BRAINSTEM AUDITORY EVOKED POTENTIAL RESPONSE IN THE PROGNOSIS OF SUPERFICIAL COMA

Potencial evocado auditivo de tronco encefálico no prognóstico do coma superficial

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ABSTRACT

The coma is the persistent reduction consciousness level, not responsive to stimuli, due to low brain activity. To check the consciousness level, a feature often used is the Glasgow Coma Scale. Another method that was been showing up is the Brainstem Evoked Response Audiometry, that evaluates the activity of the ascending auditory pathways from the midbrain to the peripheral route. The test is simple, immune to depressant drugs and electrically charged environments, being the most suitable potential for monitoring the coma. The aim of his study was to examine the characterization of Brainstem Evoked Response Audiometry in mild coma (Glasgow 7-8) and its contribution. We conducted a prospective cross-sectional study in two patients in coma (Glasgow 7), state secondary to head trauma. The test results showed the presence of electrical activity on the entire route in both cases studied, with differences changes relative to the decrease in interpeak latency, morphology, and replication of the waves. These differences were contemplated whit the evolution the each case: case 1 was favorable, but the second died. These results confirm the findings in the literature that describes the Brainstem Evoked Response Audiometry normal are associated with good outcome, while abnormal results are poor prognosis flags.

KEYWORDS: Coma; Prognosis; Evoked Potentials Auditory

INTRODUCTION

The coma is a state in which consciousness is reduced to the point the individual cannot be awakened even with strong sensory stimuli that would otherwise do it. Therefore, coma can be defined as the state in which the individual is not aware of himself and his environment, with absence or extreme decrease in consciousness level. This state of unconsciousness is variable in intensity and is directly related to the extent or structure of the central nervous system affected. It is caused by lesion or dysfunction of the ascending reticular activating system (ARAS), of the cerebral cortex diffusely or both. Countless pathologies can lead the individual to coma, such as metabolic disorders, cranial traumas, infections of the nervous system

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(meningitis), intracranial hypertension and intoxication, among others[1-3].

The level of consciousness can be assessed by neurological examination and by some quantified parameters in the Glasgow Coma Scale (GCS), namely: eye response, motor response, and verbal response, which may be obtained by various stimuli, ranging from spontaneous activity to verbal stimuli and even to painful stimuli (Table 1). The advantages of this scale are the ease of evaluation and record and its universal use, which favors the standardization of language referring to levels of coma[2-5].

Table 1 – Glasgow Coma Scale

Parameters	Response Observed	Score
Eye opening	Spontaneous	4
	In response to speech	3
	In response to painful stimulation	2
	None	1
Better Verbal	Response	5
	Confused	4
	Inappropriate words	3
	Incomprehensible sounds	2
	None	1
Better Motor Response	Follows commands	6
	Localizes and removes stimulus	5
	Localizes stimulus	4
	Flexes in response	3
	Extends in response	2
	None	1

Source: Koizumi e Araújo (2005, p. 142) [5.]

The score varies from three to fifteen, and these values are representative of severe coma (lower values) state of full alertness (higher values). Intermediate intervals indicate the levels of coma, i.e., values between three and four, deep coma; between five and six, moderate coma; between seven and eight, light coma[5].

Besides the neurological examination, the use of an objective resource in the evaluation of the brainstem, where the ARAS is located, can provide significant information about the function of this structure. The Evoked Auditory Brainstem Response (ABR) can provide such data, as it consists in recording the response to auditory stimuli that generate a chart in the form of waves. This test presents seven waves that appear in the first ten milliseconds (ms) after the presentation of the auditory stimulus. Each wave has different places of origin: wave I – distal portion of the brainstem auditory nerve; II – proximal portion to the brainstem auditory nerve; III – cochlear nucleus; IV – superior olivary complex; V – lateral lemniscus; VI – lower colliculus and VII – medial geniculate body. Among them, the waves I, III and V are those that offer the most important parameters for the interpretation of ABR [6-8]. This information indicates the electrophysiological activity of the auditory system; monitors the synapses of the auditory pathways starting from the auditory nerve, passes through the entire segment of the brain stem responsible for most of the body's vital functions, to the inferior colliculus in the midbrain level. The design of this potential can be analyzed by morphology, replication, latency and amplitude of waves [9-11].

The ABR exam is the most suitable potential for monitoring the coma status and brain death diagnosis as an aid for organ donation[12] because it is simple, fast, objective, non-invasive, immune to depressant medications and electrically charged environments. The applicability of ABR in situations in ICU has been increasing due to its high reliability and reproducibility. [7,9-11,13,14].

