Clinical Electrocochleography

By JOHN A. FERRARO & ROGER A. RUTH

The use of electrocochleography (ECoG) as a tool in the diagnosis, assessment, and monitoring of certain otologic and audiologic disorders has attracted renewed attention in the United States. In general, ECoG is a method of recording the stimulus-related potentials of the inner ear and the auditory nerve. The specific responses recorded may include the cochlear microphonic (CM) and components of the summating potential (SP) of the inner ear, and the whole-nerve or compound action potential (AP) of the auditory nerve.

The widespread acceptance and application of auditory evoked response testing in general, and the auditory brainstem response (ABR) in particular, have certainly contributed to the growing popularity of clinical ECoG. In addition, technological advancements in computer-based recording systems and techniques have made these very early responses easier to extract. We can, in fact, obtain reliable, clinically useful information using noninvasive, extratympanic recording techniques.

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Historically, it is generally acknowledged that the first recording of human CM was reported by Fromm et al. in 1935.[1] By 1950, the CM had been recorded by numerous other investigators using electrodes on or near the round window in subjects undergoing ear surgery. The human auditory nerve AP was first recorded by Ruben and his coworkers, also during ear surgery.[2] The application of signal-averaging techniques allowed for nonsurgical recordings,[3] and the subsequent evolution of clinical ECoG to the current era has generally involved the application of *transtympanic* recording methodology.

As implied by the term, transtympanic techniques involve passing the primary, recording electrode through the eardrum to rest on the promontory, or round window. This usually necessitates the use of mild sedatives and/or anesthetics and, of course, can be discomforting to the patient. With noninvasive, extratympanic techniques, the amplitude of the recorded response is considerably reduced, but there is no need for sedation/anesthetics, no discomfort to the patient, and a physician does not have to be present to insert the primary electrode and monitor the status of the patient during the recording session.

The above factors have certainly facilitated the clinical application of ECoG, at least in the United States. Perhaps the most important reason for the growing interest in this particular technique, however, is that several studies have now shown that the information derived from an examination may be helpful in the objective identification and monitoring of Ménière's disease and endolymphatic hydrops (e.g., Schmidt et al, 1974; Gibson et al, 1977; Kitahara et al, 1981; Coats, 1981; Ferraro et al, 1983, 1985a).[4-9]

This article is designed to provide an overview of current techniques and applications of clinical ECoG. As such, the primary emphasis will be on the use of extratympanic ECoG, primarily in patients suffering from symptoms associated with Ménière's disease/endolymphatic hydrops (MD/ELH).

AUDITORY STIMULUS-RELATED RESPONSES

The stimulus-related potentials of the auditory system generally recorded in ECoG include the receptor potentials of the inner ear (i.e., the CM and SP), and the whole-nerve or compound AP of the auditory nerve. Since its discovery by Wever and Bray in 1930,[10] the CM has been perhaps the most thoroughly investigated potential of the inner ear. It is an alternating-current (AC) response generated by the hair cells of the organ of Corti. At low-to-moderate stimulus intensities, the CM reflects the displacement-time pattern of the cochlear partition.[11] Whether the CM has a role in transduction at the hair-cell level or is merely an epiphenomenon of the process is still a disputed issue. Some investigators have reported changes in CM characteristics in patients with suspected endolymphatic hydrops.[12,13] It has been our observation, however, that

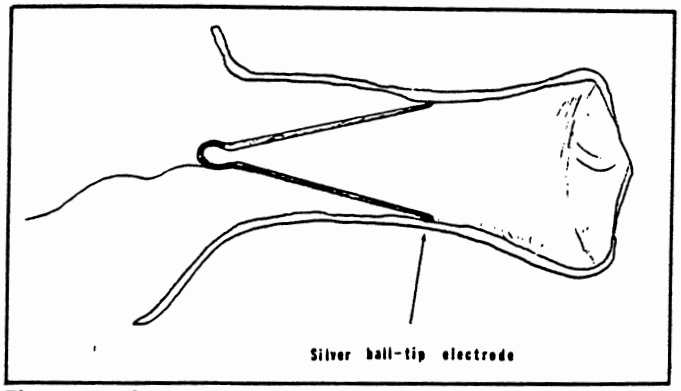

Figure 1. Schematic representation of Life-Tech Eartrode wedged into ear canal.

