EEG Artifacts in the Intensive Care Unit Setting

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ABSTRACT. Obtaining a quality EEG in the intensive care unit (ICU) is a very rewarding experience for the EEG technologist. “Quality” is defined as a measure of excellence or state of being free from defects. It takes more than knowing how to obtain a quality record; it takes hands-on experience and time. Electroencephalography is a valuable neurodiagnostic tool in critically ill patients. However, the ICU is a challenging environment to obtain a high quality EEG tracing because artifacts are exceedingly common. Dealing with artifact effectively is an essential function of the EEG technologist. The goal of this paper is to review both physiological and nonphysiological artifacts commonly encountered in an ICU setting. How to recognize, troubleshoot, and prove that an EEG pattern is an artifact will be reviewed for the novice EEG technologist.

KEY WORDS. Artifacts, EEG technologist, electroencephalography, intensive care unit (ICU), nonphysiological artifact, physiological artifact.

INTRODUCTION

In electroencephalography, an artifact is defined as any recorded electrical potential which does not originate in the brain. There are two basic types of artifact, physiological and nonphysiological. Physiological artifacts are generated from electrical activity related to normal functioning of the patient’s body. Common physiological artifacts include those generated by muscle, body movement, the heart, the tongue,
the eyes, and sweat glands. Nonphysiological artifacts are generated from electromagnetic fields outside the body. Common nonphysiological artifacts include: 60 Hertz (Hz) artifact, electrode artifact, artifact generated from medical instruments (for example, ventilators and hemoperfusion devices), and movement in the environment.

EEG monitoring in the intensive care unit (ICU) is a valuable means of monitoring brain function in critically ill patients who may be comatose, sedated, or paralyzed. The ICU presents the perfect setting for the occurrence of artifact because of its broad array of electrical equipment and hectic medical environment. In the ICU, technical EEG problems arise that are beyond those experienced in the standard EEG laboratory. There is potential for physiological artifacts as well as nonphysiological artifacts, at times making EEG interpretation difficult, if not impossible. What may seem initially to be cerebral activity, with further investigation proves to be artifact. Conversely, what may seem at first to be an obvious artifact may prove to be cerebral activity. It is essential for a technologist to: a) recognize artifact appearing in the EEG, b) have the knowledge of the many different causes of artifact, and c) know what steps to take to prove, beyond a doubt, that the EEG abnormality is an artifact. The EEG technologist is the right hand of the electroencephalographer and must be able to answer the question “Is it cerebral or is it artifact?” before it is asked. The EEG recording should never end before that question is answered.

It is the goal of this review to help the EEG technologist recognize artifacts that are commonly encountered in the ICU. This paper will review the causes of artifacts, their appearance on the EEG, and how to logically and systematically evaluate a pattern that may be an artifact.

PHYSIOLOGICAL ARTIFACT

EMG and Involuntary Body Movement Artifacts

Electromyographic (EMG) activity is electrical activity generated in muscles. EMG artifact on EEG most frequently occurs from the contraction of various muscles in the scalp, face, jaw, and neck and is most often seen when the patient is awake. EMG artifact can obscure the recording of electrical brain activity and can be seen intermittently or continuously throughout the test in one or more channels on the EEG. EMG artifact often occurs when the patient is tense or anxious and has difficulty relaxing or holding still. In the ICU this type of artifact is commonly caused by the patient biting down on the endotracheal tube which connects the patient to the ventilator. EMG artifact can simulate fast mid-voltage irregular single or serial spikes (Figure 1). This activity can simulate cerebral beta activity with the use of the high frequency filter, especially when used in combination with the 60 Hz notch filter.
Attempts to eliminate muscle artifact are often futile, especially when the patient is not able to follow commands. The technologist should make sure the patient is not cold and that his/her head is resting comfortably. Try to interact as little as possible with the patient. In some cases relaxation will come eventually, so be patient and record as long as needed to get an interpretable recording. Occasionally sleep activity might be all you can successfully record that is free of muscle artifact.

Involuntary body movement artifacts in the ICU are often related to body twitching, tremor, tics, and tonic-clonic activity. Body movements produced by the face, mouth/tongue, extremities, or trunk can cause an electrode or electrode wires to move. Such movements might occur during focal or generalized seizures or may be independent of seizure activity. Involuntary body movement artifact can be recorded in one or more channels during EEG monitoring. Rhythmic movements, like a tremor not associated with seizure activity, can cause rhythmic artifact on the EEG recording that can resemble focal seizure activity. Monitoring involuntary movements of the face, arm, or leg with two electrodes referred to each other can be correlated simultaneously with the artifact on the EEG recording.

If the patient is intubated and unresponsive, drug induced muscular paralysis might be an option. Any muscle artifact, EMG or movement, can be eliminated with neuromuscular-blocking drugs (for example vecuronium bromide, trade name Norcuron®) making it possible to evaluate brain activity more accurately (Figures 2 and 3).
Muscle paralyzing drugs have no known effect on consciousness and therefore do not alter brain function like sedatives. Since muscles of respiration are affected, only patients on mechanical ventilation are candidates for such an approach. Maximum neuromuscular block occurs in about three to five minutes but gradual disappearance of muscle and movement artifact on the EEG may be noted approximately 30 seconds following injection with a neuromuscular-blocking drug. Some ICU providers will administer an anxiolytic agent along with a neuromuscular agent to prevent potential patient distress related to generalized muscle paralysis.

With the advent of portable video cameras, digital video monitoring in the ICU is a practical and efficient way of capturing clinical symptoms prior to paralysis that may be later reviewed by the electroencephalographer. To attenuate muscle activity instrumentally, when all else fails, lowering the high frequency filter may prove useful.

**Cardiac Artifacts**

Electrocardiogram (EKG) artifact is a common physiological artifact produced by the electrical activity of the heart. The presence of EKG artifact on an EEG tracing is related to the recording of the electrical field of the heart potential over the surface
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of the scalp. EKG artifact typically has a spike or sharp morphology and can be confused with epileptiform activity to the untrained eye.

The heart can be viewed as a dipole at any instant during cardiac depolarization because some myocardial cells in certain regions are negatively charged, while those in the other regions are positively charged. These polarities reflect the fact that the outside of a depolarized cell is negatively charged, whereas the outside of a resting cell is positively charged. Referential ear electrodes, A1 and A2, are often in the field of the cardiac dipole. On a referential montage with A1 or A2 in input 2 of each channel, EKG artifact deflections are initially upgoing in channels in which electrodes are referenced to A1 which is positively charged from the cardiac depolarization, while downgoing in channels referenced to A2 which is negatively charged from the cardiac depolarization (Figure 4).

There is no way to completely eliminate EKG artifact during the EEG recording. In some instances turning the patient’s head will change the EKG field over the head and lower the amplitude of the artifact. Routinely recording EKG simultaneously during the EEG is the best solution for this and other cardiac-related physiological artifacts. EKG rhythm can be recorded by placing two EEG electrodes on any noncephalic part of the body. Refer the EKG leads to each other (input 1 to input 2) to monitor the EKG activity. The further apart the electrodes are placed on the body

FIG. 3. Following administration of a neuromuscular blocking agent, all muscle activity stops and a cerebral burst suppression pattern with spike and polyspike and wave discharges is evident. (Same patient as Figure 2).
the greater the EKG amplitude. This is a simple and accurate way of differentiating EKG artifact from sharper activity of cerebral origin. In addition, the use of a bipolar montage rather than a referential (A1/A2) montage usually reduces the voltage of the EKG artifact.

**Pacemaker**

A pacemaker is a small device, about the size of a wristwatch, that helps to regulate heart rate. A pacemaker is made up of leads (thin flexible wires) and a generator (battery). The pacemaker is implanted under the skin just below the collarbone and can be easily visualized. Cardiac pacemaker artifact is caused by the electrical stimulus delivered to the heart by the internal pacemaker. This artifact can be seen on a regular or intermittent basis depending upon the type of cardiac pacemaker implanted in the patient. Cardiac pacemaker artifact has a repetitive spike-like morphology and may be misinterpreted as a polyphasic interictal epileptiform discharge.

When applying leads to record EKG make a conscious effort not to place the electrode on the pacemaker itself since the pacemaker battery generates a high frequency, large amplitude pulse and will contaminate the EEG recording. Placing

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**FIG. 4.** A1 and A2 electrodes are often in the field of the cardiac pole. Deflections are up going in channels in which electrodes are referenced to A1. Deflections are down going in channels in which electrodes are referenced to A2.
the electrode away from the pacemaker will not eliminate the artifact but rather keep artifact to a minimum.

**Cardioballistogram**

A cardioballistogram artifact appears as widespread rhythmic delta activity on scalp EEG and may be misinterpreted as generalized slowing of cerebral activity. Again, a channel devoted to EKG is best to monitor these artifactual slow waves that are time locked to cardiac depolarization. The waves occur at the same frequency as the EKG, but are slightly delayed, beginning just after the QRS (the highest amplitude and sharpest component of the EKG rhythm) complex. Cardioballistogram artifact on EEG is typically an issue when there is marked suppression of background EEG activity or electrocerebral silence (ECS).

**Cardiac Arrhythmia**

A cardiac arrhythmia artifact is a physiological artifact caused by any variation from the normal rhythm of the heart beat. EKG artifact associated with cardiac arrhythmia may be misinterpreted as epileptiform abnormalities or focal slowing (Figure 5). Every precaution must be taken to avoid such errors of interpretation. As with the other physiological artifacts associated with cardiac activity, the simplest and most reliable way to avoid misinterpretation is to record EKG simultaneously during the EEG.

