Basrah Journal Of Surgery I read for you

Bas J Surg, September, 17, 2011

MANAGEMENT OF CRISES DURING ANESTHESIA AND SURGERY. PART II: TACHYCARDIA & BRADYCARDIA

Salam N Asfar[®] & Jasim M Salman[#]

[®]MB, ChB, MSc, Professor of Anesthesiology, College of Medicine, University of Basrah, Basrah, Iraq. [#]MB, ChB, DA, Consultant Anesthesiologist, AlSadir Teaching Hospital, Basrah.

Tachycardia

Tachycardia is a common event during anaesthesia and is frequently associated with simultaneous changes in other monitored vital signs. In most cases, the cause is easily identified and the problem promptly resolved. However, in some the cause may be rare or obscure. Under such circumstances, attempting to initiate appropriate supportive therapy and to consider a large differential diagnosis in a comprehensive manner may lead to delays which can put a patient at risk¹.

The clinical significance of tachycardia will in part be determined by the blood pressure and cardiac rhythm of the patient at the time. This may vary from a hypotensive to a hypertensive crisis. Tachycardia and hypertension have been shown to be independently associated with a poor outcome after prolonged procedures. Hypotension may worsen into a cardiac arrest or the patient may be pulseless from the beginning³.

Table I : Causes of tachycardia expressed as percentage of total causes¹.

Causes	Tachycardia	Tachy- CA	Tachy- Hypo	Tachy- Normo	Tachy- Hyper
Allergy	11	17	21	5	0
Airway	4	4	4	3	6
Hypovolaemia	3	13	4	0	0
Regional anaesthesia	1	0	4	0	3
Drugs	33	25	19	48	37
Cardiopulmonary events	8	13	11	3	6
Autonomic reflexes	9	13	4	12	9
Sepsis	1	0	0	5	0
Surgical factors	6	8	6	3	6
Pre-existing condition	1	4	0	0	0
Central nervous	1	0	0	0	3
Metabolic	2	4	0	3	3
Monitoring	1	0	4	0	0
Other	2	0	6	0	0
Light anaesthesia	4	0	0	0	18
Hypercapnia	4	0	0	8	9
Not determined	8	0	17	10	0

Tachy-CA: tachycardia in association with cardiac arrest. Tachy-Hypo: tachycardia in association with hypotension. Tachy-Normo: tachycardia in association with normotension. Tachy-Hyper: tachycardia in association with hypertension.

Examples of causes:

Cardiopulmonary events:

Tachy-CA: pressure on pericardium & myocardial infarct.

Tachy-Hypo: left ventricular failure, tension pneumothorax, haemothorax & gas

embolism.

Tachy-Normo: myocardial infarct.

Tachy-Hyper: pulmonary oedema & fat embolism.

Surgical factors:

Tachy-CA: central venous cannula wire.

Tachy-Hypo: central venous cannula wire & hip cement or reaming.

Tachy-Normo: pneumoperitoneum.

Tachy-Hyper: hip cement. *Central nervous system*:

Tachy-Hyper, ruptured cerebral aneurysm.

Metabolic: Tachy-Normo: iatrogenic hyperthermia.

Tachy-Hyper: malignant hyperthermia.

Light anaesthesia:

Tachy-Hyper: pain, light anaesthesia & awareness.

Hypercapnia:

Tachy-Normo: hypoventilation, malfunction circle or soda lime & inhaled CO2.

Tachy-Hyper: hypoventilation & malfunction circle or soda lime.

Drugs causes tachycardia: IV anaesthetic agents, Benzodiazepines, Inhalational agents, Vaporiser, Suxamethonium, Local anaesthetic, Protamine, Vasopressors, Adrenaline, cocaine, Inotropes, Salbutamol & Atropine.

Management of tachycardia⁴:

*Do not hesitate to treat as cardiac arrest.