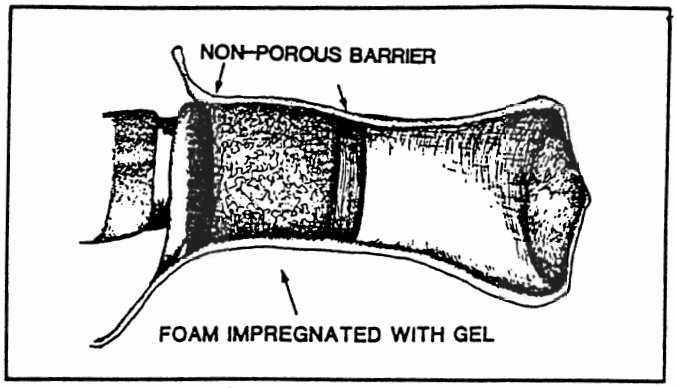

Figure 2. Schematic diagram of 3M Otodiagnostics ear canal electrode in place.

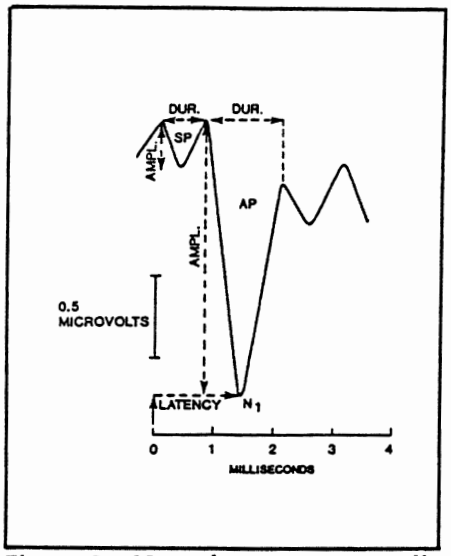

Figure 3. Normal, extratympanically recorded ECoG response to click stimuli (95 dB HL). Amplitudes and durations measured are defined and labeled. Solid arrow indicates stimulus onset. (From Ferraro et al., 1985, page 72).

response variability and the difficulty encountered in accurately interpreting the CM make this particular potential less suitable than others for study in Ménière's disease/endolymphatic hydrops.

The SP was identified by Davis et al. and independently by von Bekesy in the early 1950s. Adequate and conclusive evidence regarding its properties and role, however, is still lacking. This is due, in part, to the complexity of the response and the dependence of the SP's properties on the interaction between the parameters of the evoking stimulus and the location of the recording electrode(s). Like the CM, the SP arises from the organ-of-Corti hair cells in response to acoustic stimuli. Unlike the CM, however, the SP is a direct-current (DC) response that mimics the envelope of the eliciting stimulus.[11] Being a DC response to an AC stimulus implies that at least some of the components of the SP are representative of cochlear mechanical or mechanoelectrical nonlinearity.

In general, those studies investigating the use of electrocochleography in the Ménière's population have shown that the ECoG waveform is often characterized by an abnormally enhanced SP in patients with suspected endolymphatic hydrops. The rationale generally given for this finding is that an increase in endolymphatic volume and intralabyrinthine pressure will alter the hydromechanical properties of the inner ear. When this occurs, the normal vibratory asymmetry of the basilar membrane will be augmented. This augmentation in turn will be reflected in the SP. Support for this hypothesis is indirect, and it is obviously difficult to verify in humans. Other factors such as biochemical and/or circulatory changes may also play a role.[14] Regardless of specific pathophysiology, however, the ECoG response recorded from patients complaining of symptoms associated with MD/ELH often is characterized by an enlarged SP component.

Historically, the most popular ECoG response studied has been the auditory nerve AP.[2,15,16] Recorded from several sites on or near the nerve itself, the AP represents the summed response of several thousand nerve fibers that have fired in synchrony. AP recorded in response to click stimuli is generally referred to as *whole-nerve AP*, whereas the term *compound AP* is used when tone bursts are the evoking stimuli. During conventional ECoG recording using clicks or tone bursts, it is the synchronized population of neurons in the basal, high-frequency portion of the cochlea that produces the response.[17]

Although it is the SP that appears to be most directly affected by hydrops, the specific parameter of the overall ECoG response that seems to offer the most diagnostic consistency is the amplitude ratio between the SP and the AP.[7,18] With respect to its application in MD/ELH, therefore, the ECoG response used clinically generally consists of a complex waveform containing the SP and AP.

TABLE 1. ECoG Recording Parameters.