**Pulse**

Pulse artifact can occur when an electrode is placed over a pulsating artery or tissue. The pulse artifact can simulate focal slowing of cerebral origin. Both the frequency and form of the waves can help identify this artifact. Like the other cardiac related artifacts, a direct relationship exists between the EKG and the pulse waves. The rhythmic slowing caused by the pulse wave does not occur simultaneously with the QRS complex of the EKG, but is typically delayed by about 200 to 300 msec after the QRS complex. A pulse artifact is easily identified by touching the electrode producing it. In this way, the technologist can confirm the movement of the electrode while simultaneously altering the appearance of the artifact. In cases when the pulse artifact originates from only one electrode, the artifact can be eliminated or reduced by moving the electrode slightly away from the pulsating tissue. Repositioning the patient’s head may also prove to be helpful.

**Glossokinetic Potential Artifact**

Glossokinetic potential (GKP) artifact occurs with tongue movement because the tip of the tongue is electrically charged and is relatively more negative than the
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base of the tongue. Movement of the tongue dipole will change the electrical field around the mouth and jaw. At times the field of GKP artifact can spread to the upper part of the face. The artifact produced has a broad potential field that drops in amplitude from frontal to occipital head regions. The amplitude of the potential is greatest near the tongue and is detected best in the leads located near the mouth including lip leads, infraorbital leads, and Fp1/Fp2.

GKP artifact appears as burst of generalized slow wave activity on scalp EEG which may be misinterpreted as being cerebral in nature. With a cooperative patient, this physiological artifact can be reliably reproduced by asking the patient to repeat words that cause significant movement of the tongue such as “lalala”, “Tom Thumb”, or “lilt” and stopped by asking the patient not to talk or move the tongue (Figure 6). Chewing, sucking, sobbing, and hiccups, frequently seen in infants and young children, can cause GKP artifact that resembles spike-wave or other transient discharges.

In an uncooperative patient or in the ICU setting, it is often impossible to eliminate tongue movement and monitoring for GKP is imperative. An EEG channel devoted to monitoring tongue movement with electrodes placed above and below the patient’s lips will correlate with the artifact. Simultaneously, the technologist should note

FIG. 5. Arrhythmic artifact due to an arrhythmic heart rate is recorded on the EEG and is time locked to the QRS complex in the EKG channel. Independent bihemispheric epileptiform discharges, maximal over the paracentral head regions, are also present (see boxes).
whenever the clinical symptoms occur by indicating on the EEG that the patient is moving his tongue.

**Electroretinogram Artifact**

An electroretinogram (ERG) artifact is recorded during photic stimulation. It is the result of the response of the eyes’ retinal cells to the light. ERG artifact is maximal in frontopolar electrodes (Fp1 and Fp2), since they are closest to the eyeballs. ERG is most evident during ECS recordings since cerebral activity is suppressed and high recording sensitivities are used, but may be recorded in normal individuals at standard sensitivities. An ERG artifact can be misconstrued as cerebral activity and potentially epileptiform discharges. ERG can be noted at all or some flash frequencies, is usually bilateral, and is time locked to the photic stimulus (Figure 7).

This physiological artifact will disappear when the light source to the eye is blocked by covering the patient’s eye during photic stimulation (Figure 8). Both left and right eyes should be tested separately by covering the eyeball with an opaque material and simultaneously documenting the maneuver on the EEG tracing.

![FIG. 6. Glossokinetic potential (GKP) artifact resembling a burst of generalized slow waves and rhythmic polyspike activity is present when the patient speaks and correlates with tongue movement labeled in the lip channel.](image-url)
FIG. 7. Electroretinogram (ERG) artifact, maximal in channels with Fp1 and Fp2 electrodes, is time locked to photic stimulation in an EEG with marked background suppression.

FIG. 8. Electroretinogram (ERG) artifact is eliminated in channels with the Fp1 electrode by covering the left eye and returns when the left eye is uncovered.
Be careful to only cover the eye; **not** the Fp1 or Fp2 electrodes. If the artifact does not disappear when the eye is covered, consider covering Fp1 and Fp2 electrodes to detect a photo cell artifact (see later section “Electrode Artifacts”).

**Sweat Artifact**

In the ICU, sweat artifact is most often recorded in patients with high body temperature associated with fever. Perspiration causes slow shifts of the electrical baseline by changing the impedance between the electrode and the skin. In addition the active sweat gland itself produces slowly changing electrical potentials that are recorded by the EEG electrodes. Sweat artifact almost always appears in more than one channel, but can be lateralized or asymmetric. Sweating causes long duration slow wave artifact that is smooth in outline and of high amplitude particularly in the temporal and frontal electrodes (Figure 9), and may be misinterpreted as slowing associated with cerebral dysfunction.

Reduction of sweat artifact can be produced by cooling the patient. This can be achieved by drying the scalp with a clean dry cloth, reapplying the electrodes, and placing a cold damp cloth over the patient’s forehead and front of neck or wiping the patient’s face. It may be necessary to repeat this process as often as necessary to keep artifact to a minimum. Keep the room as cool as possible or, if available, use a cooling blanket on the patient.

**FIG. 9.** Long duration slow wave artifact due to sweat in a febrile critically ill patient.
NONPHYSIOLOGICAL ARTIFACT

Alternating Current (60 Hz Artifact)

The most common nonphysiological artifact is 60 Hz artifact. 60 Hz interference is caused by an alternating current which supplies power to electrical wall outlets. The waveform of an alternating current power circuit is a sine wave. The number of times an alternating current repeats a full cycle in one second is the frequency of the current and the maximum voltage of the current is its amplitude. 60 Hz artifact reverses directions cyclically — changing from positive to negative and back again — 60 times a second.

In the ICU, 60 Hz artifact comes from the electrical equipment in the patient’s room (Figure 10) including such devices as: the electrically powered bed, the mechanical ventilator, intravenous infusion devices, Sequential Compression Devices (SCD) or Flowtron® Excel machines, EKG monitors, dialysis machines, fluorescent lights, and heating/cooling lamps or blankets. 60 Hz artifact can sometimes be traced back to the EEG jackbox cable if it is in contact with the floor or any other power cables (including the power cable to the EEG instrument).

Poor electrode contact associated with inadequate skin preparation, defective cables, lead wires, or faulty grounding can also cause 60 Hz artifact. There is a strong likelihood of electrode instability and high impedances with prolonged recordings in the ICU. Because of high or mismatched impedances there is a failure of common
mode rejection by the amplifiers. In this situation, exogenous artifacts (i.e., 60 Hz) can be amplified and detected on the screen in one or more channels. 60 Hz artifact produces a “fuzzy” appearing baseline and can sometimes be confused with muscle artifact or fast cerebral activity. This artifact can be better identified by increasing the display (paper) speed to 60 mm/sec (Figure 11).

If electrode impedances are low (5K ohms or less) and 60 Hz artifact is still present, begin to unplug individual electrical equipment from the outlet, one plug at a time, and make a note of it on the EEG. Continue to unplug the equipment until the source of the 60 Hz is identified and the artifact is eliminated. It is always best to get permission from someone on the ICU staff before unplugging equipment associated with patient care. **Never unplug the mechanical ventilator under any circumstance.**

60 Hz can sometimes be eliminated simply by not letting the jackbox cable touch the floor. Use only as much cable as necessary to reach from the EEG instrument to the jackbox. Occasionally 60 Hz can easily be eliminated by keeping cables from touching each other. If all else fails and 60 Hz artifact persists even after making every effort to identify and remove the source of the 60 Hz, the 60 Hz notch filter can be used as a last option. The notch filter attenuates activity at 60 Hz and attenuates adjacent frequencies less extensively. It is important to remember that when the notch filter is used other faster frequencies, including cerebral activity, within the range

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**FIG. 11.** By increasing the display (paper) speed to 60 mm/sec, five seconds of EEG readily identifies 60 Hz artifact most prominent in channels with Fp1 and Fp2 electrodes.
of the filter is also attenuated. Hence, an epileptiform spike may have its displayed amplitude lowered because some components of the spike have frequency characteristics at or near 60 Hz.

**Electrode Artifacts (From Surface Electrodes)**

EEG surface electrodes are metal discs secured directly to the scalp with a conductive paste. A wire is attached to each electrode connecting the electrodes to the inputs of the EEG amplifiers. Any interruption in this path, from the scalp to the jackbox, can produce an EEG electrode artifact. Electrode artifacts can have different appearances depending on the site and type of the disruption, but are always confined to the channels that have the electrode in either input 1 or input 2.

Poor electrode contact can cause instability of impedance which leads to sharp or slow waves of varying morphology and amplitude that have the potential to be confused with focal cerebral slowing or even rhythmic activity resembling the beginning of a focal seizure. When there is an abrupt change in the electrode impedance, a sudden potential appears causing an electrode “pop.” An electrode “pop” has a characteristic morphology of a very steep rise and shallow fall resembling a direct current (DC) calibration signal (Figure 12). An electrode “pop” can simulate a focal spike or sharp wave on the EEG.

FIG. 12. An F8 electrode pop may be misinterpreted as a cerebral spike and slow wave discharge. An F3 electrode artifact looks like a sharp wave resembling a calibration pulse.
Electrode artifact associated with lead wire movement has a more disorganized morphology and can be confused with generalized slowing if all electrodes are affected or focal slowing if only certain electrodes are involved. Lead movement electrode artifact occurs at the frequency of the motion of the wires.

