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Review

Heart and lung in the postictal state

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ABSTRACT

Ictal events include those of the autonomic nervous system. This sympathetic stimulation may persist into the postictal period, affecting cardiopulmonary function. Such postictal effects include potentially fatal alterations in cardiac rhythm and ventilatory function, management challenges such as pulmonary edema, and diagnostically confusing laboratory abnormalities such as fever and cerebrospinal fluid pleocytosis. These and other effects on the heart and lung in the postictal period are accordingly reviewed.

1. Sympathetic stimulation

The stimulation of the sympathetic nervous system during seizures has been referred to as a *sympathetic storm* [1]. The effects extend into the postictal state and are evident as systemic hypertension, tachycardia, pupillary dilation, cutaneous vasoconstriction, and sweating. Even though the half-lives of both epinephrine and norepinephrine are on the order of a few minutes, circulating catecholamine concentrations in humans remain substantially elevated for 30–60 minutes after cessation of a single generalized seizure (Fig. 1). The norepinephrine elevation (12 times normal) is presumably do to activation of peripheral adrenergic neurons driven by brain stimulation and is of a level adequate to produce a direct vasoconstrictor effect. The epinephrine elevations (40 times normal) are due to neural activation of the adrenal medulla from generalized sympathetic stimulation (although hypoxia increases epinephrine concentrations as well), with levels adequate to have cardiovascular effects [2]. Furthermore, postictal epinephrine concentrations are at levels adequate to explain seizure-induced hyperglycemia [3]. During seizures in humans (Fig. 1) and in experimental animals, the increase in epinephrine is greater than that in norepinephrine.

2. Hyperthermia

Hyperthermia can result, at least in part, from muscle activity of the convulsion; however, some temperature elevation occurs during seizures during neuromuscular paralysis [4,5]. Fever was a feature of the postictal state in 27 of 93 hospitalized patients [6]. The onset of the temperature elevation could be delayed for a number of hours after seizure cessation. All but five patients had fevers above 38.3 °C (101 °F), and the mean fever duration was 21.8 hours. No infection causes were found. Observations in status support temperature elevation as proportional to seizure duration [7,8].

3. Postictal pleocytosis

Transient systemic hypertension, such as that induced by seizures, can result in blood–brain barrier opening. Particularly following multiple or prolonged seizures, postictal pleocytosis may occur in the spinal fluid. Such pleocytosis occurs in nearly 20% of patients in the postictal state after status. The maximum cell count is less than 80/mm^3, and the maximum cell count may not be seen until the day following the seizure [9]. Following single seizures in adults and children only 5% of patients are so affected [10,11].

4. Cardiovascular effects

Ictal effects on heart rate and blood pressure persist into the postictal period. Maximal postictal heart rate is similar to that at ictal onset, about

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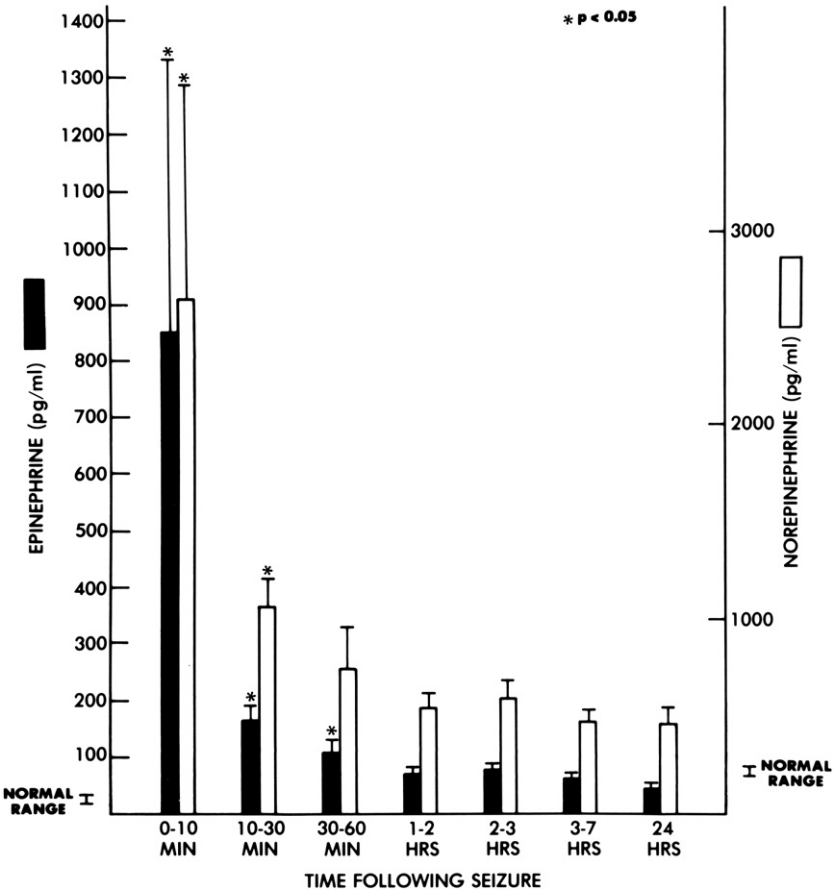

