Systemic blood pressure variations during anesthesia

Essay Submitted for partial fulfillment of master degree in anesthesia

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Contents

List of abbreviations

- **ABP:** an intraarterial catheter
- **ABP:** arterial blood pressure
- **ACC/AHA:** American College of Cardiology/American Heart Association.
- **ACE:** angiotensin converting enzyme
- **ACE:** angiotensine converting enzyme
- **ACEIs:** Angiotesin converting enzyme inhibitors
- **AGT:** intrarenal angiotensinogen
- **Ang II:** angiotensinogenII
- **ANP:**atrial natriuretic peptide
- **ARBs:** angiotensin receptor blockers
- **ASA:** American society of anesthiology
- **ATII:** Angiotensin II
- **ATLS:** Advanced Trauma Life Support
- **ATP:** Adenosine triphosphate
- **ATPase:** Adenosine triphosphatase
- **BMI:** body mass index
- **BP:** blood pressure
- **Ca^{+2}:** Calcium
- **CABG:** Coronary artery bypass graft
- **CBP:**cerebral blood pressure
- **CD:** collecting ducts
- **CI:** cardiac index
- **CNT:** connecting tubules
- **CO:**cardiac output
- **CO_2:** Carbon dioxide
- **CV:** cardiovascular
- **CVE:** cardiovascular events
- **DBP:**diastolic blood pressure
- **EACA:** epsilon aminocaproic acid
- **EC:** extracranial

- **ECG:**electrocardiogram
- **Echo-TDM:** echo-transesophageal Doppler monitoring
- **eg:** exempli gratia
- **EIT:**electrical impedance tomography
- **ESH/ESC:** European society of hypertension/ European society of cardiology
- **ESRD:**end-stage renal disease
- **$ETCO_2$:**end tidal Carbon dioxide
- **FHR:** fetal heart rate
- **HEBs:** hypotensive and bradycardic events
- **Hg:** mercury
- **HMW:** high molecular weight
- **HR:** heart rate
- **HTN:** hypertension
- **ICA:** internal carotid artery
- **ICH:** Intracranial hemorrhage
- **ICP:** intracranial pressure
- **ISBPB:** interscalene brachial plexus block
- **ISH:** isolated systolic hypertension
- **IV:** intravenous
- **JNC7:**Joint National Committee of Prevention, Detection, Evaluation, and Treatment of High Blood Pressure
- **kg:** kilogram
- **KKS:** Kallikrein-kinin systems
- **LTI:** laryngoscopy and intubattion
- **LVH:** left ventricular hypertrophy
- **MAP:** mean arterial blood pressure
- **mg:** milligram
- **min:** minute
- **mm:** millimetre
- **MR:** mineralocorticoid receptor
- **mRNA:** messenger riboneucliotide amine
- **N.B.:** nota bene

- **NADPH:** Nicotinamide adenine dinucleotide phosphate
- **NIBP:** Noninvasive blood pressure measurement
- **NIC:** Nicardipine
- **nm:** nanometer
- **NO:** nitric oxide
- **Nox:** NADPH oxidase
- **NPY:** neuropeptide Y
- **NSAIDs:** non-steroidal anti-inflammatory drugs
- **NTG:** nitroglycerine
- **O_2:** Oxygen
- **OH:** orthostatic hypotension
- **OEF:** Operation Enduring Freedom
- **OIF:** Office for Intellectual Freedom
- **OSA:** Obestructive sleep apnea
- **PAC:** pulmonary artery catheter
- **$PaCO_2$:** partial pressure of carbon dioxide
- **PACU:** Post Anaesthesia Care Unit
- **$PETCO_2$:**Partial end tidal CO_2
- **PIHP:** renal interstitial hydrostatic pressure
- **PLMA:** proseal laryngeal mask airway
- **PLR:** passive leg raising
- **PO:** by mouth
- **PVI:** pleth variability index
- **RAS:** renin-angiotensin system
- **RCT:** randomized controlled trials
- **RHTN:** Resistant hypertension
- **RND:** radical neck dissection
- **ROS:** Reactive oxygen species
- **RPP:** rate pressure product
- **RR:** Respiratory rate
- **RRT:**renal replacement therapy
- **SaO_2:**arterial blood oxygen saturation
- **SBP:** Systolic blood pressure