This study aims to determine the contributions of the use of the Brainstem Auditory Evoked Potentials in the level of light coma (Glasgow 7-8), with the presentation of clinical cases.

PRESENTATION OF CASES

Initially, the project was referred to the Research Ethics Committee of the Santa Casa de Misericórdia in the city of Franca on June 1, 2011, which was approved under protocol number 103/201. For the cases included in this study, the relatives or guardians of patients were contacted and informed about the objectives of the research, the procedures to be performed, its risks and benefits, and thus the authorization for inclusion in the study was obtained by signing the Term of Consent, pursuant to Resolution 196/96.

We conducted a cross-sectional prospective study with comatose patients in the ICU of the Santa Casa de Misericórdia of Franca, between 13 and 30 September 2011.

We considered the following criteria for inclusion and exclusion of patients in the study:

Inclusion Criteria:

- Clinical diagnosis of coma with score 3-8, according to the ECG;
- No alterations in the acoustic meatus;
- Authorization of participation from family or guardian.

Exclusion criteria:

- Infectious diseases of the central nervous system;
- Demyelinating diseases, brainstem stroke, tumors located near the auditory nerve or along the auditory tract;
- Reported hearing loss or other pathologies that could compromise the presence of ABR waves;
- Artifact and background noise that exceeded 10% of the presented stimuli;
- The information about the patient was obtained from medical records and family.

For the procedure, we used the following materials:

- Equipment *Biologic* version 5.70, Model 317, two channels, coupled to a conventional computer, installed in its proper place;
- HEINE mini 2000 otoscope and specula of different sizes;
- Surface electrodes and material for their attachment;
- Personal protective equipment (gloves, masks, aprons);
- Material for recording data.

Following these criteria, two patients were selected and tested, being both male.

Case 1

Individual of twenty years and two months of age, in a coma for six days, secondary to traumatic brain injury (TBI) due to motorcycle accident. His GCS score was 7 from admission until the day of the exam.

Case 2

Individual of thirty-eight and nine months of age, hospitalized for twenty days due to traumatic brain injury followed by coma after suffering beatings. On admission he presented Glasgow 6 and at the time of assessment, G7.

Initially, the patient was stabilized, taken to the examination room within the ICU in a bed stretcher, and he was properly monitored. The following steps were performed:

- Patient´s identification and personal data (age, gender, etiology of coma, time evolution of the state of coma);
- Patients were normothermic (36-37 °C).
- Inspection of the external acoustic meatus (EAM);
- Preparation of the attachment sites for the electrodes (cleaning and placement of conductive paste);
- Attaching the electrodes: active electrode (positive) positioned on the high forehead, reference electrode (negative) positioned in the earlobe tested and ground electrode (neutral) placed in the contralateral earlobe;
- Connection to the equipment;
- Recording of ABR waves for analysis at the intensity of 90 dB HL (decibel hearing level);
- Removal of electrodes from the patient and taking him back to his bedside in the ICU;
- Parameters used in the acquisition of AEP short latency (Table 3).

Table 2 – Parameters for ABR catchment

	Type	Click, filtered click (1KHz) - 100μs *tone pip* or *tone burst* (500Hz)
Stimulus	Intensity	. 70 a 90 dB HL (integrity of CANS) . variable (threshold estimate)
	Presentation rate (*rate*)	. 20/s (integrity of acoustic nerve and auditory pathways) . 50/s (threshold estimate – various intensities)
	Polarity	Rarefied
Acquisition	Electrodes	. Active: vertex (Cz) or front (Fpz) . Reference: ear/mastoid (A1 = left, A2 = right) . Ground: front or ear/contralateral mastoid
	Registration	Ipsi and contralateral
	Analysis time (window)	. Adults = 10 to 15ms . Children = 15 to 20ms
	Filter	30 – 3000Hz
	Sample	1000 to 2000 (with replication)
	Sensitivity	10 to 20 Mv
Patient	State	Relaxed or sleeping

Source: Katz, J (2008) [6]

All procedures were performed in the ICU.

In the analysis of the exam, we considered the aspects related to waveform morphology, i.e., the presence of waves I, III and V, the replication of the waves in order to eliminate the variability and subjectivity of interpretations; the absolute latency of responses of each wave (I, III, V) and the relative latency between the interpeak intervals (I-III, III-V, I-V), following the criteria described by Hall[15] (Table 3), according to the reference commonly used in clinical practice; the ipsilateral relation of waves I-V, in which the amplitude of wave V should be greater than wave I, and the interaural comparison of waves V, with maximum difference of 0.3 ms, based on the proposal of Figueiredo and Castro Júnior (2003)[16].

