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Heart rate variability remains reduced and sympathetic tone elevated after temporal lobe epilepsy surgery

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ABSTRACT

Purpose: There is evidence of autonomic dysregulation in temporal lobe epilepsy. The structures removed during temporal lobectomy are important centers of central cardiovascular control; therefore surgery may conceivably alter the cardiovascular autonomic function. The effects of temporal lobectomy on autonomic cardiac control are controversial. We investigated the effects of temporal lobectomy on heart rate variability (HRV) in the early and late postoperative periods.

Methods: We used 1-h ECG recordings to assess heart rate variability by spectral analysis in 24 consecutive patients who underwent temporal lobectomy due to intractable temporal lobe epilepsy. ECG recordings were performed before and twice (early and late) after surgery. The results were compared with age and sex matched controls.

Results: When compared with controls, all the time and frequency domain indices (SDRR, RMSSD, TP, LF and HF) were significantly lower in the patient group before surgery. Findings were similar in the early and late post-operative periods except that the LF/HF ratio increased in the patient group after the late post-operative period. Within the patient group, compared to pre-operative results, normalized HF was increased in the early post-operative period; however in the late post-operative period, LF/HF ratio was increased.

Conclusions: These findings show that in patients with intractable temporal lobe epilepsy, HRV is decreased globally in both sympathetic and parasympathetic domains. While the total HRV remains reduced throughout the postoperative periods, the LF/HF ratio, i.e., sympathovagal balance is altered, in favor of parasympathetic side early after surgery, but towards the sympathetic side after the first postoperative month.

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1. Introduction

There is increasing evidence for neurally mediated cardiac damage in patients with epilepsy. Previous clinical studies have indicated that cardiac autonomic functions may be altered interictally in patients with either generalized[1] or partial epilepsy,[1–3] mainly of temporal lobe origin. Cardiac autonomic control can be assessed clinically by spectral analysis of heart rate variability (HRV), which allows assessment of the sympathetic and parasympathetic components separately from each other. The literature of HRV investigations in epileptic adults is contradictory, indicating inhibition of both sympathetic and parasympathetic tones;[1,2,4,5] sympathetic dominance[6,7] or parasympathetic dominance.[1,8] These alterations are commonly attributed to spread of seizure discharges to autonomic centers in the cortex and various limbic structures, or the effect of antiepileptic drugs (AED).[9]

Reduced HRV has been associated with increased mortality in various clinical disorders and sudden unexpected death in epilepsy patients (SUDEP).[10] Several studies have reported that the mortality ratio decreases after epilepsy surgery,[11–14] which is in part attributed to decrease in the number or complete cessation of seizures. Given that some of the suprabulbar centers that are removed during temporal lobe epilepsy (TLE) surgery are also involved in autonomic cardiovascular (CV) control, any possible changes in interictal cardiac autonomic function postoperatively may also contribute to this finding. Very few studies have investigated the effects of TLE surgery on autonomic control of CV function[15–18] and the results are conflicting. Besides, the time

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course of post-operative alterations is not known; and it is not clear if the changes reflect a functional loss secondary to removal of the relevant anatomical structures, or if they are due to a malfunction secondary to late plastic changes. Therefore we designed a prospective study to evaluate the characteristics of HRV in TLE patients who underwent temporal lobectomy with amygdalo-hippocampectomy and we investigated HRV within the first postoperative week and after the first month of surgery.

2. Patients and methods

2.1. Patients

Twenty-four consecutive patients (11M, 13F; age: 15–48 (median 27.5 years)) who underwent temporal lobectomy due to intractable epilepsy in our center were included in this prospective study (Table 1). Patients with clinical or laboratory evidence of coexisting heart failure, coronary artery disease, relevant rhythm disturbances such as marked sinus arrhythmia, atrial fibrillation or flutter, frequent ectopic beats or conduction blocks, diabetes mellitus, uremia or any other disease that might affect autonomic function were excluded from the study. None of them had a clinical history or complaints regarding the ANS. General physical and neurological examinations of the patients were not remarkable. Duration of epilepsy ranged from 2 to 43 (median: 16) years. Preoperatively the patients were investigated with interictal/ictal scalp video-EEG recordings, high resolution cranial MR imaging (1.5–3 T), and interictal/ictal SPECT and PET when needed. All of them were right handed. Clinical and laboratory findings were discussed by an epilepsy surgery team before a final consensus was reached. The patients underwent standard anterior temporal lobectomy with amygdalo-hippocampectomy (13R, 11L), after which they were followed up for at least a year.

Propofol, fentanyl, nitrous oxide or desflurane were used during general anesthesia. All the patients gave informed consent. During testing in the preoperative and postoperative periods, no study participant received any additional drugs, which are known to interfere with autonomic function. Neither the antiepileptic drugs nor their dosages were changed during the evaluation period.

