Case Report

Ictal asystole as the first presentation of epilepsy: A case report and systematic literature review

Giada Giovannini, Stefano Meletti

Department of Biomedical, Metabolic, and Neural Science, University of Modena and Reggio Emilia, NOCSAE Hospital, Modena, Italy

ARTICLE INFO

Article history:
Received 16 June 2014
Received in revised form 27 June 2014
Accepted 29 June 2014
Available online 19 August 2014

Keywords:
Ictal asystole
Ictal bradycardia syndrome
Syncope
Epilepsy
SUDEP

ABSTRACT

We report the case of a 69-year-old woman who presented with recurring episodes of mental confusion/dizziness followed by loss of consciousness, intense pallor, and sweating. Cardiologic investigations were unremarkable. The electroencephalogram recorded during one typical episode allowed the demonstration of a right frontotemporal seizure with progressive bradycardia leading to a 9-second asystole. Following levetiracetam treatment up to 2500 mg/day, seizures with ictal asystole (IA) recurred. An MRI compatible pacemaker was then implanted. At 26-month follow-up, the patient has not had further episodes of loss of consciousness. A systematic review (1950–Apr 2014) searching for cases in which IA was an early manifestation of epilepsy led to the observation of 31 cases. The time lag between the first seizures and the correct diagnosis of IA was long (average: 27 months; median: 12 months). Clinical history alone was not sufficient to prompt a correct diagnosis of IA, and only 11 out of 31 cases presented with symptoms suggestive of a seizure disorder. The majority of patients had a frontotemporal epilepsy with a slight prevalence of left-side involvement (19 out of 31). Ictal bradycardia–asystole is an important condition that should be recognized by epileptologists, neurologists, as well as emergency department physicians. It is important to underscore that IA not only can occur in patients with drug-resistant epilepsy but also may be the first manifestation of the patient’s epilepsy.

1. Introduction

Epileptic seizures can influence the heart, and in particular, they frequently generate changes in heart rate (HR) [1]. Sinus ictal tachycardia (IT), defined as an increase in HR higher than the baseline plus one-third of all seizures), has generally no cardiac consequences, and it can anticipate the beginning of the seizure or occur simultaneously with it [2].

A less frequently observed arrhythmia is sinus ictal bradycardia (IB); defined as an R-R interval is greater than 2 s [3]. Ictal bradycardia can be found in <6% of seizures. A severe slowing of the HR leading to asystole is called “ictal bradycardia syndrome”. Ictal asystole (IA) is defined as the absence of ventricular complexes for >4 s accompanied by electrographic seizure onset [5]. Ictal asystole is a rare condition that can be found in 0.27–0.4% of patients undergoing video-EEG monitoring [6,7]. The asystole usually follows changes in the scalp-recorded EEG even if, in some cases, cardiac rhythm changes precede an obvious EEG discharge. Ictal asystole always goes along with a diffuse slowing and flattening of the electrical brain activity seen on the EEG that possibly causes the interruption of the ictal activity itself by an anoxic–ischemic mechanism [8,9]. Clinically, the IA corresponds to a loss of consciousness and a loss of muscle tone that sometimes may be accompanied by myoclonic components. This kind of autonomic dysregulation is generally found in focal chronic epilepsies: 80% of IA cases are associated with temporal lobe epilepsy (TLE) [10,11], while the remaining 20% are linked to extra-TLE (mainly frontal lobe epilepsy) [12].

To obtain relevant clinical information on IA when it occurs as an early (or as first) clinical symptom in the patient’s epilepsy history, we present a personal experience in a single case and a systematic review (without meta-analysis, narrative) on this topic.

http://dx.doi.org/10.1016/j.ebcr.2014.06.001
2213-3232/© 2014 The Authors. Published by Elsevier Inc. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/3.0/).
2. Methods

2.1. Case report

We, hereby, present a case of IA in a patient with new-onset epilepsy observed in our neurology ward.

