Clinical relevance of alarms from bedside patient monitors.

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Introduction: The high rate of false positive or clinically not relevant alarms from patient monitoring devices is still a concern in critical care. Hypothesis: Even with modern bedside monitors a large percentage of alarms is clinically not relevant. Methods: Full disclosure monitoring data including all alarms, alarm settings and alarm silencing was collected in 38 medical ICU patients and annotated by an intensivist off-line using continuous video recordings to identify clinically relevant alarms. Clinically relevant alarms were defined as alarms resulting in or requiring actions by a caregiver in response to the alarm. Results: During 515 hours of observation 3682 alarms were recorded of which 68% were threshold alarms. While more than 54% of all alarms were technically correct the majority of alarms (82%) were clinically not relevant. There were significant differences between different physiologic variables. 44% of the alarms resulted from manipulation. Conclusions: This ongoing study confirms that even with modern monitoring systems most alarms are clinically not relevant. As the majority of alarms are simple threshold alarms, statistical methods may be suitable to help reduce the number of false positive alarms.

Efficacy of a less-invasive monitoring tool to correct postoperative tissue hypoxia in high-risk surgical patients.

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Introduction: Hypoperfusion increases subsequent risk of multiple system organ failure in high risk surgical patients (HRSP). Their resuscitation has traditionally been guided by vital signs, and only when there is hemodynamic instability invasive monitoring is required. Hypothesis: To evaluate the efficacy of a protocol based on a less-invasive monitoring tool to correct tissue hypoxia during the initial postoperative period. Methods: Randomized controlled trial with seventeen HRSP included. Patients in treatment group (TG) were monitored with Vigileo™ (Edwards Lifesciences, Irvine, CA, USA) and resuscitated during the first 12 postoperative hours using a protocol. Responders have, at the end of resuscitation (ER), Cardiac Index (CI) ≥3l/min/m2, central venous oxygen saturation (Svo2) ≥70% and Arterial Lactate (AL) ≤5 mmol/l. Control group (CG) was managed traditionally and were responders if Mean Arterial Pressure (MAP) ≥70 mmHg and Central Venous Pressure (CVP) between 8 to 12 mmHg at ER. Results: Nine patients were randomized to TG and eight to CG. The mean age in TG and CG was 71±14 and 67±12 years, respectively. The heart rate, MAP and CVP at baseline (BL) were similar in both groups: 82±21 vs 88±25 bpm (p=0.63); 10±2.8 vs 10±2.7 mmHg (p=0.63); 9±5.1 vs 13±5.3 mmHg (p=0.31), respectively. The variation of AL between BL and ER in TG was 3.4±1.7 vs 1.3±0.4 mmol/L (p=0.01) and 3.2±1.7 vs 2.9±1.5 mmol/L (p=0.74) in CG. The variation of Svo2 in TG was 68±26.3 vs 80±9±7.3% (p=0.01) and in CG, 69.5±17.2 vs 65±4±10 (p=0.29). CI in TG at BL was 4.0±2.4 and at ER was 3.8±1.4 L/min/m2 (p=0.53). Conclusions: An optimization protocol based on monitoring CI and Svo2 using a less-invasive monitoring tool contributed to improve tissue perfusion in this cohort of HRSP.

Can cocaine abuse cause bradycardia?

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Introduction: Drug abuse can have effects on various organ systems in the body, which can attribute to the toxic metabolites, adulterants or the drug by itself. Cocaine, derived from Erythroxylon coca plant, is an alkaloid and is abused in various forms. It is one of the most common causes for the Emergency department visits by the drug abusers. Hypothesis: Cocaine or its metabolites or the adulterants in it can cause bradycardia either due to vagomimetic effect or direct action on the nodes. Methods: Hundred patients with drug abuse and EKG recordings are retrospectively reviewed at random. Cocaine, opioids, benzodiazepines, THC, barbiturates are the drugs of abuse. Drug abuse is defined as urine toxicology positive for at least one of these drugs on more than one occasion and from the history. Patients’ age, race, sex, and serum electrolytes (sodium, potassium, calcium, bicarbonate) are also considered. Results: Of the 100 charts reviewed, 68.9% are males, with mean age of 42±9.2 years, 72.2% are Afro-American, 23.3% are Caucasian, and 4% are Hispanics. 69.9% patients are positive for Cocaine, of which 40% had either normal EKG or non-specific ST-T wave changes, 28% had bradycardia with or without block and only 3% had tachycardia / tachyarrhythmia. Serum electrolytes of all these patients were within normal limits. In our patients with cocaine abuse, bradycardia is the most common electrocardiographic abnormality observed (p=0.01). Conclusions: Norcocaine, the active metabolite of Cocaine, can cause cardiovascular abnormalities and EKG changes, by the inhibition of norepinephrine reuptake into the synaptic cleft by sympathetic neurons and release of catecholamine from the stores. However our analysis shows patients with cocaine abuse had bradycardia or normal EKGs. So, does cocaine/its metabolites/adulterants in the drug have a vagomimetic effect or is it that it directly affects the sinus node/blocked conduction? Further studies are warranted.