Video-based analysis of convulsive phenomena

PhD thesis

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CHAPTER I: VEGETATIVE DISTURBANCES DURING EPILEPTIC SEIZURES. ICTAL ASYSTOLE

1. INTRODUCTION

Epileptic activity can influence the autonomic control of the cardiovascular system on 3 different levels: ictal, postictal and interictal. Interictal dysautonomia typically occurs in a long-standing, therapy-resistant epilepsy, being characterized by the disruption of the delicate balance between sympathetic and parasympathetic activity – thus increasing the risk of fatal cardiac arrhythmias (Jansen et al, 2010). Postictal cardiorespiratory dysfunction has been correlated with sudden unexpected death in epilepsy (SUDEP), showing an incidence of 7.5/1000 patient-years (Ryvlin et al, 2013). While tachycardia appears most frequently during epileptic seizures, ictal heart rate slowing is a much less common phenomenon: the prevalence of ictal bradycardia in the epileptic population is 0.24-5.5% (Moseley et al, 2010). Ictal asystole (IA) has been reported in 0.22-0.4% of monitored patients, although the possibility of underdetection has to be considered since Rugg-Gunn et al. (2004) identified IA in 16% of patients with refractory epilepsy using implantable loop recorders. Characteristics assumed to be typical of IA are 1) focal, 2) left-sided, 3) temporal lobe seizures appearing on grounds of a 4) long-standing, 5) therapy resistant epilepsy (Strzelczyk et al, 2011; van der Lende et al, 2015). However, there is a growing evidence that query not only IA's lateralizing and localizing value but also the therapy-responsive nature and the epilepsy duration: thus many aspects of IA still remain to be inconsistent. Moreover, conflicting opinions have risen regarding the overall nature of IA: some suggest its connection to SUDEP, while others argue for its benign, self-terminating nature (Benditt et al, 2015).

2. OBJECTIVES

We aimed to investigate the rarely occurring IA on the highest number of patients ever reported. (1) Defining the localizing and lateralizing value of this ictal phenomenon can serve as useful information in delineating the seizure onset zone in the course of the preoperative planning. (2) We aimed to analyze IA in the light of the latency of its occurrence. (3) Moreover, our aim was to study IA's much-debated, possible relation to SUDEP and therapy options.

3. METHODS

Search strategy

We searched for patients admitted for presurgical evaluation in our video-EEG monitoring units in the Department of Neurology, University of Pécs and Bethesda Children's Hospital between 2006 and 2016, in whom diagnosis of IA was established. We extended our search to a systematic review of the literature. Inclusion was carried out if IA was captured by a simultaneous EEG-ECG recording. Asystole was defined as the lack of electric activity of the heart at least for 3 seconds.

Database building and analysis

Data from individual cases were collected into one database. For each included case, the following variables were collected, where available: 1) regarding the patient history: age at onset of epilepsy, age at onset of IA, IA delay (delay between epilepsy onset and IA onset), gender, preexisting heart condition; 2) concerning IA: localization and lateralization of the seizure onset zone and the seizure activity at asystole beginning, presence of clinical signs of a seizure before asystole, presence of a secondary generalized tonic-clonic seizure (GTCS), asystole duration and asystole latency (duration between onset of seizure and onset of asystole); 3) diagnostic work-up; 4) therapy and therapy response.

Since localization and lateralization were not results of a standard evaluating system applied to all cases, we had different levels of evidence regarding them. Values from 0 to 4 were assigned to each data to indicate the highest level of evidence: "0" if not mentioned, "1" for interictal EEG, "2" for ictal scalp EEG, "3" for adequate neuroimaging and "4" for epilepsy surgery with Engel Class I outcome.

Comparative analyses were also carried out. Two alignments were developed based on two different variables. The first grouping was obtained according to IA delay (delay between epilepsy onset and IA onset): cases were categorized new-onset if it was <1 year and late-onset if ≥1 year. The second alignment was performed based on asystole duration. Cut off value was 30 s: in the field of cardiology >30 s long asystole is deemed "very prolonged" or "malignant" asystole (Carvalho et al, 2015).