Electrode artifact is primarily caused by improper contact of the electrode on the skin, a broken electrode wire, lead movement, or a loose electrode wire within the jackbox. Electrode artifact is likely to occur if the skin is not well prepped, if not enough conductive paste is used, or if the paste dissipates. A break in the electrode wire can occur from normal daily usage or during the recording if it is pulled or gets caught. The electrode wire can be loose in the jackbox if it is not plugged in all the way or if the wire becomes displaced.

The job of the technologist is to learn to differentiate between cerebral activity and electrode artifact. If there is questionable activity on the EEG that seems to be seen in one channel or channels with one common electrode, corrective measures must be taken. Reprep the area and reapply the same electrode with electrolyte paste. If reapplying the electrode doesn’t correct the artifact, replace with another electrode you know is good or try a new one. Finally, make sure the electrode is properly plugged into the jackbox.

**Photo Cell Artifact**

During photic stimulation high impedance in electrodes over the frontal regions may cause an unusual artifact. Each flash causes a minute photochemical reaction which, in the presence of high impedance can cause the electrode to act as a photocell. Thus in the involved channels, a brief spike-like transient appears simultaneously with the flash.

A photo cell artifact is a time locked change in the potential of EEG in a single electrode during photic stimulation. It is easily identified as it disappears if the light is blocked from the electrode in question. When proving this is a photocell artifact, be careful to cover only the electrode not the eyeball.

**Mechanical Ventilator Artifact**

Mechanical ventilation is a method to mechanically assist or replace spontaneous breathing. Mechanical ventilation artifact is caused by switching magnetic fields within the ventilator motor and by the movement of the electrodes or leads as the body is moved by the device. The artifact occurs with the motor’s activity, thus it may be constant or intermittent depending on how the ventilator is programmed.

Mechanical devices such as ventilators usually produce artifacts with slower components than other ICU electrical devices. The mechanical ventilator can cause bursts
of rhythmic high amplitude slow waves maximal over the frontal leads bilaterally. The artifact can simulate a burst suppression pattern of cerebral origin, especially if the background EEG activity is suppressed. Ventilator artifact on the EEG can be directly correlated with mechanical breaths per minute ventilator settings or with the patient’s observed respirations.

Occasionally, just repositioning the patient’s head will eliminate or reduce the artifact. If the artifact cannot be eliminated it should be monitored by placing one electrode above and one electrode below the lips or by carefully attaching two electrodes to the ventilator tubing or dial. These electrodes can be referred to each other in a separate channel on the EEG. As a last option, the ventilator can briefly be disconnected from the endotracheal tube by the respiratory staff to determine if the activity is related to the ventilator.

Movement Artifact in the Environment

Movement of persons in the vicinity of the patient generates artifacts usually of a capacitive (the ability of a body to hold and electrical charge) or electrostatic origin (a sudden and momentary electric current that flows between two objects at different electrical potentials caused by direct contact or an electrostatic field). It is likely to occur as a result of movement of the patient by the nurse or doctor or movement of other personnel or family members near the patient’s bed including the EEG technologist or the electroencephalographer. The morphology of the artifact on the EEG varies with the motion or activity of the person in close proximity of the patient.

It is important for the technologist to be able to recognize the correlation between what is going on in the room and what is being recorded on the EEG. It is helpful if the technologist asks the person in the room to repeat the activity or motion to reproduce the artifact on the EEG and simultaneously notes it on the recording. To eliminate this type of artifact, it is best to prevent people from moving in the room, often a daunting task in the ICU.

Prisma® Hemoperfusion Device Artifact

The Prisma® continuous renal replacement system is a type of hemodialysis that removes, filters, and returns the blood slowly and continuously in patients with kidney failure. The Prisma® device has rotary pumps which produce a unique EEG artifact during dialysis. The Prisma® device induces generalized saw-toothed waveforms in the range of 5.5 to 11 Hz that correlate with the movement of the rotor within the device. The unique artifact is characteristic, but could be easily misinterpreted as an abnormal cerebral rhythm if one is not aware of it. Prisma® EEG artifact
resembles rhythmic temporal theta burst of drowsiness (RTTD), a benign EEG variant (previously called psychomotor variant) in frequency and morphology. Unlike RTTD, Prisma® artifact is located more posteriorly. It can also be misinterpreted as an epileptiform discharge. The artifact should be suspected when invariant saw-toothed rhythms appear beyond the temporal and occipital lobes and contaminate the electrocardiographic channel.

When the Prism® device is paused (rotors stopped), but not switched off, the rhythmic waveforms disappear. When the Prism® is reactivated the waveforms reappear. This artifact can be verified by its disappearance on stopping and reemergence with starting of the rotary pump action of the device (of course, this should be done in conjunction with dialysis staff).

CONCLUSION

EEG monitoring is a valuable way of acquiring additional information about brain function in critically ill patients. However, in the hostile environment of the ICU obtaining quality records can be challenging. This paper was written to educate the new or inexperienced technologist. All questionable EEG patterns or activity must be investigated. It is the responsibility of the technologist to know how to identify and differentiate artifact from cerebral activity before disconnecting the electrodes from the patient. The electroencephalographer should also be prepared to assist the technologist in finding a solution for more difficult challenges at the time of the recording. Every effort should be made for an accurate interpretation to avoid further unnecessary tests, administration of inappropriate drugs, or prolonged hospitalization.

REFERENCES


Neurocysticercosis: Fireflies in the Central Nervous System

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ABSTRACT. Neurocysticercosis (NCC) is caused by cysticerci spreading to spinal or brain tissue. NCC causes headaches, seizures, and focal neurological deficits. NCC is one of the main causes of epilepsy worldwide. NCC can cause death if it is not adequately treated. NCC is preventable through education of proper handwashing techniques proper food handling, and separation of human and pig living quarters (CDC 1998). The International League Against Epilepsy (ILAE), the World Health Organization (WHO), and other international health organizations are attempting to rid the world of NCC through education.

Cysticerci create cavities in the brain and other body tissue where their tiny bodies grow sometimes into tapeworms two to seven meters in length and can live up to 25 years in the human body. On magnetic resonance imaging (MRI) wormholes appear. Often times, if best practice is followed, the patient’s entire body is scanned and more craters appear. Some authors have stated that cysticerci on a computed topography (CT) scan are similar to a starry sky or fireflies (Kapur et al. 2007).

KEY WORDS. Cysticercosis, EEG, focal epilepsy, infectious diseases, magnetic resonance imaging (MRI), neurocysticercosis (NCC), pork tapeworm, Taenia solium.

TAENIA SOLIUM

Taenia solium (Figures 1 and 2) is responsible for causing the crater like appearance on the magnetic resonance images (MRIs) of those affected by cysticercosis (Figure 3). An intermediate host, typically a pig or a human, unknowingly consumes...
Taenia solium larva. The larva attaches to the intestinal wall and releases eggs. These eggs are released via the stool of the animal and either come into direct or indirect contact with food for humans (Figure 4). This can happen through improper disposal of waste, improper handwashing, or improper cooking. When Taenia solium infects either a pig or a person the infection is known as cysticercosis. Taenia solium

FIG. 1. Taenia solium scolex form with suckers and hooks. From Hubert Ludwig, Schul-Naturgeschichte 1891 and in the public domain.

FIG. 2. Taenia solium, a parasitic cestode worm with suckers and scolex showing. From Centers for Disease Control and in the public domain.
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rapidly multiplies within the organism causing multiple cysts in muscle tissue which appear as bright spots on a computed tomography (CT) scan, often referred to as a starry sky.

NEUROCYSTICERCOSIS

Neurocysticercosis (NCC) occurs when the cysticerci from the pork tapeworm, Taenia solium, infect the central nervous system. Some people may remain asymptomatic their entire lives as carriers, while others are asymptomatic for a few years then suddenly become very ill with seizures, headaches, and other focal neurologic deficits as the cysticerci multiply within the nervous system, muscles, and other tissue. NCC can cause sudden death due to high amounts of pressure on the brain and spinal cord.
Clinical diagnosis can be challenging for many reasons. First, initial presentations of this disease are often vague complaints of headaches, focal weakness, diplopia, focal seizures, dizziness, vomiting, or hypertension which mimic a multitude of other neurological diseases (Suarez and Iannucci 1999). Secondly, the group of patients most likely to acquire neurocysticercosis is also those who are either living in endemic regions lacking or devoid of adequate medical care or immigrants to countries where services may be difficult to receive based on their income and comfort level. The United States is seeing increasing numbers of imported cases of NCC especially in border regions due to increasing numbers of immigrants from endemic regions (Yeh and Sheffield 2008).

Finally, NCC can be difficult to diagnose because the tests necessary to diagnose NCC are not always readily available, especially in endemic regions or to those with little or no health care. Histologic confirmation, direct observation of the parasite, or evidence of cysts with the scolex on CT or magnetic resonance imaging (MRI) are the absolute criteria for diagnostic certainty of NCC per Del Brutto et al. (2001).
In many endemic regions patients who do not have access to running water or proper disposal of fecal matter, also do not have access to CT or MRI or complicated blood work ups.

Once it has been diagnosed, NCC can be very difficult to treat if many cysticerci are present in the brain and surrounding tissues. In some patients no treatment is necessary, but in other patients antibiotics are required. Patients’ medical conditions when they contract NCC can make treatment more difficult. Suarez and Iannucci (1999) warn that an estimated 1000 new cases of neurocysticercosis will be diagnosed every year in the United States and many of these patients will be pregnant. If treated early there is a good prognosis for mother and child, but it may be difficult.