Fig. 1. Epinephrine and norepinephrine plasma concentrations following a single generalized tonic–clonic seizure in 17 patients. Modified and reproduced, with permission, from *Neurology* [2].

130% of baseline [12]. In a separate study, the mean ictal heart rate increase was 83/minute [13]. The magnitude of heart rate and blood pressure changes reflects the duration of the preceding seizure. Single pentylenetetrazol-induced seizures in humans result in increases of 82 and 42 mm Hg in mean systolic and diastolic pressures, respectively. Heart rate and blood pressure increases peak within a minute of seizure onset, paralleling elevations in plasma catecholamine concentrations. Tachycardia and hypertension then decline and return to baseline within 60 minutes, even though status epilepticus may be ongoing. The mechanism by which sympathetic activity and cardiovascular response diverge is uncertain, but desensitization of vascular adrenergic receptors has been suggested [14].

Catecholamine-induced cardiac decomposition resulting from contraction band necrosis has been reported postmortem after death from status epilepticus or subarachnoid hemorrhage [15,16]. These hypercontracted, transverse bands of myocardium are associated with calcium loading of cardiomyocytes and are incapable of contraction. They occur within the myocardium in the region of sympathetic endplates, thus supporting a role for norepinephrine release. Recently, two cases of transient, postictal "sympathetic cardiotoxicity" resulting in stunned myocardium were described in the postictal state following convulsive seizures [17]. Both patients had left ventricular dysfunction in a nonvascular distribution. Cardiovascular dysfunction and myocardial infarction following brief convulsive seizures have been reported as well [18].

5. Cardiac rate and rhythm disturbances

Increased heart rate occurs in most adults and children during generalized tonic–clonic (GTCSs), complex partial (CPSs), and subclinical seizures. The patterns of these heart rate changes were consistent in a given patient during recurrent seizures [19]. The degree of ictal tachycardia in the postictal period is greater for patients with GTCSs than for those with CPSs. After 30 minutes, heart rate remains above 100 bpm in GTCSs, but has normalized in CPSs. Bradycardia and asystole are rare but occur as well [20]. Video/EEG monitoring of 6825 patients recorded asystole in only 0.27% [21]. Bradyarrhythmias have been suggested to be associated with temporal and or frontal lobe seizures and may occur with or without ventilatory depression [22]. Permanent pacemaker placement has been instituted for ictal bradycardia or asystole, but a symptomatic recurrence is very rare [23]. Heart rate variability has been used as a measure of cardiac autonomic control. Impaired autonomic control of the heart results in decreased heart rate variability. In patients with cardiac disease, such decreased variability in heart rate is associated with an increased risk of arrhythmias [24]. Postictal heart rate oscillations have also been used as an indicator of autonomic instability. These high-frequency oscillations are believed to result from sympathetic activation in the setting of decreased vagal tone, a combination that may be arrhythmogenic [25].

Postictal abnormalities in heart rhythm and conduction occur in addition to abnormalities of heart rate. Although changes in both heart rate and heart rhythm begin during the seizure, the alterations persist well into the postictal period [26]. Injury indicators, ST segment elevation or depression, have been reported in approximately 40% of patients with epilepsy and in approximately 7% of individual seizures. These abnormalities were more common with generalized seizures [27–30]. Alterations of cardiac repolarization with associated arrhythmia risk are indicated by altered QT intervals. Abnormally prolonged repolarization, documented by QT prolongation, can be induced by catecholamine release, hypoxia, and hypercapnia and has