2.2. Systematic literature search

We conducted a systematic review of the literature available in PubMed (1950–Apr 2014), searching for cases in which ictal asystole was documented (EEG with ECG registration) as an early manifestation in a new-onset epilepsy or in a newly diagnosed epilepsy [13]. We, therefore, included cases in which IA was a clinical symptom that prompted the diagnosis of epilepsy. We also included cases with an already established epilepsy diagnosis whose seizures had not already failed to respond to adequate trials of two tolerated, appropriately chosen antiepileptic drug schedules (whether as monotherapies or in combination) to achieve sustained seizure freedom. As a consequence, we excluded all the cases in which ictal asystole was observed in the context of drug-resistant epilepsy (as defined by the authors themselves).

The search keywords used, according to the MeSH terms, were the following: “Epilepsy AND Asystole OR Ictal Asystole OR Ictal Bradycardia syndrome OR Ictal Bradycardia”.

The initial search identified 829 citations. After data analysis and extraction, we identified 29 reports of suspected ictal asystole in new-onset/newly diagnosed epilepsy, but eight articles were finally excluded because they lacked a clear EEG/ECG coregistration of the phenomenon (see flowchart, Fig. 1).

The primary outcomes of the review were to evaluate the time lag between the first episode of loss of consciousness and the diagnosis of ictal asystole and to define the ictal clinical symptoms associated with or preceding loss of consciousness. We also evaluated the following: lobar involvement, etiology, hemispheric lateralization of the seizure, and therapy (AEDs chosen and pacemaker implantation).

3. Case presentation

A 69-year-old woman came to the emergency department for recurring episodes over the previous month characterized by mental confusion, light-headedness, and dizziness followed by loss of consciousness, with intense pallor and sweating. Recovery was quite rapid (20–40 s), and no postictal aphasia or other deficits were reported. Interictal neurological examination was normal.

In her past medical history, the relevant elements were as follows: a thyroid papillary tumor treated with thyroidectomy (16 years before), a catamenial migraine (since her youth), a meningioma of the left cavernous sinus treated with surgery (11 years before) followed by gamma-knife radiosurgery (5 years before), and a right frontal meningioma treated with gamma-knife radiosurgery (the year before).

The carotid sinus massage and the tilt-table test were both negative for vasovagal syncope and orthostatic hypotension. The cardiologic investigations performed (transthoracic echocardiography and Holter ECG) were also unremarkable.

Even if the semiology of the events was not suggestive of seizures/epilepsy, this possibility was considered (multiple meningiomas). Indeed, a prolonged EEG monitoring allowed the recording of one typical episode demonstrating a right frontotemporal epileptic seizure with progressive bradycardia leading to a 9-second asystole (Fig. 2A). The brain MRI confirmed the presence of multiple meningiomas together with postactinic gliosis of the right temporal lobe (Fig. 2B).

Since there were no modifications from the previous MRI, a neurosurgical intervention to remove the right frontotemporal meningioma was not considered a priority, also taking into account the previous neurosurgical history of the patient and the presence of postradiotherapy white matter changes in the right temporal lobe that could have had, per se, a role in the ictogenesis of the patient’s seizures. An appropriate antiepileptic drug (AED) therapy was then started with levetiracetam progressively titrated to 1500 mg/day. The patient remained seizure-free for a month, after which, a seizure with IA and falls recurred. Firstly, the AED therapy was increased to 2500 mg/day; however, since the patient presented with two IA events in the following month, she was readmitted to the hospital, and a dual-chamber MRI-compatible pacemaker was implanted.

Fig. 1. Flowchart illustrating the literature review process.
At 26-month follow-up, the patient has had no further episodes of loss of consciousness, and no arrhythmia was recorded by the pacemaker.