**CLINICAL SIGNS AND SYMPTOMS**

Clinical signs and symptoms vary greatly because of the numerous locations of the cysts. While it may be very challenging for providers to arrive at a diagnosis, early diagnosis is important because epilepsy and other life long or life ending diseases result from untreated neurocysticercosis.

The Center for Disease Control and Prevention’s (CDC’s) website has a listing of the clinical features of NCC. On DPDx, diagnostic Laboratory Identification of Parasites of Public Health Concern, the clinical features of NCC are seizures, mental disturbances, focal neurologic deficits, signs of space occupying lesions, and headaches. The CDC also notes that extracerebral cysticercosis can cause lesions in other peripheral nerve and muscle tissue as well.

Per Sawhney et al. (1998), some uncommon features of NCC are mesencephalic syndrome, upward gaze palsy, bilateral ptosis, papillitis, hemorrhagic stroke, cervical radiculopathy, tonsillar herniation, arteriovenous malformation, transient ischemic attacks, compressive myelopathy, and unilateral dystonia. Kurl and Montella (1994) and Grondin et al. (2006) report cases of NCC camouflaged as eclampsia in pregnancy. Patients may or may not have a fever. They may express only one or more of the clinical signs listed above. The symptoms may begin individually and build over the course of many years. Often the symptoms do not become urgent until the cysts die. Upon the death of the cysts, the body reacts to the foreign substance and inflammation begins thus initiating the process of infection.

**EEG FEATURES**

The EEG in NCC is nonspecific to the disease. The EEG can show epileptiform discharges (Figure 5), periodic lateral epileptiform discharges, focal sharp waves or spikes, and/or slowing. The EEG may be normal in some patients. In a group of thirty pregnant Indian women with NCC, only 21 had an EEG and only six of those studies were abnormal (Pandian et al. 2007).
It is difficult to obtain EEGs on patients with NCC because many of them live in endemic areas which lack adequate medical care and advanced testing measures.

OTHER IMAGING FEATURES

As it has been described by many authors, MRI and CT scans of patients with NCC appear like a starry sky. Each star is a fluid filled cyst with the beginning of a living, growing tapeworm inside. The cysticerci illuminate the MRI and CT scan very brightly. They appear as a ring enhancing lesion.

MRI and CT scans can also show cysticerci life stages, enabling physicians to treat this disease or to know when not to treat the disease. In many circumstances, physicians simply let the calcified cysts remain and do no further treatment.

OTHER DIAGNOSTIC TESTS FOR NCC

NCC can be more definitively diagnosed with very specific lab tests. Garcia et al. (2003a) state that a Western blot test which uses enzyme linked immunoelectrotransfer blot assay with purified glycoprotein antigens from the cysts is 98% sensitive
and 100% specific. They also add that stool tests can be helpful as well, but they are not as sensitive or specific. Sotelo and Del Brutto (2002) state that CT and MRI are the “cornerstone tools” for diagnosis of NCC.

TREATMENT METHODS

Treatment must be individualized for each patient as it is based on viability, size, and location of the cysts (Figure 6), severity of host immune responses, varied clinical presentations, and presentation of complications such as pregnancy and hydrocephalus. Krauss (2001) reports that patients with inactive lesions generally require no treatment other than for their symptoms, i.e., anticonvulsants for seizures. Garcia et al. (2002), in the *Current Consensus Guidelines for Treatment of Neurocysticercosis*, state that most treatments fall into one of three categories: antiparasitic drugs, surgery, and symptomatic medication. The Cysticercosis Working Group in Peru is made up of scientists and physicians from all over the world. They suggest a very specific dosing schedule of the antiparasitic drugs, sometimes with steroids and concurrent treatment of seizures and other symptoms as necessary.

![Image](https://www.xray2000.co.uk)

**FIG. 6.** Multiple cysts seen in leg tissue. From Nick Oldnall (www.xray2000.co.uk) and in the public domain.
A. Clinton White, Jr. (2000) describes in detail the many different ways cysticercosis and neurocysticercosis are treated. He diligently explains why each method was used and for which stage of cyst development the method was applied. Patients with inactive neurocysticercosis are not treated with antiparasitic drugs, they are treated for their symptoms. Typically they are treated for seizures and hydrocephalus due to scarring of the brain tissue. Patients with active neurocysticercosis are often treated with antiparasitic drugs, surgery, antiepileptics, and sometimes with corticosteroids. The third class of patients he describes is those with extraparenchymal disease. These patients have the highest morbidity. According to White, ventriculoperitoneal shunting is the preferred method of treatment to reduce health risks and cost.

Garcia et al. (2002) reiterate that there is not one therapy recommended for all patients, as it is not one single disease but an infection which causes a multitude of disease processes, all of which must be treated for a favorable outcome.

PREVENTION METHODS

NCC is a preventable disease. Many groups such as ILAE, WHO, and The Cysticercosis Working Group in Peru are collaborating on how to spread education worldwide. Educating people in endemic areas on how to avoid NCC is the best treatment possible. Education in proper handwashing, waste disposal, and cooking techniques of meat can help people understand how to avoid this treatable disease.

Financial support for endemic regions is also needed. Endemic regions cannot afford the tools necessary for proper handwashing without help. They need supplies like running water, proper sewage facilities, and proper cooking tools, as well as free access to education regarding the medical conditions, such as seizures. Without financial support, no amount of education will help endemic regions overcome this preventable, treatable disease.

CONCLUSION

In summary, neurocysticercosis can be overcome. It will not be easy or inexpensive but how much more expensive is treating the estimated two million persons worldwide with the disease and the multitude of health problems NCC causes. It is one of the main causes of epilepsy worldwide. Epilepsy costs our healthcare system billions of dollars annually due to emergency room visits, prescription drug coverage, physician visits, long term video monitoring, and disability pay. As members of the electroneurodiagnostic community, participating in spreading the knowledge of NCC to fellow colleagues is one way we can help to educate others about NCC. We can also partner with ILAE, WHO, and the CDC to finance and spread
knowledge of this disease. Always keep in mind that the patient you see with odd or unusual neurological symptoms may be suffering from NCC. If there are stars on an image, either CT or MRI, in the brain or elsewhere in the body tissue, think NCC.

REFERENCES


Garg RK. Diagnostic criteria for neurocysticercosis: some modifications are needed for Indian patients. Neurol India 2004; 52(2):171–77.


Utilization of Paraspinal Muscles for Triggered EMG during Thoracic Pedicle Screw Placement

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ABSTRACT. A novel intraoperative neurophysiological technique for testing the integrity of the pedicle during screw fixation for spinal deformity surgery is presented. The thoracic paraspinal muscles at the appropriate level are used as the electromyogram (EMG) pick-up for direct current stimulation of the thoracic pedicle screw at that level. This technique is shown to give reliable and reproducible results. This technique is found to produce more reliable data than the methods most commonly used at this time.

KEY WORDS. Intraoperative neuromonitoring, paraspinal muscles, pedicle screw stimulation, thoracic pedicle screws, triggered electromyography (tEMG).

INTRODUCTION

The use of pedicle screw fixation in the thoracic spine has become a commonly used method for the operative correction of spinal deformities (Suk et al. 1995). The use of triggered electromyography (tEMG) for the testing of pedicle integrity and nerve root proximity during insertion of pedicle screws has become a commonly utilized modality for intraoperative neurophysiological monitoring (IONM) during posterior lumbar and thoracic instrumentation surgeries. The intercostal muscles are the most commonly used peripheral muscles for tEMG in the upper thoracic region.
(T2 to T5) and the abdominus rectus muscle in the mid to lower thoracic region (T6 to T12). However, these muscles present many variables, such as patient body habitus and inconsistent anatomy. Since the paraspinals are the first muscles innervated off the dorsal rami of the thoracic nerve roots, a hypothesis is made that they are an effective alternative in achieving the ability to evoke a true muscle response during thoracic screw stimulation. What follows is an evaluation and analysis of a technique that incorporates the use of paraspinal muscles as the EMG pick up for pedicle screw stimulation in the thoracic region.

METHODS

Nine consecutive patients were included in the present study. All of these patients had severe spinal deformities or instabilities requiring instrumented thoracic fusion and deformity correction. Each of these patients had multiple thoracic pedicle screws placed as spinal anchors for the instrumentation (Figure 1). Each screw was placed

FIG. 1. Intraoperative view of multiple thoracic pedicle screws in an idiopathic scoliosis surgery. Note the curvature of the spine. Picture provided, courtesy of Laurence E. Mermelstein, M.D.
in a standard fashion as described by Kim et al. (2004) with the aid of intraoperative fluoroscopy. Two sterile needle electrodes (positive and negative) were placed intraoperatively by the surgeon in the paraspinal muscles at the level and side where the surgeon inserts the thoracic pedicle screw. The paraspinal muscles used were immediately adjacent to the screws being stimulated so that the surgeon is assured that he is activating the correct muscle for that level (Figure 2). The screws are directly stimulated via a sterile intraoperative probe and EMG activity is recorded. The current (in milliamps [mA]) threshold for EMG activity is recorded. Any impedance less than 7 mA forced the surgeon to re-evaluate the screw position by either removing the screw and re-probing the pedicle hole or directly inspecting the pedicle integrity via laminotomy. All questionable screws were replaced or removed at the time of surgery. Intraoperative x-rays, postoperative x-rays, and postoperative computed tomography (CT) scans were analyzed for possible screw breach and proximity of screws to the exiting nerve roots and spinal canal (cord). All of the patients had normal neurological examinations pre-operatively and were without baseline neurologic histories.

