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## Auditory manifestations during focal seizures

Laura Tassi

Centro Chirurgia Epilessia "Claudio Munari", Ospedale Niguarda Cà Granda, Milano, Italy

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Anatomo-electro-clinical correlations permitted to define the site and the extension of the epileptogenic zone (EZ) in focal epilepsies and are mandatory in the pre-surgical work-up.

Well defined protocols permit to obtain MR highdefinition images; however even the more performing MR could be negative if the epileptologist doesn't aim the investigation to the previously defined EZ.

Interictal and particularly ictal surface and invasive recordings allow, in most of cases, the precise localization of the cortical area responsible of ictal discharges. During surface Video-EEG the localization could be a little complicated, considering the presence of artefacts, but the occurrence of a low voltage fast activity or a flattening, permits, coupling with the onset of clinical symptomatology, to define a hypothesis concerning the localization and the extension of the EZ.

Clinical symptomatology represents an enormous "reservoir" of information, but the epileptologist must be able to make a correct use.

Every patient has a different, peculiar epilepsy, and a variety of signs and symptoms. The chronology of occurrence of every subjective manifestation and objectivable sign, defines the neuronal pathological network: ictal discharges, with different modality of duration and intensity, involve cortical and subcortical regions, determining a "dysfunction" of the EZ, but also of the bordering areas (Chauvel *et al.* 1987). This neuronal process accounts for minimal differences during distinct seizures in the same patient.

The worst error, in epilepsy surgery, is to define the extension and localization of the EZ on the basis of a SINGLE sign or symptom. Seizures are the multimodal expression of the cortical involvement with a spatio-temporal dimension. Only the very initial clinical modifications rise to high localization value.

On the other hand, only few signs or symptoms allow an accurate and unquestionable localization: auditory illusions and hallucinations, lateralized simple visual hallucinations, clonic/hypertonic modification of the limbs. Other clinical modifications could be predominantly located in some regions (as psychic manifestations into the temporal lobe). Oro-alimentary automatism are generally associated with temporal lobe seizures. It is just the opposite for symptoms like oculo-cephalic deviations, gestural automatisms, the presence of a loss of contact, olfactory hallucinations (occurring in temporal but also during orbitary seizures).

According to the international classification, auditory manifestations usually occur in neocortical temporal lobe epilepsy.

Therefore, the presence of auditory modifications is consistent with a temporal origin of ictal discharges, and, particularly, auditory illusions are located in the anterior-middle part of the first temporal gyrus, and auditory hallucinations in the posterior and medial part of T1 (Heschl's gyrus). In general, auditory manifestations are present in the two ears, and may be prominent on the contralateral one.

We know that auditory manifestation is an uncommon phenomena, concerning about 1,5% of patients suffering from focal epilepsy (Florindo *et al.* 2006), and that are correlated with lateral onset of ictal discharges in temporal lobe epilepsy (Maillard *et al.* 2004).

We possess a series of experimental data (fMRI, animal models, MEG, cytoarchitectural studies) demonstrating that:

- the primary auditory cortex (also known as A1, or BA 41–42) includes Heschl's gyrus on the floor of the Sylvian cistern; the unimodal auditory association cortex deals with the superior temporal gyrus (BA 22) and is probably also parts of the middle temporal gyrus (BA 21) in the human (Mesulam 1998);

- MEG spike sources clustered in the superior temporal gyrus in patients with auditory auras (Mohamed 2006);

– using functional MRI (fMRI), the processing of sound coming from different directions has been found to activate complex neuronal pathways which not only included Heschl's gyrus and the superior temporal lobe but also the inferior and middle frontal gyri (Brunetti *et al.* 2005);

– PET and MEG show a similar complexity to the auditory pathway with involvement of both the frontal and temporal lobe in individuals with no identified brain pathology (Brunetti *et al.* 2005);

– repeated epileptiform activity interferes with temporal processing in cat auditory cortex in the interictal state. This may have implications for people with epileptic foci in auditory-related areas (Valentine *et al.* 2005).

Clinical series of patients with auditory symptoms, studied with invasive methodologies, demonstrate the first temporal gyrus as the responsible of different kinds of illusions/hallucinations encompassing the auditory spectrum (Gosh *et al.* 2001, Florindo *et al.* 2006, Mohamed *et al.* 2006, Clarke *et al.* 2003).

"The auditory hallucinations consist of elaborate phenomena of music or voices, whereas the illusions consist of modifications of intensity, tonality or resonance of surrounding voices. Unlike elementary auditory auras, which have a focal origin on the auditory cortex, complex auditory hallucinations and illusions tend to be elicited from more widespread areas involving the planum temporale or the lateral part of the superior temporal gyrus" (Mohamed *et al.* 2006).

We are, unfortunately, unable to detect the subcortical transmission of the ictal discharge.

In all probability more complex experiences of auditory sensation need the involvement of multimodal association cortical areas, including extratemporal regions.

Furthermore, the location of Heschl's gyrus on the mesial surface of the first temporal cirnconvolution, makes its functional exploration complex; subdural investigation is not sufficient to comprise all the auditory areas, particularly the medial part, and "the only way to record Heschl's gyrus is to place a depth electrode inside it" (Gupta 2006).

Finally every epileptologist owns precious information from the ictal semeiology of patients, but only the continuous correlation with neurophysiological and neuroradiological data could allow the definition of the pathway of propagation. Only very initial signs have a localization value and only few signs have a real localizing meaning.

## Correspondence:

L. Tassi

Centro Chirurgia Epilessia "Claudio Munari", Ospedale Niguarda Cà Granda, Milano, Italy <Laura.Tassi@OspedaleNiguarda.it>

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