4. Discussion

The relations between seizures and the heart are very complex. The pathogenesis of these events is not completely and clearly understood. These arrhythmic events could easily occur in patients without any cardiac alterations. The mainstream theory is that the seizures may lead to the involvement and the stimulation of a circuit comprising the insula, the cingulate cortex, the amygdala, and the hypothalamus. This circuit regulates the cardiac functions through the connections to the brainstem and the spinal cord nuclei [14]. The ictal bradycardia syndrome could be found in patients with a long-lasting history of epilepsy, in particular of refractory epilepsy caused by a continuing impairment of the neurocardiac regulatory system as a result of repeated seizures and, maybe, AED treatment. The impairment of the neurocardiac regulatory system is well demonstrated by the lower heart rate variability (HRV) in patients with TLE [15]. This can make patients more susceptible also to fibrillation and tachyarrhythmias [16–18]. In these cases, IA should be particularly suspected if the usual semiology of seizures occur together with syncopal episodes [19–21]. On the contrary, the presented case demonstrates clearly that IA can be the only and the first ictal manifestation of new-onset epilepsy, and for this reason, it could be easily overlooked.

4.1. Treatment choices in the presented case

As there are no guidelines to address the management of ictal arrhythmias, we focused on the decision-making process of implanting a pacemaker [22]. Even if these events are generally benign and self-limited, it is theorized that they could contribute to SUDEP, although a link of the IA with SUDEP is still missing [23]. When an IA is detected,
| Ref.                        | Age (years) | Sex | Baseline EKG | Duration before diagnosis | Lobe | MRI/etiology | Side | Asystole duration | AED before diagnosis | AED after diagnosis | Pacemaker implantation |
|----------------------------|-------------|-----|--------------|---------------------------|------|--------------|------|-------------------|----------------------|----------------------|-----------------------|
| Fincham R.W. et al. [27]   | 68          | M   | UNK          | Some w                    | O    | Posttraumatic | R    | 33                | –                    | PHT                  | Yes                   |
| Reeves A.L. et al. [28]    | 60          | M   | Run of SVT   | 3 y                       | T    | Normal       | R    | 6                 | –                    | CBZ                  | No                    |
| Fuhr and Leppert [4]       | 69          | M   | Normal       | First episode             | FT   | Not performed | R    | 5                 | UNK                  | UNK                  | UNK                   |
| Rugg-Gunn et al. [7]       | 34          | M   | Normal       | 1 y                       | Bil  | Normal       | Bil  | 25–30             | PHT, CBZ             | PHT, CBZ             | Yes                   |
| Dubois-Tekla F. et al. [29]| 2           | M   | Normal       | 9 m                       | T    | Normal       | L    | 20                | –                    | VPA, OXCBZ            | Yes                   |
| Carinci V. et al. [30]     | 78          | M   | UNK          | 2 d                       | FT   | Previous clipping of intracranial aneurysm | L    | 10                | –                    | –                    | Yes                   |
| Ghearing G. et al. [31]    | 72          | F   | Normal       | 3 y                       | T    | Normal       | L    | 4                 | –                    | UNK                  | UNK                   |
| Bae E.K. et al. [32]       | 61          | F   | Normal       | 2nd degree AV block       | 7 m  | T            | Normal | L                | UNK                  | –                    | LEV                   |
| Dinan A. et al. [33]       | 59          | M   | Normal       | 4 d                       | T    | Ischemic changes in insular region | L    | 4                 | –                    | LEV                  | Yes                   |
| Enkiri S. et al. [34]      | 38          | M   | Normal       | Some d                    | F    | Normal       | L    | 22.5, 8.5, 24.5   | OXCBZ                | No                   |
| Schuele S.U. et al. [35]   | 14          | F   | Normal       | <1 y                      | T    | Normal       | L    | 33                | LEV                  | LEV                  | Yes                   |
| Kouakam C. et al. [36]     | 13          | F   | Normal       | 1 y                       | Vertex| Normal    | –    | 5                 | LEV                  | LEV                  | Yes                   |
| Kouakam C. et al. [36]     | 37          | F   | Normal       | 4 y                       | T    | Normal       | L    | 30                | –                    | VGB, CBZ             | No                    |