- **SC:** supratentorial craniotomy
- **SNP:** Sodium nitroprusside
- **SpO_2:** arterial saturation
- **SVV:** stroke volume variations
- **TCI:** Target-controlled infusion
- **TEE:** Trans-esophageal echocardiography
- **TH:** transphenoidal hypophysectomy
- **TIVA:** total intravenous anesthesia
- **TOD:** target organ damage
- **TXA:** tranexamic acid

Introduction

Mean arterial pressure is a product of cardiac output and systemic vascular resistance. Cardiac output depends on heart rate and stroke volume, and therefore anything that affects these indices will impact on the blood pressure. Similarly, systemic vascular resistance depends on several factors and will vary according to the indices will impact on the blood pressure degree of sympathetic tone and the effect of vasoactive drugs, including interventions such as central neuroaxial anesthesia. The blood pressure required to maintain adequate blood flow to vital organswill vary between patients **(Bryant and Bromhead, 2009).**

Systolic blood pressure (SBP) rises continuously with age, while diastolic blood pressure (DBP) reaches a plateau in the fifth or sixth decade, and may then decrease. Thus, systolic hypertension is more common in the elderly, and consequently an increase in pulse pressure is seen in older patients. Whereas it used to be thought that DBP is the most important determinant of outcome and the prime target for blood pressure control, current thinking is that systolic pressure hypertension, where the pulse pressure exceeds 65 mmHg, is the crucial issue. Consequently, systolic hypertension is now regarded as the principal target for blood pressure control in older patients. The severity of

hypertension is categorised into fairly well defined and accepted bands, as is illustrated in Table 1**(Micheal et al, 2011).**

Table 1.Classification of hypertension (Micheal et al, 2011).

Category	Systolic arterial blood pressure (SBP)		Diastolic arterial blood pressure (DBP)
Optimal	< 120	and	< 80
Normal	< 130	and	< 85
High normal	130-139	or	85-89
Hypertension			
Stage 1: Mild	140-159	or	90-99
Stage 2: Moderate	160-179	or	100-109
Stage 3: Severe	180-209	or	110-119
Stage 4: Very severe	> 210	or	> 120
Isolated systolic hypertension	> 140	and	< 90
Pulse pressure hypertension	> 80		

During induction of general anesthesia, patients with hypertension may exhibit significant increases in heart rate and blood pressure, though the agents used for this often cause hypotension. Since some antihypertensive agents are known to interact with anesthetic agents,care should be taken to determine when or if such antihypertensive agents are to be discontinued **(Momota et al, 2009).**

Emergence from general anesthesia and especially post-extubation phase are the stages associated with cardiovascular hyperdynamic status leading to increase in oxygen consumption, catecholamine release and pain. Post-operative pain can be derived from the anesthesia techniques used for the surgery, for instance, the back pain caused following spinal anesthesia or the surgical process solely. This phase lasting 15 to 5 minutes could frequently be accompanied by tachycardia and hypertension. Most patients however could endure this temporary encountered situation appropriately. On the other hand, patients having preoperative hypertension and cardiovascular and cerebrovascular diseases and patients with increased intracranial pressure (ICP) could be affected by severe cardiac and or cerebral complications. Therefore, it is of great importance to prevent postoperative and post intubation sympathetic excitations in high-risk patients as maintaining stability in the dynamic status reduces the mortality and morbidity rates in these patients **(Hossienzadeh et al, 2012).**

Aim of the work

The aim of this work is to provide an updated review of the role of the anesthesiologist to control the systemic blood pressure variations during anesthesia.

Chapter 1 : Physiology of blood pressure

I) Hypertension physiology

Many interacting physiological systems provide homeostatic regulation of arterial pressure, and derangements in any one of them can contribute to hypertension. While the nervous system provides regulatory inputs and stability to the blood pressure mechanisms, the primary responsibility for the long-term regulation of arterial pressure is vested in the kidneys' capability to integrate endocrine, neural, and hemodynamic inputs to maintain sodium balance and arterial blood pressure. Of the many mechanisms contributing to these alterations, the renin-angiotensin system (RAS) plays a most vital role in regulating both sodium balance and blood pressure through its pleotropic actions on multiple vascular, endocrine, and renal mechanisms **(Navar, 2010).**

Regulation of blood pressure

The control of blood pressure is complex and will be reviewed only briefly.

