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MOVEMENT-INDUCED HEART RATE CHANGES IN EPILEPTIC AND NON-EPILEPTIC SEIZURES

Gisele R. de Oliveira[1,2], Francisco de A.A. Gondim[1,2,3], Edward R. Hogan[2,3], Francisco H. Rola[2]

Abstract – Heart rate changes are common in epileptic and non-epileptic seizures. Previous studies have not adequately assessed the contribution of motor activity on these changes nor have evaluated them during prolonged monitoring. We retrospectively evaluated 143 seizures and auras from 76 patients admitted for video EEG monitoring. The events were classified according to the degree of ictal motor activity (severe, moderate and mild/absent) in: severe epileptic (SE, N=17), severe non-epileptic (SNE, N=6), moderate epileptic (ME, N=28), moderate non-epileptic (MNE, N=11), mild epileptic (mE, N=35), mild non-epileptic (mNE, N=33) and mild aura (aura, N=13). Heart rate increased in the ictal period in severe epileptic, severe non-epileptic, moderate epileptic and mild epileptic events ($p<0.05$). Heart rate returned to baseline levels during the post ictal phase in severe non-epileptic seizures but not in severe epileptic patients. Aura events had a higher baseline heart rate. A cut-off of 20% heart rate increase may distinguish moderate epileptic and mild epileptic events lasting more than 30 seconds. In epileptic seizures with mild/absent motor activity, the magnitude of heart rate increase is proportional to the event duration. Heart rate analysis in seizures with different degrees of movement during the ictal phase can help to distinguish epileptic from non-epileptic events.

KEY WORDS: epileptic seizures, heart rate, movement, non-epileptic seizures.

Alterações da frequência cardíaca induzidas pelo movimento em crises epilépticas e não-epilépticas

Resumo – Alterações da frequência cardíaca são comuns em crises epilépticas e não-epilépticas. Estudos prévios não avaliaram adequadamente a contribuição da atividade motora nas alterações da frequência cardíaca, e as crises não foram estudadas durante monitoração prolongada. No presente estudo avaliamos retrospectivamente 143 crises de 76 pacientes admitidos para monitoração com vídeo-EEG no Hospital da Universidade de Saint Louis. As crises foram classificadas de acordo com o grau de atividade motora (severa, moderada e leve/ausente) em: epiléptica grave (EG, N=17), não-epiléptica grave (NEG, N=6), epiléptica moderada (EM, N=28), não epiléptica moderada (NEM, N=11), epiléptica leve (EL, N=35), não-epiléptica leve (NEL, N=33), e aura, N=13. A frequência cardíaca aumentou no período ictal nas crises epilépticas graves, não epilépticas graves, epilépticas moderadas, epilépticas leves ($p<0,05$). A frequência cardíaca apresentou tendência a retornar aos níveis basais durante o período pós ictal nas crises não epilépticas graves, mas não nas crises epilépticas graves. As auras apresentaram frequência cardíaca basal aumentada. Um limiar de 20% no aumento da frequência cardíaca pode diferenciar eventos epilépticos moderados de eventos epilépticos leves com duração maior que 30 segundos. Em crises epilépticas com atividade motora leve ou ausente, a magnitude do aumento da frequência cardíaca é proporcional à duração do evento. A análise da frequência cardíaca em crises com diferentes quantidades de movimento na fase ictal podem ajudar na diferenciação de crises epilépticas de não epilépticas.

PALAVRAS-CHAVE: crises epilépticas, crises não epilépticas, frequência cardíaca, movimento.

[1]Department of Neurology, Saint Louis University, St. Louis, Missouri, USA; [2]Universidade Federal do Ceará, Fortaleza CE, Brazil; [3]Department of Neurology, Washington University School of Medicine, St. Louis, Missouri, USA. This paper is part of the requirements for a Master of Science (MSc) dissertation, presented at the Universidade Federal do Ceará by Dr. Gisele Ramos de Oliveira.

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Dr. Francisco de Assis Aquino Gondim – Universidade Federal do Ceará - Rua Cel Nunes de Melo 1127 – CP 3157 - 60430-270 Fortaleza CE - Brasil. E-mail: gondimfranc@yahoo.com

Autonomic changes are common in epileptic and non-epileptic seizures and may lead to sudden death in epileptic patients[1-5]. These changes are not fully understood, but may be the result of increased motor activity, emotional distress or modulation of central autonomic circuitry.