4. RESULTS

157 cases were identified and included in our study. Patient history and asystolic seizure characteristics are demonstrated in Table 1.

Table 1.

PATIENT HISTORY		
Variable	**Result**	**N**
Age at onset of epilepsy (years)	28±20* (r: 0-78) (M: 24.5)	116
Age at onset of IA (years)	41±18* (r: 0-80) (M: 41)	118
IA latency (years)	14±14* (r: 0-57) (M: 9)	118
Patients according to IAL	73% late onset, 27% new onset	157
Gender	51% male, 49% female	144
Preexisting heart condition	15% had, 85% did not have	103
Basal ECG	83% normal, 17% abnormal	82
Therapy responsivity	72% resistant, 28% responsive	125
Sodium channel blocker AED	65% had, 35% did not have	103
Cardiodepressor AED therapy (CBZ, PHT)	50% took, 50% did not take	103
ICTAL ASYSTOLIC SEIZURE CHARACTERISTICS		
Variable	**Result**	**N**
Seizure onset	157 focal, 0 generalized	157
Seizure symptoms before asystole	98 was present, 13 was not present	111
Aura	60 was present, 44 was not present	104
AS during a SGTCS	12 yes, 120 no	132
Asystole latency (s)	39±45* (r: 0-268) (M: 25)	66
Asystole duration (s)	18±14* (r: 3-96) (M: 15)	148

AS: asystole; IA: ictal asystole; IAL: ictal asystole latency; M: median; N: number of cases; r: range; s: second; SGTCS: secondary generalized tonic-clonic seizure; *: mean and standard deviation

Both the seizure onset zone and the focal seizure activity at asystole beginning were lateralized to the left hemisphere (Table 2.). Regarding localization, seizure onset zone was predominantly temporal (Table 2.). Depending on the level of evidence on localization it was in 80-82% of temporal, 6-10% of frontal, 3-5% of insular and 3-11% of other origin. Focal seizure activity at asystole beginning was also localized predominantly to the temporal lobe (Table 2.). Etiologies based on neuroimaging (and histology, where available) are displayed on Table 3.

Table 2.

LATERALIZATION				
	HLOE	**Result**	**Significance**	**N**
Seizure onset zone	0	62% L, 38% R	p=0.005	142
	1	63% L, 37% R	p=0.004	134
	2	63% L, 37% R	p=0.004	132
	3	64% L, 36% R	p=0.053	53
	4	65% L, 35% R	p=0.21	23
Focal seizure activity at AS beginning		69% L, 31% R	p=0.05	32
LOCALIZATION				
	HLOE	**Result**	**Significance**	**N**
Seizure onset zone	0	84% T, 16% EXT	p<0.001	153
	1	83% T, 17% EXT	p<0.001	143
	2	83% T, 17% EXT	p<0.001	139
	3	80% T, 20% EXT	p<0.001	55
	4	83% T, 17% EXT	p=0.008	18
Focal seizure activity at AS beginning		81% T, 19% EXT	p=0.001	32

AS: asystole; EXT: extratemporal; HLOE: highest level of evidence; L: left; N: number of cases; R: right; T: temporal

Table 3.

ETIOLOGY	Percent	N
Hippocampal sclerosis/atrophy	37%	22
Neoplasm	17%	10
Developmental abnormality	15%	9
Posttraumatic lesion	8%	5
Cavernoma	5%	3
Arteriovenous malformation	3%	2
NMDA encephalitis	3%	2
Encephalomalatia	3%	2
Post-stroke lesion	3%	2
Haemorrhagic infarction	2%	1
Aneurysm	2%	1
Perinatal ischaemia	2%	1

N: number of cases

Ictal asystole latency (IAL): new onset vs. late onset IA

The onset could be determined in all cases: 73% of patients had late-onset and 27% had new-onset IA. In the late-onset group, IA appeared 18±13 years (range: 1-57 y) after the epilepsy

onset. Variables showing significant differences between the two groups are demonstrated in Table 4. We found that in the late-onset group the delay between epilepsy onset and IA onset was significantly shorter in males than in females (p=0.038).

Table 4.