Neurogenic control

Postganglionic sympathetic nerves are localized to the adventitial- medial border of most arteries, arterioles, and veins throughout the body. Venules and capillaries, which lack smooth muscle cells, are not directly innervated by sympathetic nerves.

Norepinephrine released from the sympathetic nerve terminals binds to α_1- or α_2-adrenergic receptors located on vascular smooth muscle cells to increase intracellular Ca^{+2} either by causing release of Ca^{+2} by increasing flux through plasmalemmal Ca^{+2} channels. This rise in Ca^{+2} causes contraction of the smooth muscle via the activation of calmodulin-dependent myosin light chain kinase and the subsequent phosphorylation of myosin light chain, which is required for the activation of myosin Adenosine triphosphatase (ATPase) and binding of myosin to actin filaments. In vessels that contain more than one layer of smooth muscle, only the outermost cells receive sympathetic innervation. Smooth muscle cells of the inner portions of the medial layer contract due to the diffusion of norepinephrine and cell-to-cell communication mediated by gap junctions. In addition to norepinephrine, vascular sympathetic nerves also may contain neuropeptide Y (NPY) or Adenosine triphosphate(ATP), which can be released as cotransmitters. Both can produce constriction by activating vascular NPY Y1 receptors or purinergic P_2X receptors, respectively, and increasing intracellular Ca^{+2}**(Thomas, 2011).**

Renin-angiotensin system

Increases in circulating or local angiotensinogenII (Ang II) concentrations elicit a positive augmentation of intrarenal angiotensinogen (AGT) messenger riboneucliotide amine

(mRNA) and protein leading to increased secretion of AGT into the tubular fluid. Together with the sustained or increased tubular angiotensine converting enzyme (ACE) levels, the augmented AGT increases intratubular Ang II, which further augments sodium transport via stimulation of AT1 receptors. The augmented AGT production and secretion increase AGT delivered to the distal nephron segments, which can interact with renin and ACE produced by principal cells of connecting tubules (CNT) and collecting ducts (CD) cells to form more Ang II and stimulate distal transport activity. In a pathophysiologic environment, inappropriate stimulation of the intratubular RAS may be an important contributor to the development and maintenance of hypertension and associated renal injury **(Navar et al., 2011).**

Atrial natriuretic peptide

At physiological concentrations, atrial natriuretic peptide (ANP) is recognized to have multiple actions including those on kidney to increase water excretion, on vascular smooth muscle to cause vasodilatation, and on endothelium to increase vascular permeability. The actions of ANP on kidney, vascular smooth muscle and vascular endothelium are part of the physiological mechanisms to regulate plasma volume and arterial pressure. The ANP control over albumin permeability distinguishes it from other diuretics that result in fluid loss from both plasma and

interstitial spaces. One key mechanism whereby ANP effects preferential loss of fluid from the plasma volume appears to be that ANP- dependent increase in vascular permeability to plasma proteins, occurring at the same time that water is excreted by the kidney, reduces the tendency for albumin to concentrate in the plasma due to renal water loss from the plasma. Since albumin is by far the largest contributor to the plasma colloid osmotic pressure, the expected shift of water from the interstitial space into plasma, predicted from the Starling balance of capillary exchange when plasma proteins are concentrated, does not occur in the presence of the increased permeability **(Lin et al., 2011).**

Kallikrein-kinin systems (KKS)

Plasma-derived kallikreins release bradykinin from a high-molecular-weight form of kininogen (HMW Kininogen), which has an extremely short half-life. Bradykinin is the basic vasoactive peptide of the KKS involved in the regulation of blood pressure as well as flow. Bradykinin signalsvia, a constitutively expressed receptor in the cardiovascular system. Autocrine and paracrine activation stimulate the release of nitric oxide (NO) and prostaglandins (vascular smooth muscle relaxation). Reduced bradykinin generation results in hypertension, which may be associated with genetic defects or pregnancy. KKS components have a certain therapeutic potential in hypertension. The KKS and RAS are connected by the ACE, which degrades bradykinin and