Electrode Montage	
Primary Site (voltage positive):	ipsilateral ear canal
Secondary Site (voltage negative):	contralateral ear canal
Ground:	forehead
Signal Averager Settings	
Repetitions:	1000 to 2000
Analysis Time:	5 to 10 milliseconds
Amplification:	50,000 to 100,000X
Filter Band-Pass:	3 Hz to 10 Hz - 3000 Hz
Stimuli	
Type:	broad-band clicks
Duration:	100 microsecond electrical pulse
Hearing Level:	begin at maximum output (90 dB-95 dB)
Polarity	alternating
Repetition Rate:	9.7/second

RECORDING TECHNIQUES

The parameters used to evoke and record the ECoG waveform are illustrated in Table I. As can be seen, these are very similar to those used for recording ABR, with some notable exceptions. First of all, the electrode montage is such that the primary recording site is the ear canal of the stimulated ear. The secondary site is the contralateral ear canal or earlobe, with ground at the forehead. When recorded in this manner, negative polarity will be displayed in a downward direction.

It is appropriate at this time to provide a brief discussion of extratympanic recording electrodes. Although several investigators construct their own, ECoG electrodes have become commercially available. Among the most popular is a silver, ball-tip electrode, or Eartrode, designed by A.C. Coats and manufactured by Life-Tech, Inc. (Houston, Texas). As shown in Figure 1, the ball tip is glued to a strip of mylar plastic that serves to "wedge" the electrode and hold it in place within the ear canal. More recently a disposable, foam, plug-like electrode and recording system, the Enhancer I, has been introduced by 3M Otodiagnostics, Inc. (St. Paul, Minnesota). As seen in Figure 2, the 3M earplug consists of front and back, nonporous rubber discs separated by a reticulated foam center. The back disc attaches to a plastic horn that restricts insertion depth and couples the electrode to a headband module. The module contains an acoustic transducer connected to the plastic horn (and electrode) via a concentric sound delivery tube. A recent study reported by Ferraro, Murphy, and Ruth suggests that the 3M earplug, when compared to the Eartrode and/or to a surface electrode over the mastoid process, is potentially the most suitable for clinical purposes, primarily because of its relative ease of application.[19]

Another exception to general ABR techniques is that the filter bandpass of the biologicial preamplifier must be expanded for ECoG. This is because we are attempting to record both an AC (the AP) and DC (the SP) response. Even though the high-pass cut-off is very low (3 to 10 Hz), there will still be distortion of any DC component amplified through an AC system. In addition, the SP response to click stimuli is difficult to define under ideal recording conditions. In essence, as we have noted, we probably are recording a distorted reflection of the SP.[8] Nonetheless, these recording parameters do allow us to visualize what is being labeled in the literature as the SP component of the ECoG waveform.

Finally, with respect to recording techniques, it should also be noted that our initial responses are recorded at maximum Hearing Level in order to evoke a well-defined SP-AP complex. Stimulus polarity is alternated to inhibit the appearance of CM and stimulus artifact.

RESPONSE CHARACTERISTICS

Figure 3 illustrates a normal, extratympanically recorded ECoG response to click stimuli.[9] The components or portions of the waveform routinely measured are labeled and include (1) the SP (amplitude and duration), and (2) the AP (amplitude, duration, latency of the first negative peak -N1). The SP/AP amplitude ratio is also calculated, from these measurements.

The absolute amplitudes of the AP and SP show considerable variation within and across subjects, making the clinical utility of these values questionable. SP duration, which should reflect the duration of the stimuls envelope, is very stable and will vary (in normals) according to the characteristics of the acoustic transducer and sound-delivery system. AP duration is also very stable and, from our data, will generally range between 0.8 - 1.2 milliseconds across subjects. It has been our experience that the clinical significance of changes in SP and/or AP duration is also questionable. AP-N1 latency is very stable, and should be identical to the latency of Wave I of the ABR,

because these are one and the same.

A more consistent amplitude feature within and across subjects is the SP/AP amplitude ratio. In our laboratory, this ratio usually varies between 0.1 - 0.5, with a mean at approximately 0.25. As noted, it is this particular feature of the overall response that seems to be most sensitive to the suspected presence of endolymphatic hydrops.

As in all other evoked-response testing, normative data may vary from lab to lab, depending on such factors as equipment and testing environment. For example, the mean latency of the AP-N1 at 95 dB HL recorded with the Life-Tech Eartrode is 1.5 milliseconds in our laboratory. If the 3M Enhancer 1 is used, mean latency is 1.8 milliseconds. The 0.3-millisecond delay is due to the travel time of the acoustic stimuli through a sound-delivery tube. Thus we recommend that, as with the ABR, norms for ECoG be established specific to individual laboratories.

