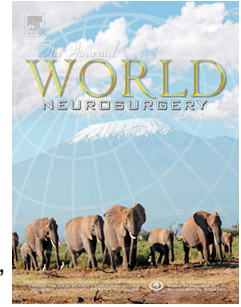


# Accepted Manuscript

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# Dislocated pacemaker electrode simulating focal epileptic state in a patient with subdural hematoma – Case report and review of the literature.

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## Introduction

Seizures are a common complication after traumatic brain injury (TBI). About 20% of patients with traumatic intracranial hemorrhage develop seizures as a neurological symptom [1]. The risk for epilepsy is highest within the first 24 hours but seizures can occur up to 10 years after TBI [2]. Additionally, it depends on the severity of the TBI and especially the type of TBI. Higher incidences of seizures are reported in literature in case of acute subdural hematoma (aSDH) and brain contusion [1, 2, 3].

Acute SDH appears in one third of TBI and mortality rates are ranging from 22% up to 60% [9, 10, 11]. Since mostly elderly patients are affected [6], the incidence of SDH is increasing due to demographic changes to an aging generation [11]. The prognosis after aSDH depends on multiple factors like age, preoperative Glasgow Coma Score (GCS), midline-shift or even signs for cerebral herniation, size of hemorrhage, type of treatment and time from TBI until operation when necessary [4, 5, 6, 7]. Besides many factors, the appearance of seizure is an independent factor for poor functional outcome in patients after aSDH [4]. Thus, it is important to detect and treat seizures as soon as possible.

Focal epilepsy can manifest with a variety of neurological symptoms depending on the site of origin. Uncontrolled muscle contraction could be one of the symptoms. However, there are many etiologies of myoclonic disorders [8].

We present a case of suspected focal epileptic state after aSDH leading to surgical evacuation of the hemorrhage and administration of antiepileptic medication without any effect. However, rhythmic muscle contraction stopped after detection and deactivation of a dislocated pacemaker electrode.

## Case Description

We present a case of an 86-years-old female with a head trauma due to syncope. The patient was treated with phenprocoumon due to a permanent atrial fibrillation. Cranial CT scan revealed an aSDH (max. width 15mm) on the right side, causing a 9 mm midline shift to the left (Figure 1A). The patient was somnolent, but had no neurological deficit. Anticoagulation was paused and reversed with prothrombin complex concentrate after admission. 2<sup>nd</sup> CT scan 6 hours after trauma showed constant hematoma without progression. Considering her age and her overall health condition, the patient was treated conservatively. After confirming a slightly reduced hematoma in CT-scan 3 days later, we reestablished anticoagulation therapy with low molecular weighted heparin. Then the patient was transferred to a neurorehabilitation clinic in a stable clinical condition.

2 weeks later the patient was sent back to our hospital with a tonic muscle contraction in the left arm, suggested to be a focal epileptic state not responding to antiepileptic therapy (Levetiracetam, Topiramate, Lacosamid). Particularly the inflexion of the fingers was clearly visible. CT scan revealed the SDH on the right side with regular, time-dependent resorption and only slight mass effect (Figure 1B). An electroencephalography (EEG) did not show any epileptic potential. However, since the sensitivity of surface-EEG is low and the tonic muscle contraction continued under antiepileptic therapy, surgical evacuation of the SDH through a bore hole was performed (Figure 1C). Intraoperatively, there was no obvious pressure on the hematoma. However, the muscle contraction in the left arm persisted. After further examination, the muscle contraction seemed to be depending on patient's arm position. With the arm lifted, the tonic muscle contraction was pronounced in the fingers.