Table 3 – Values for latency of waves I, III, V and their intervals I-III, III-V, I-V

Waves	Correspondent (likely)	Latency-adults (ms)
I	Distal portion to the brainstem of auditory nerve	1,5 a 1,9
II	Proximal portion to the brainstem of the auditory nerve	2,5 a 3,0
III	Choclear nucleus	2,5 a 4,1
IV	Superior olivary complex	4,3 a 5,2
V	Lateral lemniscus	5,0 a 5,9
VI	Inferior Colliculus	
VII	Medial geniculate body	
INTERPEAKS	I – III	2,14
	III – V	1,89
	I – V	4,02

Source: Hall III JW (1992) [13]

RESULTS

The ABR results demonstrated the presence of electrical activity on the entire route studied in both cases, with indication of different alterations in regards to the reduction in interpeak latencies, morphology, and replication of the waves. Such differences were contemplated with the evolution of each case.

Case 1

- Adequate morphology for registration A1 (left ear) and altered to registration A4 (right ear);
- Adequate replication for both registrations, being A2 for the left ear and A5 for the right ear.
- Absolute latency of waves I, III, V with appropriate values for both ears;
- Interpeak latency in intervals I-III, III-V, I-V with appropriate values for both ears;
- Wave I amplitude greater than wave V for both registrations, which may indicate impairment in the auditory processing[13];
- Interaural comparison in waves V with value of 0.20 ms, which does not indicate retrocochlear impairment.

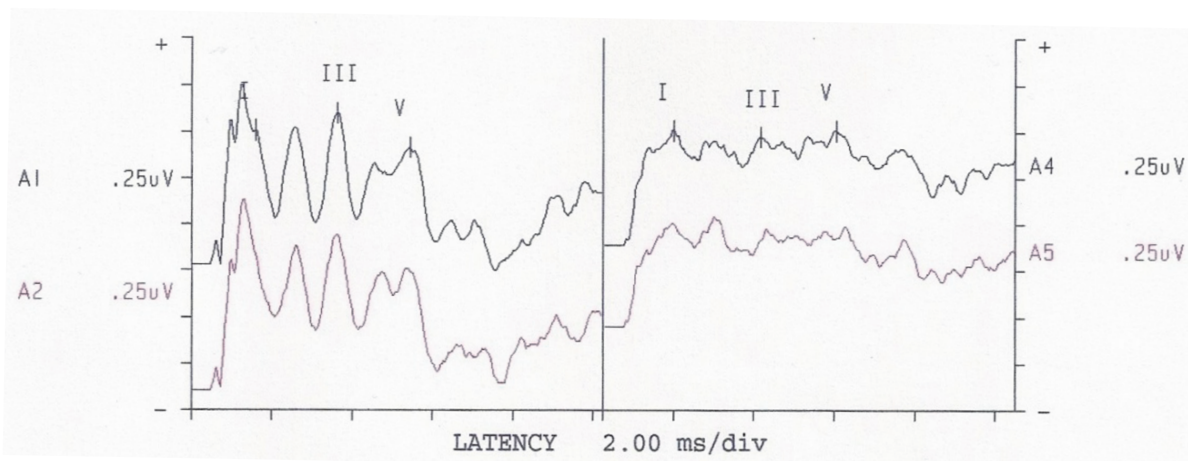

Figure 1 – Registration of ABR – Case 1

The exam results indicated preserved activity of electrical stimuli conduction in the extension of the auditory pathway evaluated, i.e., the peripheral segment and brainstem.

The design of the exam indicated the preservation of the function of the auditory pathways in the brainstem signaling integrity of ARAS and, consequently, a better prognosis, confirmed by clinical reassessment after twenty days when the patient was on the ward and in the process of discharge, with GCS score of 14.

Case 2

- Alteration in morphology, and the A6 registration (right ear) was better than A3 (left ear);
- Alteration in replication for both registrations, being A4 for the left ear and A8 for the right ear;
- Absolute latency of waves I, III, V, with adequate values for both records;
- Interpeak latency intervals I-III, III-V, I- V, with altered values for both registrations;
- Amplitude of wave I greater than wave V for both registrations;
- Interaural comparison of waves V with a value of 0.16 ms, which does not indicate retrocochlear impairment.