FIG. 2. Close-up view of the electromyogram (EMG) stimulus probe touching the pedicle screw and the EMG “pick-up” probes in the paraspinal muscle immediately adjacent to the screw at the same level. Picture provided, courtesy of Laurence E. Mermelstein, M.D.
RESULTS

A total of 121 pedicle screws were tested in nine different patients (Table 1). A reliable muscle response was obtained at all levels tested from the paraspinal muscles (Figures 3A and 3B). The impedance to the response was found to be variable from patient to patient and from level to level. In review of the postoperative

Table 1. Summary of results from testing 121 pedicle screws in nine patients.

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Abd Rect – abdominus rectus; Ht – height; Wt – weight; BMI – body mass index; Avg – average; PS – paraspinal; A R – abdominus rectus; NR – no response
x-rays and the CT scan data, there were no significant breaches in this series. In patients with truncal obesity and/or pendulous abdomens, a higher threshold from the abdominus rectus muscle was seen in comparison to the paraspinal responses (Figures 4A and 4B). In thinner patients with better-defined abdominal musculature, the thresholds were found to be similar from both acquisition sites. It was also found

FIG. 3A. Paraspinal response at 8 mA at 16:08:25. Screenshots provided courtesy of Justin Silverstein, CNIM, R.NCS.T., MS. R – right; L – left; Vas – vastus lateralis; TA – tibialis anterior; Gas – gastrocnemius; Ilio – iliopsoas; MEP – motor evoked potential; Ab Rec – abdominus rectus.
FIG. 3B. Abdominus Rectus response at 11 mA at 16:08:29. Screenshots provided courtesy of Justin Silverstein, CNIM, R.NCS.T., MS. R – right; L – left; Vas – vastus lateralis; TA – tibialis anterior; Gas – gastrocnemius; Ilio – iliopsoas; MEP – motor evoked potential; Ab Rec – abdominus rectus.
FIG. 4A. Paraspinal response at 6 mA at 16:12:10. Screenshots provided courtesy of Justin Silverstein, CNIM, R.NCS.T., MS. R – right; L – left; Vas – vastus lateralis; TA – tibialis anterior; Gas – gastrocnemius; Ilio – iliopsoas; MEP – motor evoked potential; Ab Rec – abdominus rectus.
that in patients with a body mass index (BMI) greater than 24, paraspinal responses were obtained from each screw tested. However, recordings from the abdominus rectus muscle elicited no response (NR) from certain levels up to 60 mA from these patients.

None of the patients in this series encountered any post-operative neurologic deficits related to thoracic nerve root or spinal cord injury.

DISCUSSION

Historically, the “Gold Standard” for spinal cord monitoring in thoracic spinal deformity surgery was the Stagnara “wake-up test” (Zouridakis and Papanicolaou 2001). This test required the anesthesiologist to wake the patient up from general

anesthesia, after the spinal instrumentation had been placed and the deformity corrected, but before the closure of the wound, and ask the patient to move his or her lower extremities. This technique is quite difficult at best and by definition gives the surgeon only delayed information about spinal cord integrity and function. The significant false positive rate and risk of intraoperative awareness put the surgeon and anesthesiologist at increased medico-legal liability (Zouridakis and Papanicolaou 2001). Over the last 15 years, intraoperative neurophysiologic monitoring of somatosensory evoked potentials (SSEP) and more recently transcranial motor evoked potentials (TCMEP) has essentially replaced the “wake-up test” in most spinal deformity centers (MacDonald et al. 2003). These techniques have been shown to give very high rates of sensitivity and specificity for intraoperative spinal cord injuries during spinal deformity surgeries (MacDonald et al. 2003).

Most of these studies have been performed using multisegmental “hook and rod” instrumentation techniques, which are essentially second-generation instrumentation techniques after Harrington’s original hook and rod techniques. Pedicle screw instrumentation of the thoracic spine has blossomed due to surgeons increased familiarity with the technique and the development of more sophisticated instrumentation (Suk et al. 1995). The more rigid fixation afforded by transpedicular screw placement has been shown to increase the degree of deformity correction and eliminate the need for post-operative bracing (Liljenqvist et al. 2002).

Unfortunately, the placement of thoracic pedicle screws is more technically demanding (smaller pedicles, deformity of the spine), with increased risk to the neural elements (Shi et al. 2003). The initial accuracy in cadavers ranged from 45 to 85% for conventional insertion techniques (Cinotti et al. 1999). In their study, Belmont et al. (2001) placed 279 screws in the thoracic spine (T1 to T12) in their patients. The overall fully contained accuracy rate was 57%, with 120 screws penetrating either the medial (14% [range 1 to 4 mm]) or lateral (29%) cortex (Belmont et al. 2001). It is quite difficult and at times very misleading to use intraoperative x-rays and fluoroscopy to judge the accuracy of thoracic pedicle screw placement (Shi et al. 2003).

As inferred from the above studies, a better way to assess the intraoperative placement of thoracic pedicle screws is necessary. Triggered EMG response in lower extremity muscles to assess lumbar pedicle screw placement is a fairly well established modality (Toleikis et al. 2000). A similar monitoring technique for thoracic pedicle screws is not as universally accepted. This study introduces a novel technique to assess the accuracy of pedicle screw placement in the thoracic spine. The data from this study infers a 100% sensitivity for medially misplaced thoracic pedicle screws. All screws with a low impedance were either changed or removed. There were no screws with a “normal” impedance which were found to be in the canal on CT scan. The post-op CT scans did not show any medially placed pedicle screws.
Unfortunately, because there were no “true positives” seen on EMG verified by CT scan, this statistic cannot be proven. This technique does have a false positive rate of 6%–7 screws out of 120 demonstrated a low impedance (EMG activity at 6 mA or less) with a specificity of 94%. We believe this is an acceptable rate. In addition, there were many instances in patients with truncal obesity where the abdominus rectus muscle electrodes were completely unreliable as an EMG pick-up (Figures 5A and 5B). This technique shows great promise in generating useful data to assess for clinically significant pedicle screw misplacement in the thoracic spine.

CONCLUSION

Based on the current data, the use of paraspinal muscles for tEMG was shown to be an effective alternative to currently used methods of pedicle screw testing in the thoracic spine. The paraspinal muscles gave more reproducible data when compared to abdominus rectus muscles, especially in patients with truncal obesity. Because no pedicle breaches were encountered in this study, exact threshold values indicating pedicle breach are difficult to report; a 100% sensitivity rate and a 6% false positive rate can be inferred. Continued research into developing threshold values in order to be able to accurately evaluate the pedicle integrity is ongoing.

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A New Ground and Reference Technique for Invasive EEG Recordings

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ABSTRACT. Objective: To demonstrate a subdermal wire electrode technique for establishing a ground (GND) and reference (REF) during long-term EEG monitoring (LTM) with intracranial electrodes. Usually, a separate GND and REF are required and this GND&REF pair can be selected contacts in the invasive electrode arrays themselves, special invasive electrodes, or standard surface disc electrodes which require frequent maintenance. We investigated the use of a pair of chronic Subdermal Wire Electrodes (SWE) for use as GND&REF. Methods: A pair of SWEs as GND&REF was tested in nine patients undergoing invasive EEG monitoring. SWE impedances were monitored in two patients and compared to disc electrode impedances. Results: Without maintenance, SWE impedances remained low and stable during the entire recording period (up to 20 days), whereas disc electrodes showed rapid impedance increase after the first day. In all nine patients, the consistent and durable integrity of the GND&REF pair of SWE allowed for a good quality EEG recording. No local skin complications were observed. Conclusions: A pair of SWE electrodes provides a GND&REF system that is easy to place, maintain, and provides a high quality recording with long-term stability when coupled with referential based EEG recording system from invasive electrodes. Significance: A more efficient means of

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establishing a GND&REF pair during the monitoring of patients with invasive electrodes is described.

KEY WORDS. EEG, electrode, ground, invasive monitoring, long-term monitoring, reference, subdermal wire electrode.

INTRODUCTION

Most commercial long-term EEG monitoring (LTM) systems require a ground (GND) and a reference (REF) electrode for recording high quality signals from invasive electrodes. The invasive electrodes consist of depth, grid, or strip electrodes or a combination thereof depending on the clinical diagnostic problem being evaluated. The GND&REF are critical to the referential based recording method and the general quality of the entire EEG is directly tied to these GND&REF electrodes. Currently there are several techniques for establishing a GND&REF. One method is to use any of the invasive contacts as GND&REF. Another method is to place a special 2-contact depth or strip and use these contacts as GND&REF. A further option and one used up until now at the University Hospital of Geneva (HUG) was to apply standard disc electrodes to the scalp with collodion. The basic characteristic of these acute, “wet” disc electrodes is their persistent deterioration either due to drying out, or they become loose so that frequent physical monitoring coupled with maintenance consisting of jelling and adjustments is required to maintain a good quality EEG recording. This aspect is time consuming, inefficient, and leads to seizure activity being recorded with significant artifact caused by high GND&REF impedances.

Recently, a Subdermal Wire Electrode (SWE) (Ives 2005) with “ideal” silver-silver/chloride (Ag-Ag/Cl) properties (Tallgren et al. 2005) was developed and applied to EEG recording in patients in the ICU (Young et al. 2006). These SWE are sterile and designed for single use. They record with an impedance of less than 5 kOhms, but most importantly, they do not need adjustments and will record without any maintenance for days and weeks (Ives 2005, Young et al. 2006, Martz et al. 2009), in contrast to the disc electrodes. Therefore, a pair of SWEs is a suitable candidate as GND&REF during invasive LTM.