- *Remember to treat blood pressure changes. If real hypotension present; check that vaporizer is off and give crystalloid 10ml/kg bolus and repeat if necessary.
- * If sever tachycardia use synchronized cardioversion(start at 100J) and drugs.
- * If mild use appropriate antiarrhythmic drugs:

VT (100-200/min): Lidocaine 1mg/kg IV or amiodorone 5mg/kg.

AF(100-200/min no P waves, or flutter: P waves 250-300/min and ventricular rate 100-150): Digoxin 0.05 mg/kg IV or amiodorone 5mg/kg.

SVT(150-250/min): Adenosine 6-12 mg IV or titrated beta blocker: atenolol 1mg boluses.

Review and treat probable causes:

Hypovolemia:

Consider: Blood loss, dehydration, diuresis and sepsis.

Ensure: adequate IV access, fluid replacement, cross match and check haematocrit.

Drugs: consider; Induction and inhalation anaesthetic agents, Local anaesthetic toxicity,

Atropine, Vasopressors, Adrenaline & Cocaine.

Airway: See if there is hypoventilation or hypoxia.

Anaphylaxis.

Reflex stimulation:

Consider: Laryngoscopy, CVC insertion & surgical manipulation

Ensure: Adequate anesthesia.

Cardiopulmonary problems:

Consider and treat: Tension pneumothorax, hemothorax, temponade, embolism (gas, amniotic or thrombus), sepsis, myocardial irritability (from drugs, ischemia & electrolytes) and pulmonary oedema.

Bradycardia

The most common causes of bradycardia are:

Drug events

Problems with the airway or ventilation

Autonomic reflexes

Epidural or spinal anaesthesia

A recognized cause was not determined in many cases. In addition, all cases of bradycardia are compared with subgroups based on associated blood pressure and are also cross referenced with data relating to all cases of hypotension. The aim of this was to identify patterns which may indicate treatment when bradycardia coincided with abnormalities of blood pressure.

Drug events were reported as a likely cause in many reports. In many cases it was not possible to clearly distinguish the specific contribution of any particular drug when drugs were given in combination. In this event, each drug was described as a drug event. Overall, 129 drug events were nominated.

The most commonly cited drugs were inhalational agents, opiates, intravenous induction agents and suxamethonium. Enflurane and thiopentone were the most commonly described inhalational and intravenous agents, respectively. Fentanyl was the most commonly described opiate. Suxamethonium caused bradycardia after a repeat dose in 50% of cases. Drug errors and vaporiser incidents accounted, respectively, for nine and two of the total drug events described.

A drug error was defined as the inadvertent administration of an agent and included syringe and ampoule swaps and residual agents in intravenous lines. Vaporiser incidents were identified whenever the reporter was unaware that an agent was being administered at all or in the concentration detected. This included incorrect settings and administration of multiple agents due to the absence of interlock devices.

Problems with the airway or ventilation including desaturation on pulse oximetry, difficulty with ventilation, clinical cyanosis, or symptoms of dyspnoea. On some occasions no specific cause of the problem could be found with the airway; it is possible that the desaturation represented an artefact or true desaturation resulting from altered ventilation-pulmonary blood flow matching. Airway related events appear more likely to be associated with normotension & neuraxial blockade and appears more likely to be associated with hypotension. It also appears that cardiopulmonary events causing bradycardia are more likely than other causes to be associated with cardiac arrest.

Vagal reflexes were considered to have contributed to bradycardia in many. These reflexes were surgically induced, the majority of these were attributed to either vagal stimulation in the abdominal cavity or pelvis or the oculocardiac reflex. Anaesthetic causes included laryngoscopy and intravenous cannulation.

Surgical events included reaming, cementing or manipulating fractured hips, manipulation of wires or catheters in central veins, and pneumoperitoneum⁵.

Management of bradycardia includes: Increase monitoring, Atropine, Vasopressors, Adrenaline, Antidysrhythmic/pacing agents, cardiac pacing, External cardiac compression (ECC).

