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Properties of afterdischarges from electrical stimulation in patients with epilepsy

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ABSTRACT

Objective: To investigate the properties of afterdischarges (ADs) from intracerebral electrical stimulation (ES) in patients with epilepsy who underwent stereotactic electroencephalography (SEEG) and determine the relationship between epileptogenic zone (EZ) or irritative zone (IZ) and ADs.
Methods: We retrospectively analyzed 10 patients with intractable epilepsy who underwent SEEG. ESs were delivered following the given parameters: bipolar, biphasic, 50 Hz, 0.2 ms pulse duration, 0.5–10 mA. The properties of ADs were documented, including their incidence, location, threshold, morphology and evolution.
Results: A total of 213 ADs (5%) were elicited by 4701 trains of ES. Stimulation through contacts implanted in the hippocampus (59%) generally evoked more ADs than contacts elsewhere (19%). AD thresholds for hippocampal stimulation were significantly lower than those for stimulation in grey matter. Polyspikes (58%) were the most common AD morphology. Evolution occurred more commonly with sequential spikes (47%) than with other AD morphologies (14%). There was no significant correlation between the location of ADs and EZ. However, ADs were significantly more frequently localized to IZ than areas outside IZ ($P < 0.05$).
Conclusions: There seemed to be a lack of correlation between the location of ADs and EZ. However, ADs were more likely to be elicited in IZ.

1. Introduction

Afterdischarges (ADs) are characterized by the International Federation of Societies For Electroencephalography and Clinical Neurophysiology as an EEG seizure pattern following single or repetitive electrical stimulations (ESs) of a discrete area of the brain by using cortical or intracerebral electrodes (Chatrian et al., 1974; Noachtar et al., 1999). In 1936, Adrian (1936) first stimulated an anaesthetized animal brain cortex and described ADs. Twenty years later, Jasper (1954) first observed a number of forms of ADs in patients with epilepsy. For a long period of time, ADs were considered unwanted side effects of ES in patients with epilepsy for functional mapping (Bernier et al., 1990; Lesser et al., 1984; Pouratian et al., 2004).

Despite ADs being considered the by-product of ES, ADs can be used to investigate corticocortical functional connectivity or patterns of cortical activation (Lesser et al., 2008) or as a model of epileptiform activity in human seizures (Lesser et al., 1999; Mizuno-Matsumoto et al., 2002; Motamedi et al., 2002). In 2004, Blume et al. (2004) summarized the properties of ADs from cortical ES in focal epilepsies. Furthermore, they sought to determine whether certain aspects of ADs could provide better correlation between stimulation contacts and spontaneous seizures. Unfortunately, the study failed to find the topological relationship between ADs and spontaneous seizures.

In this study, we first aimed to investigate the properties of ADs from intracerebral ES in patients with epilepsy who underwent stereotactic electroencephalography (SEEG). The incidence, location, threshold, morphology, and evolution of ADs would be emphasized in our study. Second, we wished to study stimulation contacts where the elicited ADs were recorded had a close relationship to epileptogenic zone (EZ) or irritative zone (IZ).

2. Materials and methods

2.1. Subjects

Between January 2016 and January 2017 at Xuanwu Hospital,

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Table 1
Clinical features of the patients.

Patient No.	Sex	Age (years)	Duration (years)	Electrode side	Brain MRI	No. sc	No. st	Surgery site	Pathology
1	F	32	20	L	L-HS	71	428	L-ATL + H	HS, FCDIIIa
2	F	19	10	L	L-HS	70	443	L-ATL + H + pos. Tb + Temporal Oper.	HS, FCDIIIa
3	M	28	12	B	L-HS	74	382	L-ATL + H	HS, FCDIIIa
4	M	24	6	B	L-HS	87	436	L-ATL + H	HS, FCDIIIa
5	M	28	11	R	R-TPO encephalomalacia	94	509	R-TPO	Scar with gliosis, FCDIIId
6	M	25	9	R	R-TP encephalomalacia	82	434	R-TP	PXA, FCDIIIb
7	F	12	4	R	Normal	111	540	R-pos. T + IPL	FCDI
8	M	17	10	B	Normal	116	592	R-O + pos. Tb + pH	gliosis
9	M	18	10	R	Normal	95	527	R-TPO + inf. T + H	Heterotopia, FCDIIId
10	M	29	24	B	Normal	92	410	R-ATL + H + inf. F	FCDI

Patient No., the patient's file number; Electrode side, implanted electrode side; No. sc, the number of stimulation contacts; No. st, the number of stimulation trains; F, female; M, male; L, left; R, right; B, bilateral; ATL, anterior temporal lobe; H, hippocampus; T, temporal lobe; P, parietal lobe; O, occipital lobe; F, frontal lobe; Tb, temporobasal region; pH, parahippocampus; IPL, inferior parietal lobe; Oper, operculum; pos, posterior; inf, inferior; HS, hippocampal sclerosis; FCD, focal cortical dysplasia; PXA, pleomorphic xanthoastrocytoma.