| 77 F                       | Normal       | 1st degree AV block | 5 y | T | Posttraumatic | R    | 10                | –                    | CBZ                  | No                    |
| 47 F                       | Normal       | 2 y | T | Normal       | R    | 30                | –                    | VGB                  | Yes                   |
| 54 F                       | Normal       | 8 y | T | Normal       | L    | 15                | –                    | OXCBZ, CBZ           | No                    |
| 52 F                       | Normal       | 1 y | T | Normal       | L    | 30                | –                    | CBZ                  | No                    |
| 21 F                       | Normal       | 2 y | T | Normal       | L    | 27                | –                    | CBZ, TPM             | Yes                   |
| 29 F                       | Normal       | 18 y | T | Normal       | L    | 12                | –                    | LG                 | No                    |
| 83 F                       | Normal       | 3 y | T | Normal       | R    | 20                | –                    | OXCBZ               | No                    |
| 34 F                       | Normal       | m   | T | HS          | L    | 40                | –                    | CBZ, LEV             | No                    |
| Novy J. et al. [37]        | 46           | M   | Normal       | 5 y                       | T    | Normal       | L    | 7                 | –                    | –                    | Yes                   |
| Lanzi A. et al. [38]       | 41           | M   | Normal       | 1 y                       | T    | Normal       | R    | 25                | CBZ, PRI CBZ         | CBZ                  | Yes                   |
| 63 F                       | Normal       | Left bundle brach block | 14 m | FT | DNET          | L    | 34                | TPM                  | –                   | Yes                   |
| Lee et al. [39]            | 41           | F   | Normal       | Some w                    | T    | Anti-NMDAR encephalitis | L    | 15                | –                    | Steroids, TPM, LEV   | Yes                   |
| Marynissen T. et al. [40]  | 48           | M   | Atrial fibrillation | 2 y | T | Normal       | UNK  | 15                | –                    | YES                  | Yes                   |
| Millichap J.J. et al. [41] | 15           | F   | Normal       | 1 m                       | T    | Anti-NMDAR encephalitis | L    | 22                | –                    | IVG, PHT, LEV, PB, Steroids | Yes                   |
| Strzelczyk A. et al. [42]  | 66           | F   | Normal       | 5 y                       | T    | Normal       | R    | 21                | –                    | VPA                  | Yes                   |
| Kang D.Y. et al. [43]      | 54           | M   | UNK          | 2 y                       | T    | Normal       | L    | 40                | CBZ                  | CBZ                  | Yes                   |
| Wittekind S.G. et al. [25] | 32           | M   | Normal       | 16 m                      | FT   | Normal       | R    | 18.5              | –                    | LEV                  | Yes                   |
| Heerey et al. [44]         | 24           | F   | Normal       | 1 y                       | T    | Normal       | L    | 30                | LEV, LCS             | LEV, LCS             | Yes                   |
| Present study              | 69           | F   | Normal       | Some m                    | T    | Meningiomas and gliosis | R    | 9                 | –                    | LEV                  | Yes                   |

F, female; M, male; d, days; m, months; y, years; T, temporal; FT, frontotemporal; L, left; R, right; Bil, bilateral; SVT, supraventricular tachycardia; HS, hippocampal sclerosis; CBZ, carbamazepine; CLB, clobazam; GBP, gabapentin; LEV, levetiracetam; LCS, lacosamide; LG, lamotrigine; OXCBZ, oxcarbazepine; PHT, phenytoin; PRI, primidone; TPM, topiramate; VGB, vigabatrin; VPA, valproic acid; IVG, intravenous globulin; UNK, unknown.
to avoid ictal traumatic falls and to reduce the correlated morbidity, a pacemaker is often implanted [5,24]. However, the benefit of cardiac pacing in patients with IA has not been confirmed. In a clinical series of patients with IA, the benefits of the pacemaker implantation during long-term follow-up were not clear since the recurrence rate of IA was lower than expected, and, therefore, there was no need for the pacemaker activation [2]. Since, in our case, the patient did not have refractory epilepsy (it was a new epilepsy diagnosis), we first tried to achieve seizure control (preventing asystole too) with an effective medical therapy. Indeed, it has been suggested that if one achieves, medically or surgically, seizure freedom, there is no risk of further asystole, so the pacemaker’s implantation could be avoided [25]. Contrarily, if seizure freedom could not be achieved and there is persistence of IA, a pacemaker implantation shall be taken into account, as it was in our patient [26].