The conventional management of asystole includes early adrenaline. Atropine is recommended as a second line agent in refractory asystole or in the initial management of bradycardia associated with hypotension. Many reports describe several cases of cardiac arrest in which a successful outcome was achieved with the use of atropine instead of adrenaline. Atropine without ECC was used successfully in cases of reflex induced sinus arrest. If necessary, cardiac pacing should be instituted.

The significance of any decrease in heart rate will be determined by the associated blood pressure rather than the absolute heart rate. When bradycardia occurs and the patient is pulseless, then go to the cardiac arrest algorithm because early aggressive management is required. If the rhythm is reflex induced sinus arrest, then the most effective treatment is to cease the precipitating event.

If the patient is hypotensive, it is worth noting the causes of hypotension and considering the use of vasopressors and aggressive fluid therapy. This is equally important in the context of spinal, epidural⁶ or regional blockade (especially interscalene block⁷). This can be achieved by reviewing the hypotension sub-algorithm. However, it should remain a problem of rate until the diagnosis has been made because of the important role of atropine and tertiary management options which include transvenous pacing.

If the patient is hypertensive, then the problem should be managed according to the hypertension sub-algorithm. Another issue which has the potential to complicate the problem is whether the prevailing cardiac rhythm should be diagnosed early. Sinus and non-sinus rhythms were compared with respect to ASA grading, haemodynamic status at the time of diagnosis, management and outcome. This was done in an attempt to determine whether an early diagnosis of rhythm would be of value in the overall management of the problem.

Also the management of bradycardia as described in many of the reports was based upon the guidelines of the American Heart Association (AHA)⁸ & the International Committee on Resuscitation⁹. Although it is now not well recommended, examples included the use of calcium and isoprenaline, the omission of adrenaline when indicated, an excessive dose or inappropriate route of administration of atropine and DC cardioversion for asytole.

The most commonly used vasopressors are ephedrine, metaraminol, or a combination of the two.

External cardiac compression was specified to cardiovascular collapse. It was assumed to have been given whenever you consider the situation as "resuscitation" or "cardiac life support".

References

- 1- L M Watterson, R W Morris, J A Williamson, et al. Tachycardia; Crisis management during anaesthesia, www.qshc.com.
- 2- Reich DL, Bennett-Guerrero E, Bodian CA, et al. Intraoperative tachycardia and hypertension are independently associated with adverse outcome in noncardiac surgery of long duration. Anesth Analg 2002;95:273–7.
- 3- Runciman WB, Morris RW, Watterson LM, et al. Crisis management during anaesthesia: cardiac arrest. Qual Saf Health Care 2005:14:e14.
- 4- Australian Patient Safety Foundation. Crisis Management Manual: Adelaide: Australian Patient Safety Foundation, 1996:74pp. Available at http://www.apsf.net.au/anaesthesia.htm.
- 5-L M Watterson, R W Morris, R N Westhorpe, et al. Crisis management during anaesthesia: bradycardia. (http://www.qshc.com/cgi/content/full/14/3/e9).2002.
- 6- Lesser JB, Sanborn KV, Valskys R, et al. Severe bradycardia during spinal and epidural anesthesia recorded by an anesthesia information management system. Anesthesiology 2003;99:859–66.
- 7- Sia S, Sarro F, Lepri A, et al. The effect of exogenous epinephrine on the incidence of hypotensive/bradycardic events during shoulder surgery in the sitting position during interscalene block. Anesth Analg 2003;97:583–8.
- 8- Emergency Cardiac Care Committee and Subcommittees, American Heart Association. Guidelines for cardiopulmonary resuscitation and emergency cardiac care. Part III: Adult advanced cardiac life support. 1992 National Conference on Cardiopulmonary Resuscitation (CPR) and Emergency Cardiac Care (ECC). JAMA 1992;268:2199–241.
- 9-Kloeck W, Cummins RO, Chamberlain D, et al. The Universal Advanced Life Support Algorithm. An Advisory Statement from the Advanced Life Support Working Group of the International Committee on Resuscitation. Circulation 1997;95:2180–2.