The exam results indicated preserved activity of electrical stimuli conduction; however, the activity was impaired, as described above. These findings suggest a worse prognosis, which was confirmed by the death of the patient.

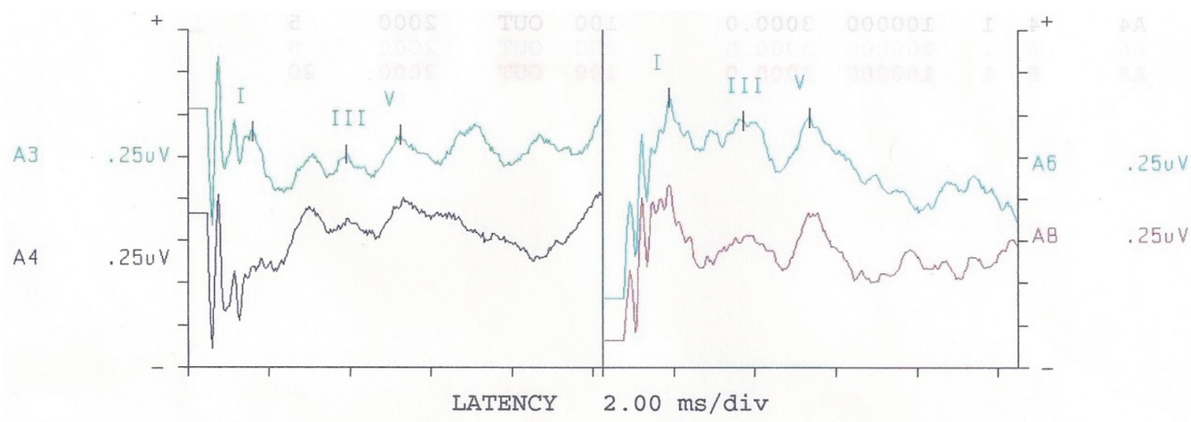

Figure 2 – Registration of ABR – Case 2

DISCUSSION

ABR stands out as a competent method in measuring the electrophysiological profile of brainstem. This happens because of the ascending auditory pathways, which occupy the entire segment of this structure in the central nervous system, which is responsible for the functions of the human organism, from the simplest ones, such as primitive reflexes until the integrated reflexes, such as those responsible for the heart rate, breathing and blood pressure control [7,11,12].

The deterioration of the function of the nervous system, most often, is rostrocaudal, this is, firstly it starts in the cortex, then through the subcortical regions and finally in the brainstem [7,11,12].

The assessment of the neurophysiological integrity of the brainstem by the ABR occurs through the synchrony of the neural element, which can be observed by wave overlapping, appropriate morphology, wave latency and interpeak intervals in normal individuals [10,12,17].

The literature contains reports of ABR as an efficient method for monitoring the states of coma, contributing to the clinical evaluation and prognosis, and also to the diagnosis of brain death. This is due to the fact that it is an exam which is objective, non-invasive, highly reliable, trustworthy, immune to depressant medications of the central nervous system and with a good reproducibility, even in environments such as the ICUs, which are electrically charged [10,11,13-15].

In this paper, we found a few studies that have confirmed the effectiveness of the ABR on the prognosis of coma. Some authors ascertained the existence of a clear relation between alterations in ABR waveform morphology with the level of dysfunction occurred in the Central Nervous System. Other authors confirmed the same proposition in their studies [15,18-21].

The cases described in this study had the etiology of head injury coma due to motorcycle accidents and beatings. In the literature, we found that the main causes that lead an individual to coma are the exogenous intoxication, the most common being alcohol abuse, traumatic brain injury (TBI) and cardiovascular diseases [22].

Traumatic brain injury, the cause of the coma, is defined in this present study as an aggression to the brain caused by temporary or permanent external physical force, which may cause a state of decreased or altered consciousness in the individual [23]. Among the leading causes of TBI are motor vehicle accidents (50%), falls (21%), hold-ups and assaults (12%), sports and recreation (10%) [24,25]. Some authors have also stated that the proportion of motor vehicle accidents is 90% for motorcycles and 9% for other vehicles [26].

Studies have shown that patients with preserved morphology in ABR research (presence of waves I, III and V), with adequate registry replication, with absolute latencies and interpeak intervals within the normal standard of normality evolved better in most cases and were discharged. Such data may highlight the role of ABR as a resource in monitoring and determining prognosis of coma [11-13].