	N	New onset IA N (percentage)	Late onset IA N (percentage)	Significance
Female gender	144	25 (65.8%)	46 (43.4%)	p=0.023
Preexisting heart condition	103	9 (28.1%)	6 (8.5%)	p=0.014
Lesion on neuroimaging	119	11 (35.5%)	54 (61.4%)	p=0.013
Abnormal interictal EEG	80	10 (40.0%)	50 (90.9%)	p<0.001
Unilateral EEG activity at the beginning of AS	75	12 (70.6%)	20 (34.5%)	p=0.012
NMDA receptor encephalitis	60	2 (16.7%)	0 (0%)	p=0.037
Auditory aura	104	5 (15.2%)	1 (1.4%)	p=0.012
AED before IA diagnosis	103	6 (17.6%)	64 (92.6%)	p<0.001
Therapy resistant epilepsy	125	1 (4.2%)	89 (88.1%)	p<0.001
Epilepsy surgery	142	2 (5.0%)	22 (21.6%)	p=0.018
	N	**New onset IA**	**Late onset IA**	**Significance**
Age at onset of epilepsy	116	44±22 (r: 3-78)	24±17 (r: 0-70)	p<0.001
Ictal asystole latency (years)		0	18±13 (r: 1-57)	

AET: antiepileptic therapy; AS: asystole; IA: ictal asystole; N: number of cases; r: range

IA duration

IA ≤ 30 s was recorded in 90% of patients, while 10% showed “very prolonged”, “malignant” asystolic periods (> 30 s) with a mean duration of 49±18 s (r: 31-96 s). We found asystole developing during secondary GTCS to be “very prolonged” (p=0.003). There was a tendency that asystoles developing in course of extratemporal seizures were mostly “very prolonged”, while ones during temporal seizures were < 30 s (p=0.074). Cardiological comorbidity did not predispose to „very prolonged” IA (p=0.621). Asystole duration did not differ between either late/new-onset groups or between patients who received/not received sodium channel blocker medication.

Therapy and outcome

All patients received antiepileptic therapy (AET). 24 patients received only AET without cardiac pacemaker implantation or epilepsy surgery. 54% of these patients obtained complete seizure freedom with a mean follow up of 64±48 months (r: 6-128 months). In 25% AET could

prevent asystole-related falls, however non-asystolic seizures still appeared. In 21% of cases AET proved to be ineffective and asystolic seizures continued. 68% of patients received cardiac pacemaker. In the late-onset group, in patients who were not candidates for epilepsy surgery, there were no attempts to stop IA by solely AET: pacemaker implantation was performed right after the diagnosis. In the new-onset group however, 7 patients received pacemaker only after the failed attempt to control seizures with AET. Epilepsy surgery was carried out in 17% of those patients whom data on surgery were available. Temporal lobe surgery was performed in 83% and extratemporal lesion resection in 17% of cases. Temporal lobe procedures resulted in predominantly favorable, Engel Class I outcome (89%), while the extratemporal ones failed to give seizure freedom.

No IA related death was reported in any of the 157 cases.

5. DISCUSSION

Ictal asystole latency (IAL)

Based on our results we propose IA to be a multifactorial condition and depending whether it appears at the beginning of the epilepsy or presents later, different predisposing factors contribute to the ictal manifestation. We found female predominance in new-onset IA. In the healthy population women show greater vagal activity compared to men (Koeing and Thayer, 2016); we suggest that this elevated parasympathetic tone leads to an increased susceptibility to seizure-induced vagal stimuli. Preexisting heart conditions were also present in this group, mainly hypertension, coronary artery disease and electrophysiological anomalies – all 3 are well-knowingly associated with autonomic instability (Schroederer et al, 2003). The seizure activity at asystole beginning showed focal pattern and its left hemispheric predominance confirmed previous observations, namely that right sided activity induces an increase, while left sided a decrease of the heart rate (Oppenheier et al, 1992). New-onset IA developed on grounds of a newly appearing seizure disorder which proved to be rather benign: it was responsive to AET, interictal EEG and neuroimaging results were usually normal, bilateral propagation of seizure activity was absent. As for the usual occurrence of auditory aura we propose two hypotheses. According to the first one, the aura is not epileptic in origin but rather a pre-syncopal symptom as a result of cerebral hypoperfusion; however - according to our second hypothesis -, considering the benign epilepsy, normal neuroimaging and interictal EEG findings, sporadic cases of autosomal dominant lateral temporal lobe epilepsy (a condition with

LGI1 mutation) could also explain the usual auditory aura. This is supported by the recent findings of Naasan et al. (2014) who recorded asystolic periods in LGI1-antibody encephalitis.