The ECoG waveform shown in Figure 3 represents an ideal "normal" response in which both the SP and AP components are clearly observed and thus easily measured. Figure 4 illustrates some of the normal variation that can occur in clinical ECoG recordings. The points selected for calculation of the SP and AP amplitude are indicated on each of the four ECoG waveforms. The SP/AP amplitude ratio is also shown to the right of each response. Although these responses all vary to a greater or lesser extent from the ideal waveform shown in Figure 3, the identification of the SP and AP components is not too difficult.

The process of peak picking can be somewhat more tedious in abnormal waveforms, as illustrated in Figure 5. Note that in these two cases of patients with confirmed hydrops, the SP component is seen as a slight shoulder or plateau riding on the AP component. Particularly in a noisy response as might result from myogenic contamination, the identification of the SP may be very difficult. We recommend that in such cases, several replications of the ECoG be obtained under identical stimulus and recording conditions.

Figure 6 illustrates the ECoG responses and pure-tone audiogram of a patient with unilateral Ménière's disease (left).[9] The right response is within normal limits for all parameters, whereas the left tracings are characterized by an abnormally enlarged SP component and SP/AP amplitude ratio.

The incidence of abnormal SP enhancement in the Ménière's population has generally been estimated to be between 60% to 65%[5-7,14] However, whether or not an abnormal SP is observed may depend on the symptoms (i.e., the inner-ear pressure) the patient has at the time of testing. A recent study by Ferraro et al. has shown that there was a significant difference between ECoG findings when symptoms of inner-ear dysfunction were absent and those obtained when one or more symptoms were present.[9] Furthermore, the presence of hearing loss combined with aural fullness or pressure was found to be the strongest predictor of an abnormal ECoG (i.e., an enlarged SP/AP amplitude ratio). This study recommends that perhaps the best application of ECoG in the Ménière's population is to perform the exam during an attack or when the patient is symptomatic, and to repeat the exam for comparative purposes when the symptoms subside.

CONCLUSIONS

We have found electrocochleography to be a valuable component of the test battery administered to patients suspected of having Ménière's disease or endolymphatic hydrops. The ECoG response seems to provide an "objective" indication of hydrops, and this information, in turn, can be used to help substantiate a diagnosis, justify treatment, and monitor the effects of that treatment. It must be recognized, however, that ECoG results are influenced by the clinical symptoms

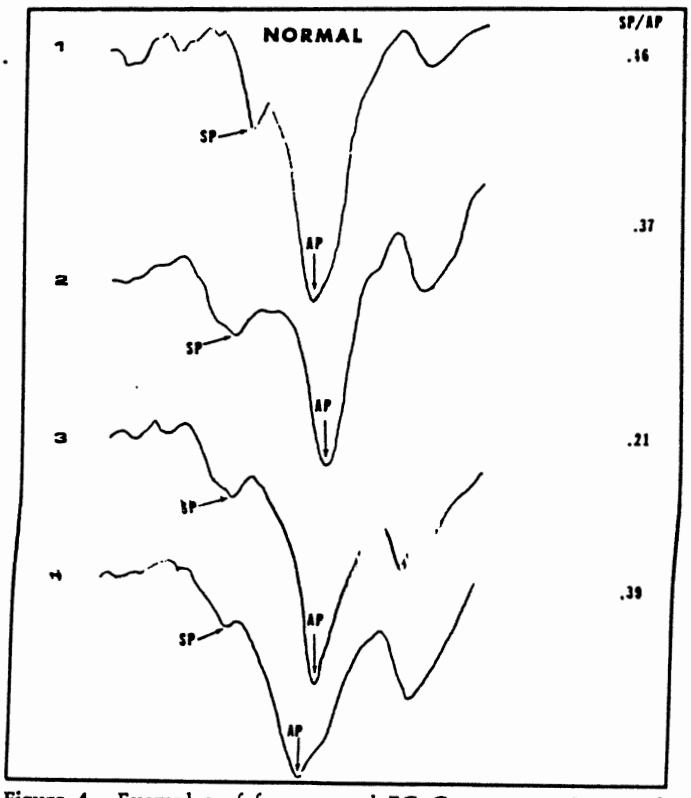

Figure 4. Examples of four normal ECoG responses. Points of measurement of SP and AP are indicated by arrow on each waveform. The SP/AP amplitude ratio is shown to the right of each response.