Case 1 showed no alterations in the analysis of the ABR exam. We observed only a change of morphology in the right ear, the side on which the patient fell during the accident. The patient who suffered traumatic brain injury may show alterations resulting from impairment of the auditory pathway at the peripheral level, external ear, middle, and inner auditory nerve, or also at the level of the central auditory pathway, alterations that can be detected by ABR exam [27].

In the second case, which showed poor morphology, however, with the presence of waves I, III and V, with inadequate replication (for there was no overlapping in the design), interpeak intervals latency with altered values, showed abnormality in the auditory pathways at the level of brainstem, as well as impairment in this system. Such findings are signals of worse prognosis, which occurred with the patient in the study. In the literature, in similar situations, the patients also died because of brain impairment [11,13].

CONCLUSION

In the present study, it was possible to confirm the importance of using ABR as an auxiliary method in the evaluation of the comatose patient and the relationship between the examination findings and the prognosis. This was exemplified by the cases described, contemplated by their results, in which the presence of normal ABR was associated with good evolution of the clinical case, whereas alterations in the ABR signaled poor prognosis.

In case 1, it was ascertained that the ABR waveform morphology, worse on the right, when compared to the left side, was justified by the affected side in TBI; however, the possibility of verifying the integrity of auditory functions indicated a good prognosis, culminating in the discharge of the patient.

In case 2, the ABR test results indicated impaired activity, confirmed by the worse prognosis, and the patient evolved to death.

RESUMO

O coma é a redução persistente do nível de consciência, arresponsivo a estímulos, devido à baixa atividade cerebral. Para verificar o nível de consciência, um recurso frequentemente utilizado é a Escala de Coma de Glasgow. Outro método que se destaca é o Potencial Evocado Auditivo de Tronco Encefálico, o qual avalia a atividade elétrica das vias auditivas ascendentes, desde o trajeto periférico até o mesencéfalo. O exame é simples, imune a medicamentos depressores e ambientes eletricamente carregados, sendo o mais adequado dos potenciais para a monitoração dos estados de coma. O presente estudo teve por objetivo verificar as características do Potencial Evocado Auditivo de Tronco Encefálico no estado de coma leve (Glasgow 7 – 8) e suas respectivas contribuições. Foi realizado um estudo prospectivo transversal em dois pacientes em coma (Glasgow 7), estado secundário a traumatismo cranioencefálico. Os resultados do exame evidenciaram presença de atividade elétrica em toda extensão da via estudada, em ambos os casos, com indicações de diferentes alterações, quanto à redução na latência entre os intervalos, morfologia e replicação das ondas. Tais diferenças foram contempladas com a evolução de cada caso: caso 1 evoluiu a alta hospitalar e caso 2 evoluiu a óbito. Os resultados confirmaram os achados da literatura, que descreve que a presença do Potencial Evocado Auditivo de Tronco Encefálico normal está associada à boa evolução do caso clínico, enquanto alterações no exame podem sinalizar para um mau prognóstico.

DESCRITORES: Coma; Prognóstico; Potenciais Evocados Auditivos

REFERENCES

1. Goldman L, Ausiello D. CECIL, Tratado de Medicina Interna. 22ª ed. Rio de Janeiro: Elsevier; 2005.

2. Bacheschi LA, Nitrini R. A neurologia que todo médico deve saber. 2ª ed. São Paulo: Atheneu; 2008.

3. Greenberg MS. Handboob of Neurosurgery. 7ª ed. Tampa, Florida: Thieme; 2010. p. 279.

4. Rabello GD. In: Stávale M. Bases da Terapia Intensiva Neurologica. 2ª ed. São Paulo: Santos; 2011. p. 281

5. Koizumi MS, Araújo GL. Escala de Coma de Glasgow – Subestimação em pacientes com respostas verbais impedidas. Acta Paul. Enferm. 2005;18(2):136-42.