Capital Medical University, patients were diagnosed at an interdisciplinary presurgical conference depending on semiology, video EEG, and imaging data. Ten patients with intractable epilepsy underwent SEEG to identify EZ and underwent ES for functional mapping. Details of the patients are summarized in Table 1.

2.2. Stereotactic implantation of intracerebral electrodes

Presurgical preparations included T1 magnetic resonance images (3.0 T, 1 mm thickness, gap: 0) for location, magnetic resonance angiography, and magnetic resonance venography. We used a Cosman-Roberts-Well (CRW) human stereotactic instrument under local anesthesia and performed computer tomography (CT) scan (1 mm thickness, gap: 0) on the operation day. The above imaging data were transferred into the navigation (Stealthstation Tria Plus, Medtronic Inc., USA). Planning of the procedure included implantation trajectories that were based on the location of the arteries and veins. Because of the angle limitation of the CRW instrument, it was difficult to reach the target point safely and precisely by a lateral orthogonal approach. Our methodology targeted the trajectories using an oblique approach. Electrodes (HuaKe HengSheng, Beijing, China) were 0.8 mm in diameter, and each contact was 2 mm long, with interval distances of 1.5 mm. Depending on the length, each electrode had 8–16 contacts.

2.3. Determining the anatomic location of the contacts

Postimplant CT scan was performed within 24 h postsurgery. Using surgical navigation (Stealthstation Tria Plus, Medtronic Inc., USA), postimplant CT and presurgical brain MRI were merged together to display anatomical relationships. According to the length and interval of the contacts, screenshots were captured at intervals of 3.5 mm to precisely localize contacts on the MRI images.

Locations of contacts were classified into five groups (Fig. 1): grey matter, white matter, hippocampus, grey-white matter transition, and hippocampus-white matter transition. Each contact was classified according to the images captured in the axial, coronal, and sagittal plane.

2.4. Intracranial EEG monitoring

Intracranial EEG monitoring was performed using a 128-channel EEG (Brain Quick, Micromed, Italy) after SEEG implantation. Our conventional settings were as follows: The EEG signals were sampled with 1024 Hz and filtered between 0.5 and 100 Hz. The sensitivity was 600–800 uV/cm. The dose of anti-epileptic medication was variously reduced during the monitoring. The location and extent of the interictal and ictal EEGs were assessed during the extraoperative recording (DuanYu et al., 2010). EZ was defined as the site of origin and site of the primary organization of epileptic seizures (Kahane et al., 2006). IZ was the area of cortex that generates interictal spikes and was only measured by intracranial EEG in our study (Luders et al., 2006).

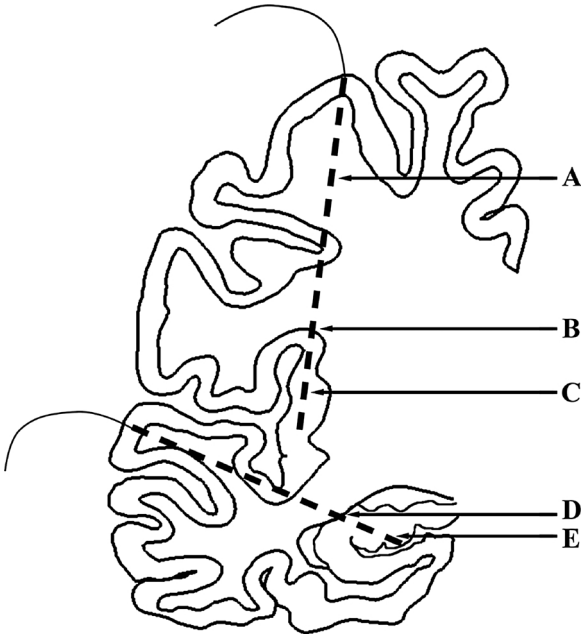

Fig. 1. The scheme of one hemisphere with potential locations of electrode contacts. Contacts were classified into five groups depending on their locations. A: white matter; B: grey-white matter transition; C: grey matter; D: hippocampus-white matter transition; E: hippocampus.

2.5. Stimulation procedure

ES (SD LTM STIM, Micromed, Italy) was performed primarily for the purpose of functional mapping. The full dose of anti-epileptic medication was re-administered before the ES. To localize motor, somatosensory, visual, and language functions, we tested each patient approximately for 1–3 h per session. Usually, there were one to two sessions per day for 1–3 days. We used 0.2-ms-duration biphasic pulses, which were repeated 50 times per second. The stimulation train lasted for 3 s. When testing each contact, we routinely commenced at a low-intensity current of 0.5 mA and increased by 0.5–2.0 mA at a time. The current increased until (1) a current of 10 mA was reached, (2) a functional change occurred, or (3) ADs were obtained (Lesser et al., 1999). When ADs stopped, we waited for at least 30 s for the trace of EEG to return to baseline to avoid further ADs (Jayakar et al., 1992; Lee et al., 2010).