Twenty-three age- and sex-matched healthy volunteers (13F, 10M; age: 15–50 (median 27 years)) served as a control group.

2.2. HRV

The preoperative evaluation was performed 1–21 (median 2.5) days before the surgery, while postoperative evaluations were carried out twice: (a) 3–8 (median 5) days after surgery (early postoperative period) and (b) 35–188 (median 59.5) days after surgery (late postoperative period). Although we aimed to perform the late postoperative tests at 6 weeks to 3 months after surgery, some of the patients admitted before or after the proposed time-window for postoperative control, leading to the large variation in timing of the late postoperative period. Heart rate variability recordings were performed between 9 AM and 4 PM. Patients were tested in relaxed, supine position in a room with ambient temperature of about 20 °C and were awake during the procedure. Approximately 1-h of recordings were made for each session. All but one (who had a complex partial seizure 16 h before the test) were free of seizures for at least 24 h prior to the testing. None of them reported auras or had seizures during the test.

The ECG signals were recorded with the negative electrode 5 cm right of the suprasternal notch, the positive electrode at the left anterior-axillary line in the sixth intercostal space, and the indifferent electrode on the right mid-axillary line. The analog output of the ECG amplifier (model PM4; Graseby Medical, UK) was connected to a custom-made triggering device, which generated a TTL pulse (a standard +5 V pulse for "Transistor–Transistor Logic") for each incoming R wave of ECG. The pulses were fed to the serial port of a PC. Subsequent stages of recording, editing, and analyses were performed using software developed in our laboratory. The software continuously scanned the computer's serial port and detected the time intervals between incoming pulses at a

Table 1
Clinical charateristics of the study group.

Patient no.	Age (years)	Gender	Seizure type	AED and daily dose (mg)	MRI findings/pathology	Side of surgery	Outcome (Engel class)
1	15	M	SPS, CPS, GTCS	DPH (300)	Left TL tumor (ganglioglioma)	L	I
2	15	F	SPS, CPS	CBZ (800), GBP (900)	HS	R	I
3	25	M	SPS, CPS, GTCS	OXCBZ (1800)	HS	L	I
4	17	F	SPS, CPS, GTCS	OXCBZ (1200)	HS	L	I
5	22	M	SPS, CPS	CBZ (1200), PHB (100)	Right amygdalar hyperintensity	R	I
6	23	F	SPS, CPS	VGB (2000), GBP (1600)	HS	L	I
7	24	M	CPS, GTCS	DPH (100), CBZ (400), GBP (800)	HS	R	I
8	27	F	CPS, GTCS	CBZ (800), GBP (600)	HS	R	II
9	37	M	SPS, CPS	GBP (1600), PHB (150)	HS	R	I
10	28	F	SPS, CPS	OXCBZ (1200)	Left hippocampal hyperintensity without atrophy	L	III
11	41	F	CPS, GTCS	VGB (2000)	Left TL cavernoma	L	I
12	33	M	SPS, CPS, GTCS	CBZ (800)	HS	R	I
13	31	F	SPS	CBZ (800), LTG (300), PHB (200)	Right TL cortical dysplasia	R	II
14	28	F	SPS, CPS	CBZ (2000), PRM (1000), LTG (125)	HS	R	I
15	44	F	SPS, CPS, GTCS	CBZ (1200), GBP (900)	HS	R	II
16	21	F	SPS, CPS, GTCS	OXCBZ (900), PRM (500)	HS	R	II
17	48	M	SPS, CPS, GTCS	CBZ (1200), VGB (2000); PHB (125)	HS	L	I
18	36	F	SPS CPS GTCS	CBZ (800), TPM (100)	HS	L	I
19	23	F	SPS CPS	CBZ (800), PHB (200), GBP (2000)	HS	L	I
20	34	M	SPS, CPS, GTCS	PRM (500), CBZ (800)	HS	L	II
21	43	M	SPS, GTCS	TPM (150), CBZ (400)	HS	R	I
22	18	F	SPS, CPS, GTCS	CBZ (600)	Ganglioglioma, HS	R	I
23	28	M	SPS, CPS, GTCS	CBZ (1200), LEV (1500)	HS	R	I
24	43	M	CPS, GTCS	TPM (200), OXCBZ 1800	Ganglioglioma	L	I

AED, antiepileptic drug; SPS, simple partial seizure; CPS, complex partial seizure; GTCS, generalized tonic clonic seizure; R, right; L, left; TL, temporal lobe; HS, hippocampal sclerosis; DPH, phenytoin; CBZ, carbamazepine; GBP, gabapentin; OXCBZ, oxcarbazepine; PHB, phenobarbital; VGB, vigabatrin; LTG, lamotrigine; PRM, primidone; TPM, topiramate; LEV, levetiracetam.

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