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Auris Nasus Larynx 37 (2010) 314–321

AURIS NASUS LARYNX
INTERNATIONAL JOURNAL OF ORL & HNS

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# Eye movement abnormalities in somatic tinnitus: Fixation, smooth pursuit and optokinetic nystagmus

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Received 12 May 2009; accepted 16 October 2009
Available online 17 November 2009

**Abstract**

*Objective:* Smooth pursuit (SP), optokinetic nystagmus (OKN) and fixation were investigated in five subjects with somatic tinnitus modulated by eye movements, jaw or neck.
*Methods:* Eye movements were recorded with the EyeLink II video system.
*Results:* (1) Fixation was characterized by high frequency and amplitude of saccade intrusions; (2) SP had low gain particularly in the vertical direction, and it was characterized by high frequency of catch-up saccades with high amplitude, including predictive saccades; (3) OKN also had low gain particularly in the vertical direction. Each subject showed abnormality for more than one type of eye movement, and for specific directions.
*Conclusions and significance:* The results suggest mild dysfunction of cortical–subcortical and cerebellar structures involved in the control of these eye movements. Particularly deficits for vertical pursuit eye movements and fixation instability in line with cerebellar signs. Further studies of more patients with or without modulated tinnitus are in progress.


*Keywords:* Tinnitus; Fixation; Smooth pursuit; Optokinetic nystagmus; Abnormality

## 1. Introduction

Recent studies have shown abnormalities of smooth pursuit (SP) and optokinetic nystagmus (OKN) for patients with tinnitus such as gain decrease in SP and incorrect OKN [1,2]. These authors suggested that persistence of abnormal oculomotor findings could be a manifestation of central vestibular system impairment. Other studies reported a special tinnitus, i.e. gaze-evoked tinnitus (GET), the loudness and pitch of tinnitus increase with lateral gaze [3,4] by use of Positron-Emission-Tomography (PET) to do scans with eyes fixed on the target in the central gaze position, on the right target, or on the left target. They found that GET-associated gaze appeared to activate the cuneus (in the occipital lobe) and the cerebellar vermis. However, there is no quantitative analysis of fixation in patients with tinnitus. Even for normal population the fixation is not perfectly stable. For instance, saccade intrusions (SI), small in amplitude less than 0.5°, can occur at frequency about 60 times/min [5]. SI consist of conjugate, horizontal saccadic eye movements that take the form of an initial fast eye movement away from the desired eye position, followed after a variable duration by a return saccade or drift. The etiology of SI remains unknown. Many hypotheses have been proposed for production of SI, such as omni pause neuron dysfunction [6], a spurious spranuclear error signal [7], enlarged microsaccades [8,9] or altered superior colliculus [10]. On the other hand, it is also possible that they are a consequence of descending commands related to

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0385-8146/$ – see front matter 
doi:10.1016/j.anl.2009.10.004

attentional shifts arising from areas such as the parietal cortex, anterior cingulate, thalamus, basal ganglia or cerebellum [11–15]. Activity from these areas may stimulate the SC build-up cells that have been suggested as possible neurophysiological correlates of attention allocation, resulting in a saccade [16]. This would suggest that the fixation system, rather than existing as a separate entity or as a part of another system such as smooth pursuit, is comprised of a network of interacting areas such as the dorsolateral prefrontal cortex, frontal eye fields, basal ganglia and SC that are governed by a supramodal attention system and task demands [17,18].

Thus, this study aims to further analyze quantitatively eye movements, such as SP, OKN but also fixation in five cases of tinnitus. Patients were selected because their tinnitus was modulated by gaze, and/or jaw or neck movements.

## 2. Methods

### 2.1. Patients

All patients attended a tertiary care tinnitus clinic at the European Hospital Georges Pompidou in Paris; tinnitus perception being stated as their main medical complaint. They were selected because they showed in common modulation of their tinnitus perception by oro-facial, neck or eye movements. The investigation adhered to the tenets of the Declaration of Helsinki and was approved by the institutional human experimentation committee. This study has been approved by our local ethical committee and patients gave informed consent before testing. Epidemiological data are given in Table 1. All patients suffered from tinnitus for at least one year (mean 9.6 years). Mean age was 53.3 years; sex ratio was 3 women for 2 men. Tinnitus was left sided in 3 patients (1 with oblique) and right sided in one patient. One patient had bilateral tinnitus but predominantly heard on the left side (with downward gaze also). All five patients heard a high pitch tone (described as a hissing) even though one described the concomitant perception of a broadband noise.

Hearing impairment was present in two patients (i.e. left cophosis after acoustic schwannoma surgery via translabyrinthine approach, and moderate high frequency sensory neural hearing loss of indeterminate origin). Other subjects showed hearing within normal range. No patient had history of middle ear or tympanic pathology. Suspected aetiology of tinnitus onset was head trauma in one patient, acoustic schwannoma removal in one patient, stress associated with abnormal oculomotricity (decompensated exophoria) in one patient. For two other patients tinnitus happened without any clearly identified cause.

As measured by means of the French version of Hospital Anxiety Depression scale, four patients showed a pathological anxiety state and two a depression state.

Ongoing therapy during oculomotor tests consisted of sound therapy (sound enrichment in order to alleviate tinnitus perception and handicap), or medical therapy (clonazepam 0.5 mg/day); note that only a large dose of 2 mg clonazepam can reduce the peak velocity of saccades [19].

Two patients (T1 and T3) also described fluctuations of tinnitus intensity after periods of sleep (during the night or after short periods of naps during the day).

### 2.2. Oculomotor tests

The subject was comfortably seated in an adapted chair with a chin rest to stabilize the head; he/she viewed light dot target binocularly. The stimulation was presented on the PC screen; the PC screen was placed at 57 cm from the subject.

### 2.3. Eye movement recording and analysis

Horizontal and vertical eye movements were recorded binocularly with the EyeLink II device. Each channel was sampled at 250 Hz. The system has a resolution of 0.025°. Before each type of eye movement, the subject made a sequence of saccades of 10° between the white dots, in the four directions; each dot was turned on during 1000 ms (a period of time allowing accurate and stable fixation). The calibration factors for each eye were extracted from these recordings; a linear function was used to fit calibration data.

#### 2.3.1. Fixation task and analysis

Five dots were presented as in Fig. 1 consecutively by sequence at center, at 10° left, at 10° right, at 10° up or at 10° down; each dot lasted for 20 s. Vertically, the central dot was

Table 1
Epidemiological data.

| Subject | Age | Sex | Direction | Pitch | Aetiology | Duration | Hearing impairment | Hyperacusis | Dizziness | Anxiety | Depression | On going therapy |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| T1 | 64 | F | L > R | High | Stress, diplopia | 8 years | Moderate | No | No | Yes | No | Sound therapy |
| T2 | 45 | F | L | High, WN | Acoustic neuroma | 6 years | Yes | Yes | Yes | Yes | Yes | Clonazepam |
| T3 | 43 | M | R | High | Idiopathic | 3 years | No | No | Yes | No | No | Clonazepam |
| T4 | 58 | F | L | High | Head trauma | 30 years | No | No | Yes | Yes | No | Sound therapy |
| T5 | 44 | M | L | High | Idiopathic | 1 year | No | Yes | No | Yes | Yes | Hypnotics |

*Note*: SNHL: sensory neural hearing loss; WN: white noise; L: left; R: right.