4.2. Literature review

There are relatively few reported cases of ictal asystole in the context of a new-onset/newly diagnosed epilepsy. Twenty-one articles in 31 patients (18 females) were fully analyzed [4,25,27–44] (see Table 1). The asystole was self-limiting in every case and lasted 20 s on average (ranging from 4 to 40 s).

Notably, the time between the first presentation of epilepsy and the diagnosis of ictal asystole was, on average, 27 months (median: 12 months), ranging from 1 day to 18 years.

Interestingly, subjective symptomatology suggestive of a focal seizure preceding loss of consciousness was reported in only seven out of 31 cases: visual illusion [27], hallucinations [36], jamais vu [43], fear [37], psychic aura [35], and epigastric aura [28,42]. Ictal motor behaviors suggestive of a seizure disorder (tonic and clonic contractions and automatisms) were described in four patients [31,34–36]. Finally, postictal confusion or focal neurological deficit was described in seven cases [4,25,29,30,36,40,43]. Overall, in the majority of cases, as in the described patient, seizure-related auras or ictal seizure-related semiology was lacking: blurred vision, dizziness, nausea, and light-headedness were the most commonly reported symptoms.

The average age at presentation was 46 years (ranging from 2 to 83 years, median: 47 years). In five cases, interictal alterations of the basal ECG were described, and this could be identified as a further risk factor. The majority of patients had a frontotemporal epilepsy with a slight prevalence of left side involvement (19 out of 31 cases), supporting the idea that there is not a strict side effect [45,46], even if previous treatment with carbamazepine or valproate in four cases [25,43]. Overall, in the majority of cases, as in the described patient, the diagnosis was, on average, more than two years. This delay in correct diagnosis was, on average, more than two years. This delay in correct diagnosis can potentially expose the patient to risks of traumatic falls and, theoretically, to sudden unexplained death in epilepsy (SUDEP) [51–55]. Notably, the absence in the clinical patient’s history of symptoms or signs suggesting the diagnosis of a seizure disorder is insufficient to exclude IA. Indeed, only a minority of patients with IA presented with signs/symptoms suggestive of seizures/epilepsy. Therefore, an EEG with ECG monitoring is mandatory in these cases.

5. Conclusions

Ictal asystole is an important condition that should be recognized by epileptologists, neurologists, as well as emergency department physicians as the nonrecognition of this entity leads to a misdiagnosis (syncope) with consequences that can be dangerous for the patient. In particular, it is important to know that IA not only can occur in patients with a diagnosis of epilepsy already known but also may be the first manifestation of the patient’s epilepsy.

Disclosure of conflicts of interest

None of the authors has any conflict of interest to disclose. No financial or material support was received by any of the authors in conducting this research or in preparing this manuscript. We also confirm that we have read the Journal’s position on issues involved in ethical publication and affirm that this report is consistent with these guidelines.