Late-onset IA appeared 18±13 years (r: 1-57 y) after the diagnosis of epilepsy, implying to be a result of an increasing epileptogenicity or evolvement of pathological neural networks over time, both processes already proven in refractory epilepsy (van Diessen et al, 2013). The bilateral seizure activity at asystole beginning, characteristic in this group, also supports this theory. We found male predominance, moreover, we found that IA delay was shorter in men than in women. In our previous study we reported on more extended and frequent seizure spread in males compared to females, which was fortified by other investigations describing enhanced seizure-associated brain damage and different seizure propagation in males (Janszky et al, 2004; Briellmann et al, 2000) We suggest this gender difference to be the cause of the male predominance, as well as the shorter IA delay in men. Heart rate variability studies have shown increased interictal sympathetic activity in patients with chronic epilepsy (especially in chronic therapy resistant epilepsy) (Lotufo et al, 2012) and this constantly elevated sympathetic function, in turn, could result in transient parasympathetic overflows, demonstrated by Alshekhlee et al. (2008).

IA duration

Our results showed that "very prolonged" asystole is characteristic after focal seizures' secondary evolvement into a GTCS. It seems plausible that more widespread and prolonged seizure discharge could generate more severe asystolic periods. We found that extratemporal seizures tended to eventuate > 30 seconds long IA, implying that epileptic activity of various cortical structures could evoke asystole.

IA's connection to SUDEP

No IA related death was reported among the 157 patients. The link suggested between IA and SUDEP is rather weak for several reasons. Analyzing SUDEP cases in the video-monitoring unit (MORTEMUS study), SUDEP was associated with secondary generalized seizures, developing postictally due to a generalized EEG suppression leading to cardiorespiratory failure (Ryvlin et al, 2013). IA on the other hand is characteristic for focal seizures and is, by all means, an ictal phenomenon, with asystole developing within seconds after seizure onset. Furthermore Moseley et al. (2011) suggested its self-terminating, thus seizure duration-reducing effect due to the asystole-induced cerebral hypoxia. However, IA should not be deemed a benign condition considering the increased risk of suffering severe injuries. Our results show that both AET and

surgical therapies are mostly effective in reducing ictal asystolic seizures. This result queries the widespread use of cardiac pacemakers in the treatment of IA and argues for its more limited application: in AET refractory patients who are not candidates for epilepsy surgery – concordant with the therapeutic algorithm proposed by Strzelczyk et al. (2011).

6. CONCLUSION

Characteristics considered to be typical of IA are only partially legitimate. Most likely it is a result of an ictal central autonomic dysregulation, and depending whether it appears as new-onset or late-onset, different predisposing factors contribute to the ictal manifestation. In new-onset IA, we hypothesize that female gender and a preexisting heart condition serve as predisposing factors; and their co-occurrence with focal epileptic activity could evoke IA - in an otherwise benign epilepsy. Regarding late-onset IA, we hypothesize that asystolic seizures are consequences of the changes in neuronal networks in a chronic, therapy resistant epilepsy, more pronounced in males; accompanying autonomic dysregulation serving as a further facilitating factor. IA is not restricted to temporal structures: frontal and insular cortical areas could also owe IA evoking capability.