6. Katz, J. Tratado de audiologia clínica. 4ª ed. São Paulo: Manole; 1999.

7. Musiek FE, Rintelman WF. Perspectivas atuais em avaliação auditiva. São Paulo: Manole; 2001.

8. Møller AR, Janetta PJ. Compound action potentials recorded intracranially from the auditory nerve in man. Exp. Neurol. 1981;74:862-74.
9. Jardim M, Person OC, RapoportPB. Potencial evocado auditivo de tronco encefálico como auxílio diagnóstico de morte encefálica. Pró-Fono R. Atual. Cient. 2008;20(2):123-8.
10. Sousa LCA, Piza MRT, Ferez M, Rodrigues LS, Ruiz DB, Schmidt VB. O BERA como instrumento de avaliação funcional do tronco cerebral em cirurgias com hipotermia profunda e parada circulatória total. Rev. Bras. Otorrinolaringol. 2003;69(5):664-70.
11. Luccas FJC, Lopes JA, Caivano ABS, Lourenço FMR, Silva MMR, Silva MLI. in Stavale M. Bases da Terapia Intensiva Neurologica; 2ª ed. Santos; 2011. p. 431.
12. Souza Jr AA , Cuehuen JAN, Fukuda Y , Almeida JM , Gonçalves YP , Lara R. Audiometria de Tronco Encefálico (ABR) e estadiamento clínico (Glasgow) no diagnóstico de morte encefálica em candidatos à doação de órgãos. Rev. Bras. Ter. Intensiva. 2004;16(2):82-7.
13. Sousa LCA, Piza MRT, Alvarenga KF, Cóser PL. Eletrofisiologia da audição e emissões otoacústicas. 1ª ed. São Paulo: Novo Conceito Saúde; 2008. p. 49-82, 349-53.
14. Marseillan RF, Oliveira JAA, Vecchio FD. Audiometria de respostas elétricas do tronco cerebral humano. Rev. Bras. de Otorrinolaringol. 1997;43:229-38.
15. Hall III JW. Handbook of auditory evoked responses. Boston: Allyn and Bacon; 1992.
16. Figueiredo MS, Castro Jr NP de. Potenciais evocados auditivos precoces. In: Campos CAH de, Costa HOO. Tratado de otorrinolaringologia. São Paulo: Roca; 2003. p. 522-9.
17. Sousa LCA, Rodrigues LS, Pizza MRT, Ferreira DR, Ruiz DB. Achado ocasional de doenças neurológicas durante a pesquisa da surdez infantil através do BERA. Rev. Bras. de Otorrinolaringol. 2007;73(3):424-8.

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18. Uziel A, Benezech J. Auditory brain-stem responses in comatose patients: relationship with brain-stem reflexes and levels of coma. Eletroencephalogr. Clin. neurophisiol.1978;45:515-24.
19. Facco E, Munari M, Liviero MC, Caputo P, Martini A, Toffoletto F et al. Serial recordings of auditory brainstem responses in severe head injury: relationship between test timing and prognostic power. Intensive Care Med. 1988;14:422-8.
20. MjoenS, Nordby HK, TorvikA. Auditory evoked brainstem responses (ABR) incoma due to severe head trauma. Acta Otolaryngol. 1983;95:131-8.
21. Tsubokawa T, Nishimoto H, Yamoto T, Kitamura M, Katayama Y, MoriyasuN. Assessment of brainstem damage by the auditory brainstem response in acute severe head injury. Journal of Neurology, Neurosurgery and Psyquiatry. 1980;43:1005-11.
22. Andrade AF de, Carvalho RC, Amorim RLO de, Paiva WS, Figueiredo EG, Teixeira MJ. Coma e outros estados de consciência. Rev Med. 2007;86(3):123-31.
23. Morgado FL, ROSSI LA. Correlação entre a escala de coma de Glasgow e os achados de imagem de tomografia computadorizada em pacientes vítimas de traumatismo cranioencefálico. Radiol Bras. 2011;44(1):35-41.
24. Oliveira SG, Wibelinger LM, Luca RD. Traumatismo Cranioencefálico: uma revisão bibliográfica. [homepage na internet]. Site Fisioweb Wgate; 27 set. 2005 [Acesso em: 04 out. 2011]; [cerca de 15p.] Disponível em: http://www.wgate.com.br/conteudo/medicinaesaude/fisioterapia/neuro/traumatismo_tce.htm
25. Carlotti Jr CR, Boullosa JLR, Dias LAA, Oliveira RS, Colli BO. Traumatismos cranioencefálicos. Medicina.1995;28(4):765-76.
26. Koizumi MS. Padrão das lesões nas vítimas de acidentes de motocicleta. Rev. Saúde Pública. 1992;26:306-15.
27. Marangoni AT, Santos RBF, Suriano IC, Ortiz KZ, Gil D. Avaliação eletrofisiológica da audição em indivíduos após traumatismo cranioencefálico. Rev. CEFAC [periódico na Internet] 2013; [acesso em: 25 de março de 2013];15(1):58-68]. Disponível em: http://www.scielo.br/scielo.php?pid=S1516-18462011005000138&script=sci_arttext