References

[1] Neil M, Ho RT, Sperling MR. EEG abnormalities during partial seizures in refractory epilepsy. Epilepsia 2000;41(5):542–8.
[2] Schuele S, Bermeo AC, Locatelli E, Burgess RC, Luders HO. Ictal asystole: a benign condition? Epilepsia 2008;49(1):168–71.
[3] Deveno C, Struijk JJ. Autonomic alterations and cardiac changes in epilepsy. Epilepsia 2010;51(5):725–37.
[4] Fuhr P, Leppert D. Cardiac arrest during partial seizure. Neurology 2000;54:2026.
[5] Moseley BD, Ghearing CR, Mungur TM, Britton JW. The treatment of ictal asystole with cardiac pacing. Epilepsia 2011;52(4):e16–9.
[6] Rocamora R, Kurthen M, Lüdtke L, von Oertzen J, Elger CE. Cardiac asystole in epilepsy: clinical and neurophysiologic features. Epilepsia 2003;44(2):179–85.
[7] Rugg-Gunn FJ, Duncan JS, Smith SJM. Epileptic cardiac asystole. J Neurol Neurosurg Psychiatry 2000;68:100–26.
[8] Nguyen-Michel V-H, Adam C, Dinkelacker V, Pichit P, Boudali Y, Dupont S, et al. Characterization of seizure-induced syncope: EEG, ECG and clinical features. Epilepsia 2014;55(1):146–55.
[9] Schuele SJ, Bermeo AC, Alexopoulos AV, Burgess RC. Anoxia–ischemia: a mechanism of seizure termination in ictal asystole. Epilepsia 2010;51(1):170–3.
[10] Carvalho KS, Salanova V, Markand ON. Cardiac asystole during a temporal lobe seizure. Seizure 2004;13:595–9.
[11] Duplyakov D, Golovina G, Lyukshina N, Surkova E, Elger CE, Surges R. Syncope, seizure-induced bradycardia and asystole: two cases of clinical and pathophysiological features. Seizure 2014 Mar;21:501–5. [Epub 2013 Dec 14].
[12] Mascia A, Quaroni P, Sparano A, Esposito V, Sebastiani F, Occhiogrosso G, et al. Cardiac asystole during right frontolateral lobe seizures: a case report. Neurol Sci 2005;26:340–3.
[13] Thurner D, Buchler P, Kugler C, Bentz J, Neuhaus C, et al. Postictal confusion in four patients with intractable epilepsy. Seizure 2011;20:527–26.
[14] Zuber S, Arshad AB, Saeed B, Luqman S, Oommen KJ. Ictal asystole – late manifestation of partial epilepsy and importance of cardiac pacemaker. Seizure 2009; 18:455–61.
[15] Jensen K, Læge L. Cardiac changes in epilepsy. Seizure 2010;19:455–60.
[16] Espinoso PS, Lee JW, Tedrow UB, Bromfield EB, Dworetzky BA. Sudden unexpected death near in epilepsy: malignant arrhythmia from a partial seizure. Neurology 2008 May;72:1702.
[17] Ferasli M, Torelli M, Carletti M, Moretto G, Zanoni T. Seizure-induced ventricular fibrillation: a case of near-SUDEP. Seizure 2013;22:294–91.
[18] Vedovello M, Baldacci F, Nuti A, Cipriani G, Ulivi M, Vergallo A, et al. Peri-ictal prolonged atrial fibrillation after generalized seizures: description of a case and etiopathological considerations. Epilepsy Behav 2012;23:377–8.
[19] Belk JC, Sogawa Y, Ceresnak SR, Mahgerefteh J, Moise SL. Late onset ictal asystole in refractory epilepsy. Paediatr Neurol 2011;45:253–5.
[20] Varade P, Rayes M, Basha M, Watson C. Ictal syncope in a patient with temporal lobe epilepsy. Neurology 2013;80:e172–4.
[21] Rubboli G, Bisulli F, Michelucci R, Meletti S, Ribani MA, Cortelli P, et al. Sudden falls due to seizure-induced cardiac asystole in drug-resistant focal epilepsy. Neurology 2008 May;70:1913–5.
[22] Lim ECH, Lim SH, Wilder-Smith E. Brain seizes, heart ceases: a case of ictal asystole. Seizure 2008, May 12;72:1702.
[23] Lim ECH, Lim SH, Wilder-Smith E. Brain seizes, heart ceases: a case of ictal asystole. Seizure 2008, May 12;72:1702.
[24] Lim ECH, Lim SH, Wilder-Smith E. Brain seizes, heart ceases: a case of ictal asystole. Seizure 2008, May 12;72:1702.
[25] Lim ECH, Lim SH, Wilder-Smith E. Brain seizes, heart ceases: a case of ictal asystole. Seizure 2008, May 12;72:1702.
[26] Lim ECH, Lim SH, Wilder-Smith E. Brain seizes, heart ceases: a case of ictal asystole. Seizure 2008, May 12;72:1702.
[27] Lim ECH, Lim SH, Wilder-Smith E. Brain seizes, heart ceases: a case of ictal asystole. Seizure 2008, May 12;72:1702.
[26] Rugg-Gunn FJ, Simister RJ, Squirrel M, Holdright DR, Duncan JS. Cardiac arrhythmias in focal epilepsy: a prospective long-term study. Lancet 2004;364:2212–9.