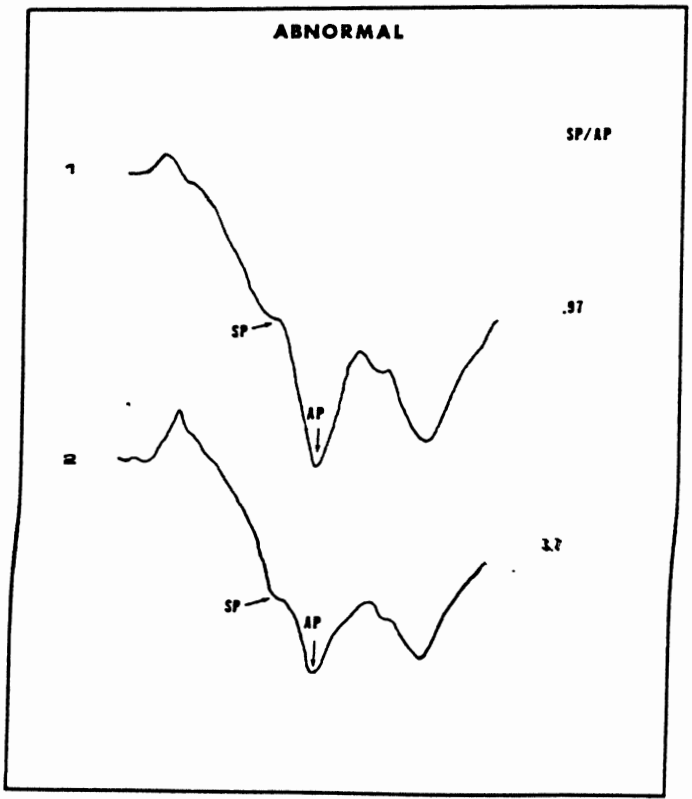

Figure 5. Examples of two abnormal ECoG responses. Points of measurement of SP and AP are indicated by arrow on each waveform. The SP/AP amplitude ratio is shown to the right of each response.

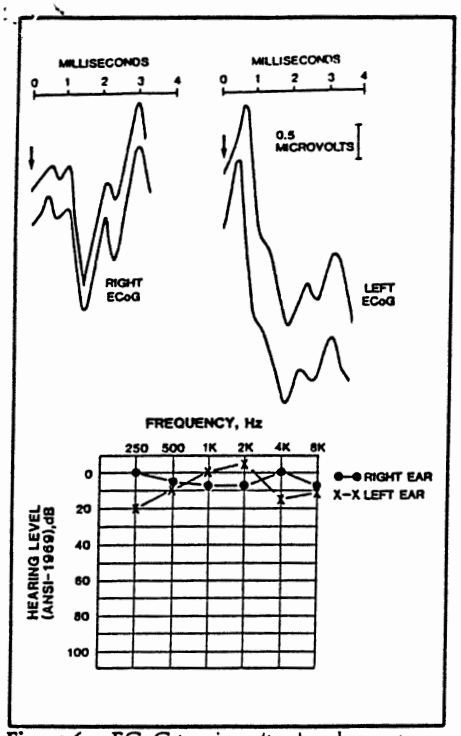

Figure 6. ECoG tracings (top) and pure-tone audiogram (bottom) from a patient with unilateral (left) Ménière's disease. Right ECoG response is normal, left response displays abnormally enlarged SP and SP/AP amplitude ratio. (From Ferraro et al., 1985, page 72).

of inner-ear dysfunction (this may be true for the other tests for hydrops as well), and this should be taken into account when the exam is administered.

There also are other limitations to the application of ECoG. Due to the expanded bandpass of the preamplifier filter, there is more artifactual "noise" to contend with, and impedance-matching difficulties are encountered more frequently with ECoG than with, for example, the ABR. It is also our experience that severe-to-profound, high-frequency (2000 Hz and above) hearing loss precludes the recording of reliable responses using extratympanic techniques. The threshold of the AP-N1 in normal listeners may range between 30 dB and 50 dB across subjects and, of course, is elevated in the hearing impaired. This implies that extratympanically recorded ECoG is not the technique of choice when some indication of hearing sensitivity is needed. Wave V of the ABR, for example, persists longer than the ECoG AP, which corresponds to Wave I.

Finally, it has also been our experience that ECoG should be used in conjunction with other tests such as the ABR to provide the most complete and accurate diagnostic information. This statement is based on a small percentage of patients we have seen who had been diagnosed, without the ABR, as having Ménière's disease and whose ECoG tracings supported this. ABR, when measured, was also abnormal, and these individuals were subsequently found to have acoustic tumors.

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