Several studies have demonstrated that heart rate (HR) is usually increased during seizures[1-7], but bradycardia[8] and even cardiac asystole[9] can occasionally occur during temporal lobe seizures. Opherk et al.[7] demonstrated that ictal HR analysis could aid in differentiating non-convulsive epileptic seizures from non-epileptic events. They proposed that in "quiet spells", if ictal HR was increased by more than 30% of baseline, there was a 97% chance that the spell was epileptic. However, no adequate control for movement was performed and HR was evaluated only during the first 10 seconds of the ictal period and postictally only in the first 1–2 minutes.

In this study, we evaluated the effect of motor activity on the heart rate changes in the pre-ictal, ictal and post ictal phases in patients with epileptic and non-epileptic seizures recorded during inpatient video EEG-EKG monitoring. The events were classified according to the degree of ictal motor activity in severe, moderate and mild/absent. Part of this study has been reported in abstract form elsewhere[10].

METHOD

Patients

We performed a retrospective analysis of 143 seizures and auras of 76 patients recorded during prolonged video EEG-EKG monitoring at Saint Louis University Hospital. We have not included more than 3 events per patient to avoid selection bias. This study was approved by the Institutional Review Board from Saint Louis University.

The events were classified according to the degree of ictal motor activity (severe, moderate and mild/absent) in: severe epileptic (SE, N=17), severe non-epileptic (SNE, N=6), moderate epileptic (ME, N=28), moderate non-epileptic (MNE, N=11), mild epileptic (mE, N=35), mild non-epileptic (mNE, N=33) and mild aura (aura, N=13).

Data acquisition

We analyzed the HR, as well as behavioral changes and EEG patterns during, and 1h prior to and 1h after each event. HR was measured by counting the QRS complex in 10s epochs throughout 3 different periods: baseline [one 10s epoch every 1 min for 10 min starting 1 h prior to each seizure (from −60 to −50 min); every 10 min from 50 until 10 min prior to the seizure (−50, −40, −30, −20, −10 min) and every 1 min from 5 min until the ictal onset (−5, −4, −3, −2, −1, −0.5 min), ictal (one 10s epoch every 30s) and post-ictal (one 10s epoch every 30s in the first minute, every 1 min during 10 min (from +1 to +10 min), and thereafter every 10 min until 60 min post event (from +10 to +60 min). The onset of the electrographic epileptic events was established by the presence of scalp EEG changes and was considered as the initial point to start ictal HR measurements in the epileptic seizures. The onset of a non-epileptic event was defined by the observation of a behavioral change and patient's subsequent report or family's assessment (in each event the patient or relative pushed the button to indicate the onset of the event). Patients with non-epileptic events were exhaustively investigated, being admitted for continuous inpatient monitoring during several days in at least one occasion. Most of them had more than one hospital admission for inpatient monitoring. The aura onset was defined by a behavioral change during monitoring, which was not considered by the patient to be his/her usual seizure event. None of these events progressed to full-blown epileptic seizures with scalp EEG changes.

Statistical analysis

We used ANOVA for Repeated Measures and Bonferroni's test to evaluate HR changes during the event progression (Baseline versus Ictal versus Post-ictal periods) for each individual seizure, within each group and between the different groups. Mean values from the first 30 min, last 10, 5 and 1 min of baseline monitoring and the first 10 and last 30 min of the post ictal period were also compared with the baseline HR values to evaluate the progression of HR changes over time. The Spearman correlation test was also employed to evaluate whether there was a significant relationship between the heart rate changes and seizure duration. Differences were considered significant if $p<0.05$.

