial temporal epilepsy. The limbic network is involved in the generation of complex visual symptoms with memory or emotional content, such

as the recall of scenes from the past (Gloor *et al.*, 1982; Fish *et al.*, 1993). Visual illusions like micropsia, macropsia and metamorphopsia were produced by electrical cortical stimulation of basal temporo-occipital cortex (Lee *et al.*, 2000). Ictal visual motion illusions, kinetopsia, have been reported in seizures arising from the temporo-parietal-occipital junction (Laff *et al.*, 2003), superior parietal lobule, and intra-parietal sulcus (Perumal *et al.*, 2014).

There are rare reports of visual auras arising from the frontal lobe (Fornazzari *et al.*, 1992; La Vega-Talbot *et al.*, 2006; Ye *et al.*, 2012). Electrical cortical stimulation of the prefrontal cortex has produced complex visual hallucinations (Blanke *et al.*, 2000b), particularly facial hallucinations (Vignal *et al.*, 2000). Primate as well as human studies suggest that both the dorsal and ventral visual streams extend into the prefrontal cortex (Wilson *et al.*, 1993; O Scalaidhe *et al.*, 1997; Ungerleider *et al.*, 1998). One hypothesis for the complex visual hallucinations from prefrontal cortex is that there is interference of the ventral visual object-process stream (Courtney *et al.*, 1996). Another possible explanation for facial hallucinations from the prefrontal cortex is its interaction with the fusiform gyrus, an established site for facial recognition (Marinkovic *et al.*, 2000).

Conclusion

The importance of epileptic auras lies in the fact that they can help the clinician in better understanding brain function and a patient's epilepsy. With the understanding of epileptic networks, an ictal pathway can be traced by utilizing the semiological information available and the aura (when present) is of utmost importance as it precedes any other sign. This information can guide further investigations, treatments and surgical evaluation, as needed. □

Supplementary data.
Summary didactic slides are available on the www.epilepticdisorders.com website.

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TEST YOURSELF

(1) What is the difference between aura and prodrome?

(2) Approximately what percentage of neurons are recruited during an aura?

(3) What aura sensations are most specific to insular seizures?

Note: Reading the manuscript provides an answer to all questions. Correct answers may be accessed on the website, www.epilepticdisorders.com, under the section "The EpiCentre".