CHAPTER II: CONCUSSIVE CONVULSIONS

1. INTRODUCTION

According to their latency, following traumatic brain injury, we categorize immediate (within seconds of impact), early (<7 days) and late (>7 days) convulsions (Frey, 2003). While early and late seizures are results of epileptic activity, the pathogenesis of immediate concussive convulsions (CC) is still under debate. As they appear right after the concussion, their analysis on a human sample is extremely difficult. A video-based, objective analysis of the CC phenomenon was carried out by McCrory et al. (1997), who examined the sport related head injuries on video recordings of the Australian Football League matches, and observed a prevalence of 1.4%. They analyzed 6 cases and reported on an almost identical ictal semiology: at the moment of the impact the casualties became unconscious, then 0-2 s later a brief, asymmetric tonic posturing appeared, followed by irregular, bilateral myoclonic jerking.

Normal CT, MRI, EEG and neuropsychological results could be obtained during hospitalization and follow-up. McCrory et al. (1997, 2000) speculated, that at the moment of impact the mechanical force evokes a transient functional decerebration (similar to corticomedullary dissociation happening in convulsive syncope) and this disinhibition is responsible for the motor phenomena in CCs. Sander and O'Donoghue (1997), on the other hand, suggested the phenomenon's epileptic origin.

2. OBJECTIVES

Our aim was to analyze the semiology of video-documented CCs and to examine its possible pathomechanism from a phenomenological aspect.

3. METHODS

Search strategy

We performed a search on the video-sharing website, YouTube from videos uploaded between February 2005 and January 2016. Inclusion criteria were the following: 1) impact to the head is clearly visible, 2) convulsive motor manifestations appear after impact, 3) the length of the video was sufficient for determination of clonus characteristics: localization, symmetry, and clonus frequency, 4) visuality index is above 60% (visuality index: length of clearly observable convulsive phenomena/whole convulsion x 100), 5) no appearance of cardiorespiratory problems after impact in need of resuscitation.

Video analysis

25 videos were analyzed. The following data were collected: 1) gender; 2) estimated age; 3) the event leading to head injury; 4) the number and localization of blows to the head; 5) presence of any visible signs of head wound; 6) general characteristics of the CCs: duration of motor symptoms, loss of consciousness and state of the eyes; 7) tonic motor phenomena: fencing response (FR) (asymmetrical posturing of the upper extremities – flexion of one arm, extension of the other - presented within 1 s after head trauma), „bear hug position" (BHP) (symmetrical abduction and elevation of semiflexed arms and shoulders) and other tonic motor manifestations; 8) clonic motor symptoms; 9) lateralizing signs; 10) postictal period.

Symptoms appearing within 1 s after the hit were considered to be short-latency phenomena; otherwise they were deemed long-latency responses.

4. RESULTS

Observations of the casualty and the injury

Twenty-five CCs of 25 separate casualties were analyzed. Gender ratio was 24:1, male:female. 12% of the casualties were estimated to be in the 10-15, 44 % in the 15-20 and 44% in the 20-30 year age interval. Although we aimed to search sport-related concussions only, since some of our search terms (e.g. “fight”, “knock out”) had a wider range of meaning, 24% of the CCs resulted from assault. The 3 most common sports, resulting in CC were skateboarding, MMA (mixed martial arts) and boxing. Although at least two blows to the head could be observed in almost all events, the definite hit leading to the CC could be surely identified by the onset of symptoms. Lateralized hits were the 8 left-sided (32%) and the 7 right-sided (28%) ones, while non-lateralized were the followings: 6 occipital (24%), 3 frontal (12%) and 1 blow on the vertex. With the exception of one case, these wounds appeared to be superficial. One person suffered zygomatic bone fracture.

CCs in general

The total length of CCs ranged 7-72 s (30±18s). Apart from one casualty who seemed to show signs of only slight disorientation, loss of consciousness appeared to occur in all cases. The durations were almost always identical to the length of the CCs (31±18s), except of two cases where the unconsciousness lasted longer (10 and 45 minutes) but no intracranial injury was reported on the CT examination. Where evaluation was possible, eyes were open during 12 and closed during 3 events; and 8 non-vocalizing and 3 vocalizing (moaning, snoring) events could be observed.

Tonic motor phenomena

Fencing response (FR) occurred in 16 cases within 1 s after the impact and with the mean duration of 12±13 s (r: 4-53 s). Head turning correlated with the side of the extended arm, being ipsilateral ($p=0.0002$). Among the cases where clear left or right hit could be observed, the side of hit was ipsilateral to the side of the extended arm ($p=0.039$).