METHODS

The SWE (Ives EEG Solutions, Inc., Manotick, Ontario, Canada; part number: SWE) consists of a 5 cm length of a Teflon® coated, 0.25 mm in diameter, multi-stranded pure silver wire. Stripping off 3 mm of Teflon® insulation from the distal end and then chloriding the exposed silver creates the recording element/tip (Ives 2005). The chloriding process creates an “ideal” Ag-Ag/Cl EEG electrode (Tallgren
et al. 2005) which is essential for a high quality, low impedance GND&REF. During their assembly, the recording tip is inserted into the lumen of a 25 gauge × 16 mm hypodermic needle, as shown in Figure 1. The wire is established on the outside of the needle, which permits prior connection of the small female mating connector. The SWE is very similar in design as the original chronic sphenoidal (Ives and Gloor 1977, Ives and Gloor 1978). The hypodermic needle is used to carry the SWE into the subdermal space, the wire is released, folded back, and the needle immediately withdrawn and disposed of. This leaves the very flexible Ag-Ag/Cl recording element/tip in place to record the local biopotential (shown in the lower photo).
GROUND AND REFERENCE FOR INVASIVE EEG
during the surgery for the placement of the invasive electrodes. As an alternative, and in the procedure used at HUG, the SWEs can be placed once the patient comes back to the LTM unit and he/she is being connected to the LTM recording system. This venue also permits measurement of the head for accurate placement of other 10–20 System located SWEs, which could allow simultaneous surface and invasive EEG recordings. Subdermal scalp placement of just two SWEs in the fully awake, adult patient does not present too much discomfort, as they are very quick and easy to place. The insertion needle is relatively small, 25 gauge (0.50 mm diameter), and all sensation dissipates as soon as the hypodermic needle is withdrawn. There is no sensation of the wire itself as the SWE is only 0.25 mm in diameter and very flexible. A minimal amount of collodion was used to fixate the wire on the scalp to prevent the SWE from being pulled out because of head movements, seizures, or nursing manipulation of the head. The SWE’s interconnection, 1-meter extension wire, is terminated with a standard safety connector to permit connection into the EEG recording equipment. For impedance comparison, standard gold-coated, pure silver, disc electrodes (Grass Technologies, Astro-Med, Inc. RI, USA) were placed close to the C3/C4 sites.

This GND&REF system was used in nine consecutive adult patients who underwent intracranial EEG recording at our institution with intracerebral depth electrodes or subdural strip/grid electrodes. All patients were fully awake during application of SWE and all were treated with oral non-steroidal analgesics during LTM. If necessary, they also received intravenous cefazoline (1g two times/day) during and after implantation of the intracranial electrodes.

In two patients, the impedance was measured daily during the monitoring using the local EEG recording system (Ceegraph, Biologic®, Natus Medical Inc. San Carlos, CA, USA). Patient 6 had direct impedance measurement of GND&REF electrodes against one intracranial contact by plugging the SWE electrodes into alternative jackbox positions, normally occupied by intracranial electrodes, and one intracranial electrode was transitorily used as REF. The impedance measured is, therefore, the impedance of the circuit constituted by the SWE and the REF-intracranial electrode, which overestimates the SWE proper impedance, by 4 to 7 kOhms (the actual impedance of the intracranial electrode). Patient 2 had impedance measurement of additional SWE located at the C3/C4 positions, as well as disc electrodes placed at C3/C4 positions.

RESULTS

Following invasive electrode implantation, at least two SWEs were placed subdermally in nine fully awake patients in the LTM unit. The placement of the SWEs was very well tolerated except for one patient who complained about some moderate transitory pain during placement of the SWE. No consequent bleeding was noticed at implantation or removal of the SWE. We observed neither skin irritation nor
infection (under intravenous prophylactic antibiotherapy) throughout the entire study on these nine patients. In one patient, severe, rhythmic head movement during the course of a generalized tonic-clonic seizure disconnected the pin connector of the SWE and this was promptly reconnected.

Figure 2 shows a patient with midline GND&REF with additional standard gold-coated disc electrodes placed at the C3/C4 sites.

Figure 3A demonstrates the high quality of the EEG that was obtained using a pair of SWE as GND&REF. No maintenance of the SWE was necessary during the entire duration of all recordings, with a maximum of 20 days (mean 9.6 ± 4.9 days). After 24 hours, the non-sterile, standard disc electrodes placed at C3/C4 already showed a significant signal decline (Figure 3B). Even by refilling them with jell every day, the impedances of C3/C4 steadily increased, accounting for 50 Hz artifacts that were not seen with SWE.

Figure 4 shows that the impedance for four SWEs in two patients remained well under 20 kOhms (this impedance measurement also included the invasive electrode’s impedance as well). Over the entire seven day recording period the impedance of the two metal disc electrodes (ME) showed a marked increase after 24 to 48 hours, associated with significant artifacts disturbing the recording (as discussed above and shown in Figure 3B).
Clinical and electrophysiological data for the nine patients is summarized in Table 1.

DISCUSSION

In this technical report, we describe an alternate GND&REF electrode system for invasive monitoring which is based on a pair of Subdermal Wire Electrodes (SWE). As already reported in the case of EEG monitoring of comatose patients (Young et al. 2006), the SWE has superior recording characteristics compared to other scalp electrode systems. In these nine patients studied using the SWE as a GND&REF pair, no indication of increased impedance was noted during the continuous invasive EEG. Our measurements confirm those of Young et al. (2006) with excellent invasive EEG signals based on the maintenance-free SWEs used as GND&REF. In this application, low, steady impedance were maintained for up to 20 days. Serial impedance measures were obtained only in two patients (only one had additional scalp disc electrodes for comparison). These impedance measurements confirmed those of previous studies measuring consistently low impedance of the SWE (Young et al. 2006) compared to standard gold cup electrodes. In a more recent study of a patient whose

**FIG. 3** A) Partial display of the high quality EEG obtained using the two Subdermal Wire Electrodes (SWEs) as ground and reference (GND&REF). During the entire recording process, the SWE did not need any adjustment nor maintenance as there was no deterioration in EEG quality attributed to the GND or REF electrodes. B) Deterioration of the signal of C3/C4 disc electrodes after 48 hours, requiring frequent maintenance in the context of sterile bandaging. The other channels show persisting good recording quality, with two SWEs as REF&GND. High Pass = 1 Hz, Low Pass = 100 Hz. Scale bar in right lower corner applies to all EEG channels except the two lowest (scalp C3/C4) where the vertical bar is 20 µV instead of 200 µV.
EEG was continuously recorded with 10 SWEs for 60 days with daily monitoring of the impedance, it was found that the impedance remained steady at less than 2.5 kOhms (Martz et al. 2009). At the end of 60 days there were no signs of irritation or infection associated with any of the 10 SWE placements. The antibacterial properties of silver have been well known for decades and are certainly beneficial here.

In the situation where a GND and a REF are selected from the array of invasive electrode contacts, these electrodes are removed from the recording field. The advantage of choosing an intracranial contact is to obtain a complete recording system made of the same electrode material. However, the site available/selected might not be optimum for a GND or a REF because, as a guideline, REF should be placed as far away from the recording electrode as possible. The same problem potentially arises when using a special 2-contact invasive electrode for GND&REF. Thus external, surface, non-invasive electrodes may be a reasonable choice, despite a basic guideline of using electrodes with similar materials in recording systems.

Similar to the SWE, a pair of sphenoidal electrodes (Ives and Gloor 1978) which have been commercially available since the late 1970s could also be used as a
Table 1. Clinical and electrophysiological data of the nine patients recorded with Subdermal Wire Electrodes (SWEs) used for ground and reference (GND&REF).

<table>
<thead>
<tr>
<th>Patient, gender, age (years)</th>
<th>Epileptic Syndrome</th>
<th>Type of Invasive monitoring</th>
<th>Duration of recording (days)</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>P1, F, 18y</td>
<td>Nonlesional L TLE</td>
<td>Depth</td>
<td>9</td>
<td></td>
</tr>
<tr>
<td>P2, F, 30y</td>
<td>Bilateral periventricular nodular heterotopia</td>
<td>Depth</td>
<td>14</td>
<td>C3/4: GCE: placed for comparison</td>
</tr>
<tr>
<td>P3, M, 45y</td>
<td>L HS + R frontal FCD</td>
<td>Depth</td>
<td>8</td>
<td></td>
</tr>
<tr>
<td>P4, F, 35y</td>
<td>L mesial frontal FCD</td>
<td>Depth + Grid</td>
<td>10</td>
<td></td>
</tr>
<tr>
<td>P5, F, 33y</td>
<td>L HS</td>
<td>Depth</td>
<td>3</td>
<td></td>
</tr>
<tr>
<td>P6, M, 26y</td>
<td>R insular FCD</td>
<td>Depth + Grid</td>
<td>6</td>
<td>SWE placement transientsly painful</td>
</tr>
<tr>
<td>P7, M, 18y</td>
<td>L HS + L temporal FCD</td>
<td>Depth + Grid</td>
<td>7</td>
<td>SWE disconnected during 1 GTCS</td>
</tr>
<tr>
<td>P8, M, 12y</td>
<td>R temporal porencephaly</td>
<td>Depth + Grid</td>
<td>9</td>
<td></td>
</tr>
<tr>
<td>P9, M, 23y</td>
<td>Multiple bilateral early ischemic lesions</td>
<td>Depth + Grid</td>
<td>20</td>
<td></td>
</tr>
</tbody>
</table>


GND&REF pair; however, a citation for this application could not be found. The advantage of the SWE is they are placed with a smaller size needle (25 gauge x 16 mm) compared to the sphenoidal (22 gauge x 65 mm). Also, most commercial sphenoidal electrodes are stainless steel; while the SWE is Ag-Ag/Cl and thus more stable (Tallgren et al. 2005).