[27] Finnham RW, Shivaour ET, Leis AA, Martins JB. Ictal bradycardia with syncpe: a case report. Neurology 1992 Nov;42(11):2222–3.

[28] Reeves AL, Nelson KE, Klass DW, Sharbrough FW, So EL. The ictal bradycardia syndrome. Epilepsia 1996;37(10):983–7.

[29] Dubois-Teklali F, Nguyen-Moret MA, Douchin S, Defaye P, Vercueil L. Clustering of epilepsy patients undergoing long-term video-EEG monitoring. Seizure 2011;20:817–9.

[30] Carinci V, Barbato G, Baldrati A, Di Pasquale G. Asystole induced by partial seizures: a rare cause of syncpe. PACE 2007 November;30:1416–9.

[31] Ghearing GR, Munger TM, Jaffe AS, Benarroch EE, Cascino GD. The ictal bradycardia syndrome: persistence of ictal asystole. Clin Auton Res 2007;17:221–6.

[32] Bar EK, Park K, Kim H, Jung KH, Lee ST, Chu K, et al. Ictal asystole and eating syncope in a young male with temporal lobe seizures. Dev Med Child Neurol 2006;48:687–9.

[33] Carinci V, Barbato G, Baldrati A, Di Pasquale G. Asystole induced by partial seizures: a rare cause of syncpe. PACE 2007 November;30:1416–9.

[34] Ghearing GR, Munger TM, Jaffe AS, Benarroch EE, Britton JW. Clinical cues for detecting ictal asystole. Clin Auton Res 2007;17:221–6.

[35] Carinci V, Barbato G, Baldrati A, Di Pasquale G. Asystole induced by partial seizures: a rare cause of syncpe. PACE 2007 November;30:1416–9.

[36] Ghearing GR, Munger TM, Jaffe AS, Benarroch EE, Britton JW. Clinical cues for detecting ictal asystole. Clin Auton Res 2007;17:221–6.

[37] Carinci V, Barbato G, Baldrati A, Di Pasquale G. Asystole induced by partial seizures: a rare cause of syncpe. PACE 2007 November;30:1416–9.

[38] Carinci V, Barbato G, Baldrati A, Di Pasquale G. Asystole induced by partial seizures: a rare cause of syncpe. PACE 2007 November;30:1416–9.

[39] Carinci V, Barbato G, Baldrati A, Di Pasquale G. Asystole induced by partial seizures: a rare cause of syncpe. PACE 2007 November;30:1416–9.

[40] Carinci V, Barbato G, Baldrati A, Di Pasquale G. Asystole induced by partial seizures: a rare cause of syncpe. PACE 2007 November;30:1416–9.

[41] Carinci V, Barbato G, Baldrati A, Di Pasquale G. Asystole induced by partial seizures: a rare cause of syncpe. PACE 2007 November;30:1416–9.

[42] Carinci V, Barbato G, Baldrati A, Di Pasquale G. Asystole induced by partial seizures: a rare cause of syncpe. PACE 2007 November;30:1416–9.