Bear hug position (BHP) was noted in 5 cases, among which 2 occurred with the latency of 0 s, (basically *in place* of the FR); while 3 had a longer, 7±4 s latency, (mostly *following* the FR). There was a tendency that if the casualty fell backwards, BHP appeared more frequently as an immediate response (p=0.12).

Tonic motor symptoms, other than FR or BHP, were identified in 12 cases. They occurred with short latency (0-1 s) in 3 cases. In this group tonic extension of both legs occurred and lasted 13±12 seconds (r: 6-43 s). In one case where the blow caused flexion of the neck, the tonic leg extension was accompanied by flexion of both arms. Tonic phenomena with a longer latency of 10±4 s were observed in 9 CCs and appeared always after the onset of the clonus, lasting 25±17 s. Generalized tonic posturing resembling typical epileptic generalized tonic-clonic seizures was not observed. Unilateral tonic posturing did not occur.

Clonic motor symptoms and lateralization

Mean latency of clonus was 6±3 s (r: 2-14 s) and mean duration was 27±19 s (r: 5-72 s). According to the onset and the spreading-pattern of the clonus, 16 bilateral, 6 focally evolving bilateral and 3 focal convulsions could be identified.

Three types of lateralizing signs could be observed: unilateral clonus (in focal and focally evolving cases), clonus asymmetry (in bilateral convulsions) and eye deviation. When lateralized, the side of the clonus was usually contralateral to the blow (p=0.039). Eye deviation was noted in one case, it was consistent with the other lateralizing sign, and was contralateral to the side of the blow. In 5 out of 25 cases the side of hit, the FR and the clonus were all consistent; meaning that the extended arm in FR was ipsilateral while the lateralized clonus was contralateral to the hit. Clonus frequency ranged 1-5 Hz, while tremulousness (>5 Hz) appeared in 3 cases. Clonus frequency did not correlate with clonus duration.

Postictal period and outcome

Thirty-six percent of the casualties seemed slightly disorientated postictally but by the time the ambulance arrived; adequate, reactive condition could be noted. Two casualties showed immediate recovery, while in another 2 cases unconsciousness persisted after the termination of the convulsion (45 and 10 minutes). In 44% the postictal period could not be evaluated.

According to YouTube comments and press releases 52% of the outcomes were favorable, no persisting symptoms remained. In 48% the outcome was unknown. No event was analyzed with a known unfavorable outcome.

5. DISCUSSION

Our data showed that CCs can be divided into two main phases: a short-latency and a long-latency phase. Some motor phenomena occurred with a short-latency (0-1 seconds) after the head trauma, while the long-latency phase involved symptoms occurring >1 second after the hit. Short latency responses were FR, BHP, and bilateral leg extension. FR was the most common immediate motor response. Based on our data - similar to McCrory et al (1997, 2000) - we hypothesize that the immediate posturing is a result of the reoccurrence of neonatal reflexes which take place on the ground of transitory disinhibition of the brainstem due to head trauma: FR is equal to the asymmetrical tonic neck reflex, BHP to the Moro-reflex and bilateral leg extension to the symmetrical tonic neck reflex.