RESULTS

In severe epileptic events HR increased ($p<0.05$) from 80.3±3.5 (baseline) to 129±3 (ictal) and 97.1±3.6 beats/min (post ictal). In severe non epileptic events, HR increased from 73.6±3.4 (baseline) to 124.2±14.2 (ictal, $p<0.05$) and 88.8±5.9 (post ictal) beats/min. In moderate epileptic events, HR increased from 80.7±2 (baseline) to 115.8±3.7 (ictal, $p<0.05$) and 85.6±2.4 beats/min (post ictal). In moderate non epileptic events, HR did not change significantly: 77.4±2.1 (baseline), 87.3±4.9 (ictal) and 79.8±4 beats/min (post ictal). In mild epileptic events, HR increased from 74.3±3.9 (baseline) to 109.3±2.9 (ictal, $p< 0.05$) and 81.3±1.8 beats/min (post ictal). In mild non epileptic events, HR did not change significantly: 77.2±1.4 (baseline), 79.5±1.6 (ictal) and 78.1±1.5 beats/min (post ictal). Aura events in epileptic patients had a higher baseline HR (86±2.6, $p<0.05$, versus baseline mild epileptic and moderate non epileptic) but HR did not change significantly: 91±2.6 (ictal) and 88.2 ±2.8 beats/min (post ictal).

In mild epileptic patients, a significant positive correlation between event duration and % ictal HR increase was observed ($R^2=0.44$, $p<0.05$), as well as a significant positive correlation between event duration and % post ictal HR increase ($R^2=0.43$, $p<0.05$). No significant correlation be-

tween event duration and % ictal or post ictal HR increase was observed in non-epileptic patients. No single moderate non epileptic patient had an ictal HR increase greater than 39.3% (versus baseline), and no single mild nonepileptic or aura had an ictal HR increase greater than 16.3 and 20.6% (respectively, versus baseline).

DISCUSSION

HR changes during seizures may increase the risk of sudden death[11]. Although sinus tachycardia is by far the most commonly reported autonomic change during seizures[4], bradycardia and many other types of arrhythmias have been described during focal or generalized epileptic events[6,8,9]. Its mechanisms may include altered baroreflex function, decreased total autonomic variability and relative increase in baseline sympathetic tone, which may further explain the propensity to cardiac arrhythmias and sudden death in epileptic patients[11]. A recent study from India also reported autonomic dysfunction in more than half of the epileptic patients[12]. Although one study has correlated the amount of dissemination of the epileptiform activity throughout the cortex with the tachycardia magnitude[6], simple partial seizures can also be associated with HR changes[5]. Experimental data have also proved that selective stimulation of areas that belong to the central autonomic network, such as the insula[13], can trigger major autonomic changes. Other important factors which can determine autonomic changes include motor activity and emotional distress.

We have previously demonstrated that dialeptic complex partial TLS (which is not associated with significant motor changes) is characteristically accompanied by tachycardia (100% of the events)[14]. Since increased motor activity could not be the culprit for the tachycardia, it is likely that it was due to the activation of components of the central autonomic network. Our interpretation is further supported by our observation that tachycardia started with focal EEG changes and was not necessarily related to spreading of the epileptiform activity.

We have also previously demonstrated that a baseline state of enhanced HR was common prior to the dialeptic partial TLS (auras) and was not due to increased motor activity during the baseline period. This finding could be the result of the self-perception (body awareness) of an imminent complex partial TLS, which was not fully developed subsequently, since ictal tachycardia was present in each individual (all) dialeptic complex partial TLS[14].

In this study we demonstrated that HR analysis can help to distinguish epileptic from non-epileptic seizures with different degrees of motor activity. HR tends to return to baseline levels during the post ictal phase in severe non-epileptic but not in severe epileptic patients. Moderate epileptic and mild epileptic events have ictal HR increase. On the other hand, moderate non-epileptic events as well as mild non-epileptic events did not have ictal HR increase.

We also observed that a cut-off of 20% HR increase may distinguish moderate epileptic and mild epileptic events lasting more than 30 seconds. This study also revealed that in epileptic events with mild or absent motor activity, the magnitude of HR increase was proportional to the event duration.

In summary, movement-induced heart rate changes may help to distinguish epileptic from non-epileptic seizures. Based on this rational, computer softwares could be created and used in the future in epileptic units to help in the distinction of epileptic and non-epileptic events by analysis of different patterns of movement-induced heart rate changes. Prospective studies are necessary to confirm the present results and to establish guidelines for combined analysis during video EEG monitoring.

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