Up to now, the only available scalp electrode type was the standard disc electrode and its associated drawbacks of constantly degrading due to increasing impedance or becoming loose and thus requiring periodic random maintenance. In addition, the disc electrode and its accompanying gauze, collodion, and jell cannot be adequately sterilized, so they have to be placed away from the invasive electrodes’ entry wounds. Moreover, they have to be placed at a spot which can be easily accessed by the technologists. Easy access for maintenance also means easy access for the patient who may scratch at these irritating electrodes, thereby increasing the risk of infectious complications and/or causing them to become loose.

In summary, the SWE offers a maintenance-free alternative for the GND&REF electrodes during intracranial recording that is minimally invasive and well tolerated. The only concern of the SWE is it can be dislodged without adequate fixation. This is easily controlled by some form of fixation such as collodion, Steri-Strips™, Tegaderm™, or medical grade “crazy-glue” such as Dermabond®. The SWE interconnection wire should also be secured with some form of strain-relief to prevent
accidental disconnection. Finally, it is worth mentioning that the SWE is compatible with magnetic resonance imaging (MRI), computed tomography (CT), and digital subtraction angiography (Ives 2005, Vulliemoz et al. 2009), so that the system does not need to be removed if the patient needs acute or planned imaging during the invasive work-up.

**DISCLOSURE**

There is an obvious conflict of interest here as JRI developed, manufactured, and supplied the Subdermal Wire Electrodes (patent pending), for this study. However, this was inevitable during the first study using these new electrodes in this particular application. The Subdermal Wire Electrode is currently FDA 510k approved for recording the EEG in comatose patients in the ICU. It has been used “off-label” in this clinical situation where its advantages were identified as superior to traditional clinical practice and is not available from any other source.

**ACKNOWLEDGEMENTS**

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TECHNICAL TIPS

What Doctors Expect from Technologists

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ABSTRACT. Lack of communication between electroencephalographers (EEGers) and technologists (techs) causes frustration for both parties. EEGers have trouble reading and interpreting tests and techs are left wondering if they are providing adequate examinations so that the physician can diagnose and treat patients. This article identifies barriers to communications and offers suggestions to remove these barriers.

KEY WORDS. Appropriate language, communication, electroencephalographer, patient rapport, technical impression, technical notes, technologist.

INTRODUCTION

It has come to my attention that some technologists (techs) have no idea what the electroencephalographer (EEGer) expects from them. In some labs the tech never sees the EEGer, let alone reads with him/her. I have heard uncountable myths, legends, fables, and folklore from these labs. Things like: “We can only do hyperventilation (HV) on the double banana montage,” “We have to do the montages in a certain order,” “I have to calibrate for 30 seconds on the digital machine,” or “I have to do each montage for 2 minutes 30 seconds.”

When I spoke to the EEGer at those facilities, he/she had made no such rule. It was just what the last tech did and told the new tech to do. Those lab myths may have been started three EEGers and four techs ago, then just oozed down through the ages. These myths are nothing but distorted accounts of real events, which were

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passed along to the next generation as facts, because the tech and EEGer didn’t communicate with each other.

**INTERVIEWS**

With this in mind I contacted several EEGers around the country. I asked them what they expect from their technical staff. I asked things like:

- Are the staff techs allowed to use instrumentation to enhance the recording or do you not want them to change anything?
- How about extra electrodes or special procedures?
- Do you want to see tech notes or do you prefer a “clean” record?
- Do you allow techs to select montages or are they limited to the montages that you alone select?
- How about a technical report of the EEG?
- Anything else they felt they wanted to talk about.

The answers were many and varied. Some were quite specific:

- **EE:** I would like to start the recording in ear reference montage... then a routine EEG should include at least two other montages. One bipolar longitudinal wake and one transverse (during drowsy and sleep)...

- **MS:** Discontinue (DC) photic if it is abnormal.

Good idea, but how does the EEGer know that the tech has the necessary skills to recognize an abnormal response to photic stimulation unless there has been some sort communication between them?

Some EEGers were specifically asked about how they communicate with the technical staff:

- **MN:** I don’t have much communication with the staff. [MN] has offered to read with them, but they [the techs] don’t have time. The Fellows [at MN’s facility] provide negative feedback. The Fellows should do more positive feedback.

- **EE:** I communicate in person or the techs can call me on my cell phone in case of emergency.

What/who defines an “emergency”? Did the EEGer and tech discuss emergencies? Does the tech have the necessary skills to recognize EEG emergencies? Communication between the EEGer and the technical staff can sometimes be a matter of patient safety.

When I initiated communication with physicians from around the country I found there were several common expectations, most of which they were not only willing to discuss, but eager to discuss.
PATIENT RAPPORT

Nearly every one of the EEGers that I contacted commented on the rapport the technologist should establish with patients prior to and during the procedure.

WT: Conduct the study in a comfortable and professional manner in an effort to relax the patient, facilitate the study, and promote a team approach that reassures the patient.

GF: I expect good patient interaction and courtesy, of course. . . .

WT: Introduce yourself; explain the procedure, the reason for referral, and the steps to perform the EEG without providing feedback on the results to the patient.

They offered several ways that you can make the experience of having an EEG pleasant. Some suggested that you begin your interaction with introductions. “Hi, my name is Poly Spike. I am a registered EEG technologist and I will be conducting your test today.” Shake their hand, check the identification (ID) band, and verify the patient’s name by having them say it. If the patient can’t or won’t speak, verify their identity through a family member or other healthcare professional.

Others said to escort the patient to the testing room, and explain why you are doing the test and how it will be done. If the patient is in a wheelchair, sit down yourself. Try not to tower over the patient and put yourself in a “higher” position than they are. Sure you need to take notes on the history sheet, but don’t make history taking seem like an interrogation.

It can be difficult for some patients to get up on a cart or gurney. If you have one that has been in your lab since 1980 you may want to reconsider something else. Maybe a recliner chair would be easier to get in/out of. Many patients who present to the EEG lab have limited use of limbs or have weakness that inhibits their ability to climb up on a gurney.

RECORDING

Electrode Placement

BA: Correct placement is a must.

EEGers expect that when you indicate that an electrode is placed at T3, they expect it to be at T3. Not near T3 or close to T3, or approximately T3, or where you always put T3. It is supposed to be at T3, period. Same goes for all the other electrodes. That means you have to measure, and measure correctly every patient, every time. If you are not measuring, then you should indicate that your electrodes are “10–20 placement approximated.”

You are dealing with a portion of a patient’s medical records. To indicate that the placement is “10–20” when it isn’t could be considered falsifying medical records. Be careful.
Impedance

BA: Low impedance is a given.

JK: I can’t improve data [change impedance or placement errors]; just be careful, and do it right the first time.

What happens when electrodes have high or unbalanced impedance is a topic for another paper. But, again all the EEGers with whom I discussed this said that they expect the impedance to be below 10 Kohms, at least. If your lab uses the American Clinical Neurophysiology Society (ACNS) Guidelines (ACNS 2006) or has a policy that says impedance is to be below 5 or even 10 Kohms before you start the recording, then you must maintain that level of impedance.

I once visited a lab where they checked impedance on every patient. If there were high impedance electrodes, they simply went on with the test. They never corrected them. If you do that, you are simply documenting your malpractice.

TECHNICAL NOTES

It is unanimous. All the EEGers I talked with wanted lots of notes on the recordings. So, what do you write and when?

GF: I expect copious annotation on the records so I can get a flavor for what the patients are up to. Without that, I’m flying “blind” as an interpreter.

WT: Annotate where appropriate and use descriptive terminology to represent “events” i.e., avoid the term seizure and rather describe in detail. For example, side-side head shaking, back arching, sobbing, etc., to help the interpreting MDs identify the events in question.

WT: Be the eyes of the EEGer during the recording, as after the study is complete, there is no way (unless video is available) to go back and capture the moment.

One EEGer suggested that students and staff techs alike annotate something on the EEG every 30 seconds, at least! Write when the patient moves and when patients don’t move. And even note when the tech moves.

TECHNICAL IMPRESSIONS

Along those same lines we must visit the issue of technical impressions. Some EEGers put it this way:

WT: Interpretation is required for everything in life. Pattern recognition is essential to ferret out EEG emergencies and to troubleshoot artifacts or augment abnormalities. Techs are smarter than they think.

JM: Preliminary impression from the tech is important.

GF: I appreciate it when the tech flags potential abnormalities, but it isn’t a requirement.
COMMUNICATION

BA: What I have always wanted from a tech is awareness of what is going on and an ability to respond.

JK: Comments on the EEG from the techs are a must.

EE: I like the tech’s impression.

So there you have it. Most EEGers expect some sort of description of the EEG from the tech performing the study. So what if you make a mistake and call a vertex sharp wave a focal spike? It becomes a learning experience and may even trigger some communication between the tech and the EEGer.

More comments on tech reports:

GF: I appreciate a tech impression ... since it’s a way for us to interact with each other and hone our skills.

GF: ... the tech’s sharp eyes have, from time to time, definitely improved the quality of care the patients are getting ...

MB: I expect the tech should be able to give me a verbal brief impression of the EEG he/she just recorded.

MN: They [the techs] don’t use tech reports at this facility.