The long-latency response phase was characterized by rhythmic clonus, sometimes superimposed with tonic motor phenomena. Although the hypothesis about convulsive syncope-like transient decerebration seems legitimate when applied to the short-latency phase, it could not serve as an explanation to the long-latency motor responses according to our data: 1) all 25 casualties showed massive, rhythmic clonus which is unlikely to originate from brainstem activity and be a product of convulsive syncope-like decerebration since the inhibitory process (which turns the tonic phase into a clonic one in grand mal) lacks in convulsive syncope, thus typically no clonus, but myoclonic jerking is associated with convulsive syncope; 2) clonus latency was longer (6±3 s, r: 2-14 s) which makes a reflex decerebration response unlikely; 3) clonus duration was too long (27±19 s, r: 5-72 s) to be deemed convulsive syncope-like; 4) unilateral or asymmetrical clonus was present in many cases, moreover, the side of the lateralized clonus and the side of the blow was contralateral – which makes a brainstem origin unlikely. Based on our findings, we speculate that motor symptoms in the long-latency phase are of cortical origin and are not in connection with the immediate responses. Even though the hypothesis about CC being a “benign” symptomatic epileptic seizure has already been raised (Sander and O’Donoghue, 1997), our data do not confirm this. Evolution of seizure semiology during CCs either was not present or was strongly atypical to grand mal: 1) separate tonic phase could be observed neither in bilateral nor in focally evolving bilateral CCs; 2) in some cases tonic posturing occurred after and superimposed to the clonic movement; 3) Jacksonian march (or other focal seizure evolution) could not be detected. 4) The big variability of clonus duration in CCs also goes against the theory of grand mal similarity. 5) Moreover, there was no evidence on tongue biting, enuresis or typical postictal confusion.

Although in the absence of electrophysiology an epileptic mechanism can not be excluded completely, based on our phenomenological approach we suggest the hypothesis that motor symptoms of the long-latency phase are attributed to cortical structures, however they may not be of epileptic origin but rather a result of a transient cortical disturbance induced by the mechanical forces.

6. CONCLUSION

CCs consist of two phases. The short-latency phase (appearing within 1 s after the impact) encompasses motor phenomena resembling neonatal reflexes and can be attributed to the disinhibition of brainstem activity. The main motor symptom of the long-latency phase is the clonus. Clonic movements are massive, rhythmic, usually long and variable in duration, appear with some latency and may carry lateralizing signs. We suggest that the motor symptoms of the long-latency phase are attributed to cortical structures, however they may not be of epileptic origin but rather a result of a transient cortical neuronal disturbance induced by the mechanical forces. Although our information regarding the outcome – due to the methodology of our study - was incomplete, in those casualties where it could be evaluated, it proved to be favourable; which underlies the previous data on the benign nature of CCs.

III. PUBLICATIONS

A. Articles related to the thesis

Tényi D., Gyimesi C., Horváth R., Kovács N., Ábrahám H., Darnai G., Fogarasi A., Büki A., Janszky J. Concussive convulsions: a YouTube video analysis. **Epilepsia** 2016; 57: 1310-1316. **Journal ranking according to MTMT: D1 IF: 5.295**

Tényi D., Gyimesi C., Kupó P., Horváth R., Bóné B., Barsi P., Kovács N., Simor T., Siegler Z., Környei L., Fogarasi A., Janszky J. Ictal asystole: a systematic review. **Epilepsia** 2017; 58: 356-362. **Journal ranking according to MTMT: D1 IF: 5.067**

Impact factor: 10.362

B. Abstracts published in scientific journals, related to the thesis

Tényi D., Rare epileptic seizure phenomena. **Archives of the Hungarian Medical Association of America** 2013; 55-56.

Tényi D., Ritka epilepsziás rohamjelenségek. **Orvostudományi értesítő** 2014; 87 (Suppl.1): 67-68.

Tényi D., Gyimesi Cs., Tényi T., Janszky J., Rare epileptical seizure phenomena. **Archives of the Hungarian Medical Association of America** 2014; 22: 37-38.

Tényi D., Ritka epilepsziás rohamjelenségek. **Orvosképzés** 2015; 2: 531.

Tényi D., Gyimesi Cs., Janszky J. Combined permanent cardiac pacemaker implantation and epilepsy surgery as treatment of ictal asystole. **Archives of the Hungarian Medical Association of America** 2015; 23: 30.

C. Oral and poster presentations related to the thesis

Tényi D. Különös epilepsziás rohamjelenségek – esetbemutatások. VII. Nemzetközi és XIII. Országos Interdiszciplináris Grastyán Konferencia, Pécs, 2015. március 19-21.

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Tényi D., Gyimesi C., Horváth R., Kovács N., Fogarasi A., Büki A., Janszky J. Fejtraumát követő akut konvulzió: YouTube videó analízis. Magyar Epilepszia Liga XIII. Kongresszusa, Szeged, 2016. május 26-28.

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