Reviewing her medical record, previously the patient had an implantation of an automatic implantable cardioverter defibrillator (AICD) via the right subclavian vein because of cardiac arrhythmia due to dilatative cardiomyopathy. As more often bradycard phases occurred, the AICD had been explanted and a cardiac resynchronization therapy device (CRTD) had been implanted on

the left side. In the meantime, her left ventricular pump function was decreasing due to a dysfunction of the LV-lead and a new LV-lead had to be implanted 4 weeks before the trauma, which was leading to the initial admission to our hospital. Reviewing the X-ray scans of her chest from the first and second hospital stay, we retrospectively discovered spontaneous stepwise retraction of the recently implanted LV-lead into the left subclavian vein next to the plexus brachialis (Figure 2). After deactivation of the LV-lead by a consulting cardiologist, the tonic muscle contraction in the left arm stopped immediately. After few days, we were able to transfer the patient back to the rehabilitation clinic without any neurological deficit.

## Discussion

Posttraumatic seizures are common after aSDH and occur mostly within 7 days after the initial insult [1, 18]. About 25% of the patients after necessary surgical treatment of aSDH or subacute SDH developed seizures, which is associated with poor functional outcome [1, 2, 3, 4, 18]. For this reason, even the administration of prophylactic antiepileptic medication like phenytoin or levetiracetam has been suggested in literature [18]. A retrospective study revealed that prophylactic antiepileptic therapy resulted in a significant reduction of seizures (32% in groups without prophylactic antiepileptic drugs vs. 2.6% in groups with prophylactic antiepileptic drugs) and mortality [19].

Especially due to the aging population, the incidence of aSDH and the use of oral anticoagulation (OAC) are increasing [11]. Pancyzkowski et al. showed that 50% of TBI patients had one or more OACs [11]. Especially OAC associated aSDH, like in the present case, are susceptible for recurrence, with or without bridging of the OAC [13, 15, 17]. This may contribute to prolonged seizures and affect the functional outcome. Nevertheless, some studies showed controversially that pre-morbid OAC administration does not increase recurrence rates of aSDH and does not influence mortality. This may depend on the rapid reversal of OAC and the point of OAC readmission [11, 12, 14].

Epileptic complications can manifest in multiple ways. In the presented case, the tonic muscle contraction in the left arm (especially inflexion of the fingers D I – D V) was assumed as a focal status epilepticus and was futilely treated with antiepileptic medication. However, after observing series of X-ray scans during the clinical course and through measurement of pacemaker by cardiologists, the assumed “focal epilepsy” was identified to be rhythmic muscle contractions due to the dislocated LV-lead, which was stimulating the medial and lateral fascicle of the brachial plexus.

Complication rates after implantation of cardiac rhythm management device varies from 2.6% to 12.2% per year [16, 19]. Common complications are lead dislodgement, lead failure, device infection, non-septic pocket decubitus and pocket hematoma [16]. Above all, the lead dislodgement with approximately about 30% of the complications is the most frequent early complication [16, 19]. To

our knowledge, there is no data existing about the rate of lead dislodgment in patients with implanted pacemaker due to trauma. However, the trauma (for example aSDH) could be one of the provoking factors for dislocation of pacemaker electrodes.

Muscle contraction due to dislocated pacemaker electrodes has been reported previously. Most often, the dislocated electrode was the obvious reason due to the effected muscles (intercostal muscles, diaphragm or phrenical nerves) [20, 21]. It was more difficult to differ between seizures and a dislocated electrode in a case report from 1979, in which muscle contractions of the legs appeared after implantation of a pacemaker [19]. The unique and misleading feature in the presented case was the aSDH on the contralateral side of the effected muscles strongly suggesting an epileptic origin.

## Conclusion

In case of refractory treatment of epilepsy, dislocation of pacemaker electrodes is a, most certainly, rare but possible differential diagnosis. Confirmation of electrode position and function is easily and quickly feasible and will help prevent futile seizure-directed therapy.