APPROPRIATE LANGUAGE

If two people are trying to talk to one another and one speaks French and the other speaks Spanish, there are bound to be problems. Oh, sure, there may be a few words that are the same in each language. But clear communication will be impossible.

Simply saying “there is some slow stuff in the back” does not describe an EEG. How slow-delta, theta? Back-occipital, posterior temporal, parietal, all of them? Be clear. You must learn to speak the language of EEG. A few EEGers suggested reading sessions, once a week.

WT: If any questions of a serious nature arise, i.e., status, contact the referring MD to inform him or direct EMS.

How do you decide if a particular case is “serious”? This is a clear example of the need for clear communication between the EEGer and the technical staff. The EEGer must make clear what he/she considers an emergency, and the technical staff has to have the knowledge to recognize and report it appropriately.

Techs must be able to recognize abnormalities and be able to describe them. How are you going to inform the EEGer of an emergency if you can’t describe the EEG or know what is important and what is not? With the EEGers help, you can learn how and when to call him/her.

INSTRUMENTATION/SPECIAL ELECTRODES

Another topic brought up by several EEGers is instrumentation changes.
WT: Use creativity to answer the question of why the patient was referred... apply extra electrodes, i.e., T1/T2, monitor movements, look for additional sources that are helpful to explain/augment the primary features of the recording.

GF: They need to add electrodes or use special montages when an abnormality is not clear.

BA . . . the choice of montage is critical with each case

JK: Take the job seriously. It isn’t a factory job. Every patient is different and the test should be tailored to the patient undergoing the study.

RF: Ask yourself ‘How can I run this EEG to answer what the doctor is asking?’

MB: What I hate to see is a thoughtless EEG recording.

MS: Think for yourself, do extra things, be curious. All patients are different.

In order to be completely fair, and prove that I don’t only use EEGers who agree with me, I should say that one EEGer asked that no special montages be used. He wants the tech to just stick to his 8 routine montages. That EEGer did not mention the use of extra electrodes or instrumentation changes.

MB: Given that I can digitally change montages, filter settings, and seconds per page, it doesn’t matter that much to me how exactly the EEG was recorded.

MN: Use instrumentation as necessary. I prefer filters at 0.3 and 70, but will settle for 1 to 70. Don’t use filters to eliminate artifact.

ARTIFACTS

We have to discuss how to handle artifacts. My preference would be that EEGers would never be very good at identifying artifacts, because they never ever see any! Clearly this isn’t going to happen. There has never been an EEG recorded that was completely artifact free. Patients will swallow, blink, or move sometime during the EEG recording. One EEGer did say he could identify the tech that ran the record by the type of artifact he saw!

MISCELLANEOUS COMMENTS

JM: I need to know the gestational age. (I’ll bet he is a pediatric neurologist.)

GF: I expect our techs to welcome ENDT students into the lab, teach them, and treat them with respect. Wow! That is interesting. How often are we asked to “train” the next tech or to accept students who are in our labs as part of their “clinicals”? Do we look at this as an opportunity to pass along our knowledge, a way for us to bring the next generation of END techs up to speed, or maybe even to “pay back” the time someone else spent teaching us?

WT: Perform the test in accordance with the ACNS Guidelines.

MS: It is critical to know that the technologist is aware of laboratory policies and procedures and national guidelines regarding practice and follows these.
Another wow! Do you have a policy and procedure manual in your lab? How specific is it?

SQUARE WAVE CALIBRATION/BIOCALIBRATION

When I asked RF about calibration, he told me not to go there, because calibration and biocalibration (biocal) are very confusing and controversial topics for digital EEG. In the majority, if not all digital EEG instruments, the calibration signal on the screen did not go through an amplifier. It just put a pretty picture on the screen.

There is, in most instruments, following the introduction of a known voltage, something called a “stored numeric table.” Ask your vendor to tell you how to get to this file, and how to interpret it. Please check with your manufacturer to get specifics regarding calibration. Then discuss it with your EEGer. It may or may not be required.

As for biocal, it only checks the two inputs you used. You could run the entire digital record on biocal and go back later and re-montage. What does your written laboratory protocol call for? Know the advantages/disadvantages of square wave/biocalibration for your instrument and discuss the need for them with your EEGer.

COMMUNICATION

Finally one last comment from an EEGer:

MS: I don’t think your original question can be answered in the absence of answering the converse question: What does a technologist need from an EEGer? The EEGer should be dedicated to helping the patient through their skills in EEG. . . . The technologist must be confident that when they come to the EEGer with a question that they will be treated with respect. The technologist must also trust that the EEGer provides ongoing education about the field and helps set appropriate standards of practice. One of the key reasons why we want to raise standards for technologists is to encourage technologists to become our colleagues and foster these critical needs.

Clearly, communication between the technical staff and the EEGer is a key component in performing EEGs. Communication can begin when you take every opportunity you can to read with your EEGer. Ask him/her to explain the findings. See if they have suggestions to improve the technical aspects of the recording. Offer your input in the form of a technical report, written or otherwise. Make it a team effort.

Unfortunately reviewing EEGs together is not a common activity for the EEGer and technologist. However, most EEGers will be willing to sit down with you and tell you how they want records done. You may have to initiate the conversation, possibly
even nag about it a bit, but they will come around. You just have to make it known that you are trying to make things easier for them and better for the patients. EEGers have a responsibility to the technical staff. They need to let them know what they want.

I urge you, as technologists, to talk to your EEGer. It may mean that you have to come in early, stay late, or even do it by phone, but talk to him/her. See what he/she expects of you. Don’t just rely on what the last tech said, it may or may not be accurate. Don’t be timid about speaking to your EEGers. How simple it is to just have a five minute meeting with the EEGer and ask, ”Do you care what order I do the montages in?”, “When do you want to see HV?”, “How do you want the photic program to run?”, and anything else you can think of. There should be a continuing dialogue between the EEGer and the technical staff. Rarely will the EEGer get upset because you are trying to do what he/she wants.

Don’t dump this off on your supervisor either; especially if your supervisor is not part of the technical staff. The Director of Respiratory Therapy doesn’t know the questions to ask, you do. It is your responsibility to see that you are performing EEG procedures in the manner the EEGer wants them done.

CONCLUSION

That great fountain of information, Wikipedia, says that to dispel a myth means to scatter it and drive it away, to cause it to vanish. You can dispel the myths in your lab by contacting your EEGer, setting up a time/place to discuss lab protocol, and maybe even choose a time to read together. The important thing is to open the lines of communication.

We, as technologists, cannot give our EEGer what he/she wants without knowing his/her expectations. Communicate, talk, share, speak, synergize, learn, learn, and learn some more. Don’t ever settle for, “. . .we have always done it that way.” Step out of your comfort zone and most importantly, talk to your EEGer to find out just what he/she expects.

REFERENCE

WAVEFORM WINDOW #16

14 and 6 Hertz Positive Spikes

14 and 6 Hz positive spikes have been a controversial pattern since they were discovered in 1951 by Gibbs and Gibbs who felt 14 and 6 were evidence of thalamic and hypothalamic epilepsy. Subsequent studies correlated 14 and 6 Hz positive spikes with autonomic nervous system dysfunction, psychiatric disorders, and behavioral disorders. As more and more EEGs were recorded, 14 and 6 Hz positive spikes were seen in normal individuals, especially adolescents. 14 and 6 have a peak occurrence at ages 13 to 14 years.

Also called ctenoids and 14 and 6 Hz positive bursts, 14 and 6 Hz positive spikes have a characteristic comb-like shaped waveform with a negative smooth rounded component and a positive sharp component. 14 and 6 hertz positive spikes are seen predominantly over the posterior temporal regions at the T5 (P7) and T6 (P8) electrodes during drowsiness and light sleep. Usually the 14 Hz discharge is seen but the slower 7 to 6 Hz discharges can occur either independently or in association with a 14 Hz train.

14 and 6 hertz positive spikes can occur unilaterally, bilaterally, synchronously, and independently. 14 and 6 hertz positive spikes are best recorded from widely spaced electrodes. A referential montage to the contralateral ear can be used to confirm 14 and 6.

14 and 6 Hz positive spikes have been reported in patients in coma, especially children with Reye’s Syndrome. 14 and 6 Hz positive spikes have been recorded by the author in comatose children with severe hepatic disease.

Figures were provided by Russ Phillips, R. EEG T., MA

Lucy Sullivan, R. EEG T., CLTM
Alternating left over right referential montage to the ipsilateral ear. Note the 14 and 6 Hz positive spikes in the fifth second.
FIG. 2. Alternating left over right referential montage to the ipsilateral ear. Note the positive spikes at 7 Hz in the fourth second and the vertex waves in the eighth second. Note the comb-like appearance of the positive spikes. LOC – left outer canthus; ROC – right outer canthus
Anterior to posterior left over right bipolar montage, sometimes called the “double banana.” 14 and 6 Hz positive spikes seen in the first and second second and then again in the sixth and seventh second. LOC – left outer canthus; ROC – right outer canthus.
FIG. 4. Same EEG sample as Figure 3 now reformatted into an alternating left over right referential montage to the ipsilateral ear. Note the 14 and 6 Hz positive spikes now clearly seen in the tenth second. LOC – left outer canthus; ROC – right outer canthus
FIG. 5. Alternating left over right referential montage to the ipsilateral ear. 14 and 6 Hz positive spikes seen in the third and fourth second mainly on the right. Note the 14 and 6 Hz positive spikes in the bottom channel RE – A2. The A2 electrode is in the field of the discharge and is “contaminated.” LE – left eye; RE – right eye