Title: CEREBRAL HYPOXIA DURING VENTRICULAR DYSRHYTHMIAS

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INTRODUCTION Current management of refractory ventricular dysrhythmias includes the insertion of an automatic internal cardioverter-defibrillator (AICD). During the implantation procedure, multiple episodes of ventricular tachycardia (VT) and ventricular fibrillation (VF) are produced, causing severe hypotension. This study was undertaken to assess the impact of this hypotension on the adequacy of cerebral perfusion as indicated by changes in the EEG.

METHODS Eleven patients scheduled for elective AICD implantation were studied. Patients were ASA 3 or 4, with major cardiac disease including prior MI, cardiomyopathy, CHF, hypertension, and recurrent dysrhythmias. Anesthetic management was at the individual anesthesiologist's discretion. Balanced anesthesia utilizing narcotics and either isoflurane or enflurane in oxygen was most commonly used. Vecuronium was used for muscle relaxation. Monitoring included arterial and central venous pressures, EKG, and a 6-channel parasagittal bipolar EEG montage. The EEG bandwidth was 1-35 Hz and data were digitized at 128 Hz. Power spectrum analysis was performed using standard techniques with 2-second epochs. Hemodynamic events were defined as periods when the systolic BP fell below the prior steady-state diastolic BP. EEG recordings were displayed in density-modulated (DSA) format with synchronous blood pressure tracings and examined for EEG changes typical of hypoxia (loss of high frequency activity and shift of power to the lower frequencies). Each event was analyzed for duration of hypotension, type and duration of arrhythmia, lowest BP achieved, presence of EEG change, and the temporal relationship between hemodynamic and electroencephalographic changes.

RESULTS There were 138 episodes of hypotension with an average duration of 36 seconds, 65% (101) of which produced EEG changes. Delay from the onset of arrhythmia to EEG change averaged 20 seconds for both VT and VF and did not differ between the dysrhythmias. Although there was no difference in mean BP between episodes of VT and VF longer than 20 seconds (23 mmHg), there was a significant difference between the incidence of EEG changes with VT and VF. Only 66% (20/30) of VT episodes showed EEG changes compared to 93% (66/71) of VF episodes (χ^2:$p < 0.01$). Excluding 3 events in which hypotension exceeded 1 minute and recovery was correspondingly prolonged, patients showed EEG evidence of recovery from hypoxic change within an average of 9 seconds of return to normotension. The difference between the delay in onset of EEG changes and the delay to recovery was highly significant (paired t-test: $p < 0.001$). Although pre- and post-op testing have not been performed, no patient has demonstrated gross neurologic damage.

DISCUSSION In this study, abrupt reduction of pressure and flow during arrhythmias commonly resulted in EEG changes of hypoxia. The delay seen from the onset of hypotension to EEG change averaged 20 seconds for VF and VT, confirming that under these anesthetic conditions the threshold for hypoxic EEG changes is comparable to previously published results[1]. For episodes of VF greater than 20 seconds, a condition which should result in no cardiac output, the incidence of hypoxic EEG changes was 93%, suggesting either a diagnostic specificity of the EKG of 93% or a diagnostic sensitivity of the EEG of 93%. Because spontaneous organization and disorganization occasionally made it difficult to classify the arrhythmia, it is likely that the sensitivity of the EEG for the identification of cerebral hypoxia is even higher.

The lower incidence of EEG changes during episodes of VT despite a mean duration and BP similar to episodes of VF suggests that during some episodes of VT there is sufficient cerebral blood flow to prevent the onset of cerebral hypoxia despite the similarity of perfusion pressures.

Recovery from short hypoxic episodes was more rapid than the onset, implying that the oxygen debt was repaid faster than it accumulated. Alternatively, EEG recovery may not be indicative of complete metabolic recovery. In contrast, the few lengthy hypotensive episodes required prolonged periods for recovery, suggesting the occurrence of more extensive dysfunction.

During the insertion of an AICD, the average patient is subject to multiple (10-15) hypotensive episodes many of which produce evidence of cerebral ischemia. While further study of this unusual population has much to offer in increasing our understanding of both cerebral metabolism and ischemia, the predictable behavior of the EEG during these episodes suggests that EEG monitoring is not required for routine management.

REFERENCES

1. Prior, PF: EEG monitoring and evoked potentials in brain ischemia. British Journal of Anaesthesia, 57:63-81, 1985.