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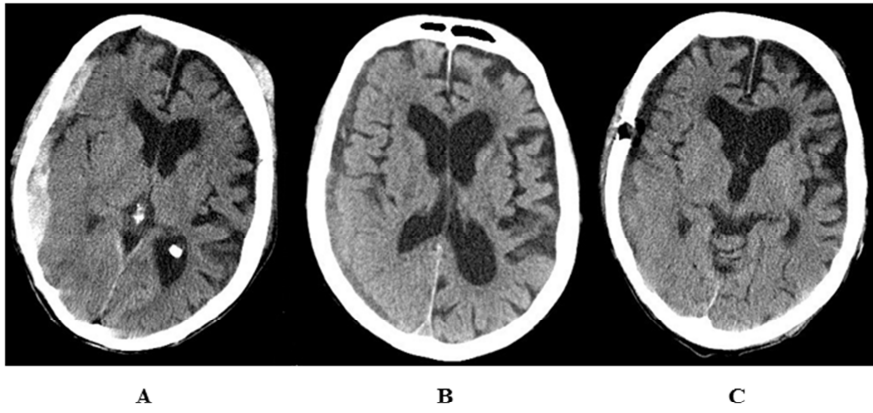
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## Figure Legends

**Figure 1. Series of CT scans:** **A.** Initial CT scan revealing acute subdural hematoma (SDH) on the right side with maximum width of 15 mm. **B.** CT scan 2 weeks later showing the SDH in time-dependent resorption with maximum width of 12 mm; **C.** CT scan after surgical evacuation of the SDH.

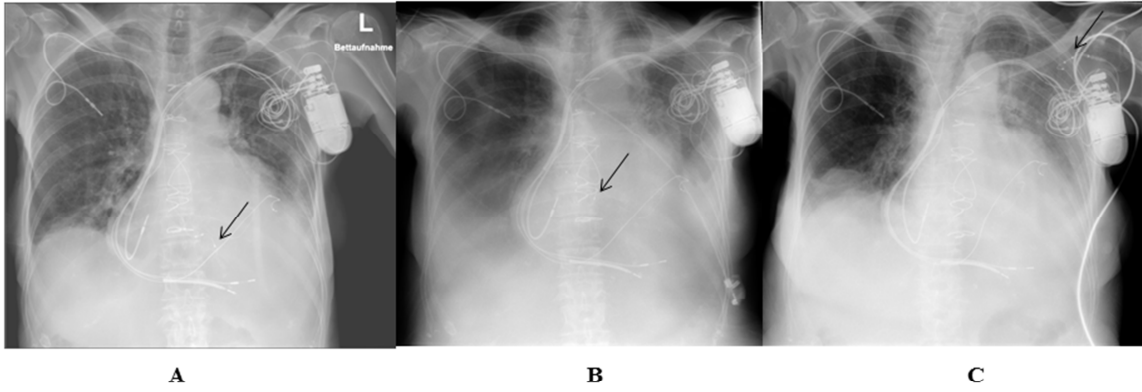
**Figure 2. Chest-X-rays:** **A.** Right Chest: electrode remaining after explantation of an automatic implantable cardioverter defibrillator (AICD); Left Chest: implanted CRTD with right atrium (RA)/ventricle (RV) electrode and left ventricular (LV) electrode, situated in the middle of coronary sinus (arrow) (2015.08.07); **B.** LV-electrode retracted into the RA (arrow) (2015.08.12); **C.** Further dislocation of the LV-electrode into the subclavian vein (arrow). At this point, the patient developed the tonic muscle contraction in her left arm (2015.09.07).

Figure 1.



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Figure 2.



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## Abbreviations list

Acute subdural hematoma	aSDH
Automatic implantable cardioverter defibrillator	AICD
Cardiac resynchronization therapy device	CRTD
Electroencephalography	EEG
Glasgow coma scale	GCS
Left ventricular	LV
Oral anticoagulation	OAC
Right atrium	RA
Right ventricular	RV
Traumatic brain injury	TBI

## Highlights

1. Epilepsy is a common complication of subdural hematoma (SDH) and lead dislocation is a common early complication after implantation of cardiac resynchronization therapy device (CRTD).
2. Dislocation of pacemaker electrode may simulate a focal epileptic state leading to an antiepileptic treatment.
3. In case of refractory treatment of epilepsy, dislocation of pacemaker electrodes is a possible differential diagnosis.