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Noradrenaline versus glypressin for prevention of hypotension after deflation of tourniquet in knee arthroplasty

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Abstract
Hypotension, rapid heart rate, and an increased cardiac index are hemodynamic disturbances that can be triggered by a tourniquet's deflation. Patients with impaired cardiovascular function and the elderly are at increased risk due to these changes, even though they may not be noticeable to those with healthy cardiovascular systems.
Vasopressor medications work by reversing the effects of sympathetic blockage on the blood vessels. As a result of their actions, venous return and heart filling are preserved and vascular tone is restored. An effective method for managing spinal hypotension is the administration of vasopressors, either by bolus injections or continuous infusion.
Researchers are interested in studying the efficacy and safety profiles of norepinephrine and glypressin since they have gained increased attention as possible alternatives for the prevention and treatment of hypotensive episodes.

Keywords: Noradrenaline versus glypressin, hypotension, blood vessels, cardiovascular systems

Introduction
An artery and vein can be controlled for a set amount of time with the use of a tourniquet, which is a device that compresses or narrows an extremity [1].
When a tourniquet is deflated, it can cause changes in hemodynamics, such as low blood pressure, rapid heart rate, and an elevated cardiac index [2]. People whose cardiovascular systems are healthy may not notice these changes, but those with impaired cardiovascular function or who are elderly may be at danger [2, 3].
A growing body of research suggests that norepinephrine, a classic vasopressor, could be an effective alternative to other methods for treating or avoiding hypotension [4].
The catecholamine norepinephrine has a benzene ring with two hydroxyl groups in the meta-para position and an ethylamine side chain with a hydroxyl group attached in the benzylic position. In contrast to epinephrine, which has a methyl group connected to its nitrogen atom, norepinephrine has a hydrogen atom in its structural position [5]. Glypressin is another type of vasopressor that can be used to treat hypotension. It works by acting on the vasopressin system, which is an important physiological system for controlling the pressure in the blood vessels [6].

Tourniquet in Knee Arthroplasty
Surgery to repair or improve the function of a patient's lower or upper limb often makes use of tourniquets to keep the affected area from bleeding during the procedure. Misuse of the device, however, can cause long-term harm and functional loss to the limb. Therefore, in order to guarantee the safe application of tourniquets, it is crucial that personnel in the operating room have a thorough awareness of their physiological effects [7].

Types of tourniquets
Tourniquets come in a few different varieties, each suited for a specific purpose. There are emergency tourniquets, Surgical tourniquets, Non-inflatable tourniquets, and Finally, pneumatic tourniquets.

Components of pneumatic tourniquets

A pneumatic tourniquet isn't just a simple band. It's a system with several key parts. First, there's the inflatable cuff, which acts like a balloon around the limb. This cuff is filled with air from a compressed gas source. A pressure regulator controls how much air fills the cuff, and a pressure display shows how much pressure is being applied. Finally, connecting tubing links the cuff to the system that controls the air pressure.

Tourniquet deflation induced hypotension

Hypotension, caused by metabolic and lactic acidosis and hyperkalemia, may occur briefly after a tourniquet is deflated. Hyperkalemia has the ability to cause myocardial depression and, in severely ill or elderly patients, cardiac arrest after long lower limb surgeries. Tourniquet deflation can cause hemorrhage at the surgical site, but reactive vasodilation and increased microvascular permeability in the reperfused limb in the first hour are the main causes of hypovolemia, which can develop following tourniquet deflation [8].

In addition to reducing PaO2 and pH levels, limb occlusion causes metabolic changes in the ischemic limb, such as increased lactic acid, potassium, and PaCO2. There is a correlation between the length of time that these harmful metabolites are released into the systemic circulation and the severity of the pathophysiological alterations that occur. Importantly, after deflating the tourniquet, all of these alterations are completely reversible in within thirty minutes. Bilateral tourniquets increase the severity of metabolic disruptions [9].

An important function of the sympathetic nervous system is to modulate vascular tone, which in turn regulates arterial blood pressure (ABP). The role of this system in regulating ABP, however, varies from person to person. In light of this inter-individual heterogeneity, it is possible to attain the same ABP in people with low sympathetic nerve activity as in people with high activity. Different people react differently to consistent hemodynamic insults (such as spinal anesthesia or trimethaphan-induced ganglionic blockade) that dampen sympathetic nerve activity because of this diversity. A lower arterial blood pressure (ABP) after exposure to hemodynamic insults is more noticeable in patients with higher baseline sympathetic nerve activity. This suggests that ABP is drastically reduced with removal of tonic sympathetic support (hemodynamic insults) if ABP is strongly dependent on it [10, 11].

Due to redistribution of intravascular volume to the lower limb that was previously ischemic, bleeding, and vasodilation, ABP is reduced after tourniquet deflation (TD) in the context of total knee arthroplasty. Patients whose sympathetic tone was predominant before transdermal angioplasty (TD) may have reduced ABP after the procedure because the vasodilation is mediated by this tone reduction [11].

Constant blood pressure monitoring, hydration therapy, non-pharmacological approaches, and vasopressor administration are all part of hypotension care. The conventional method of volume restoration through fluid therapy, which can be given as a preload or co-load, typically involves crystalloids or colloids. Leg compression and positioning are two non-pharmacological approaches. Leg compression can be accomplished by bending the hips, using elastic bandages, or wearing stockings, while the Trendelenburg posture can enhance venous return to the heart [12]. Infusion or bolus administration of vasopressors is an effective way to treat spinal hypotension [12, 13].

Norepinephrine

Biochemical mechanisms

Biosynthesis

Through an enzyme cascade, the amino acid tyrosine is converted into norepinephrine in the sympathetic nervous system's postganglionic neurons and the adrenal medulla. The process of converting tyrosine to dopamine mainly takes place in the cytoplasm, whereas the conversion of dopamine to norepinephrine by dopamine β-monooxygenase mainly takes place within neurotransmitter vesicles [14]. Only the alpha1 and beta1 receptors are strongly activated by norepinephrine, whereas the beta2 and alpha2 receptors show only weak activation. It is possible that the beta1 effects may increase cardiac output at dosages below 2 mcg/min. Alpha1 effects may be more pronounced, nevertheless, at doses higher than 3 mcg/min. When the alpha1 receptors are activated to a higher degree, the blood vessels constrict and the systemic vascular resistance increases depending on the dose. Generally speaking, venous to arterial activity ratios are around equal [15]. However, the increase in heart rate is temporary since the baroreceptor response to the rise in blood pressure, along with heightened vagal tone, eventually leads to a prolonged decrease in heart rate [16]. The alpha receptors are the ones that norepinephrine activates most strongly [17].

The sympathetic effects of norepinephrine

Numerous sympathetic actions are exerted by norepinephrine throughout the body. It helps the eyes in two ways: first, by increasing tear production, which improves ocular lubrication; second, by contracting the iris dilator, which dilates the pupils [18]. From a cardiovascular perspective, norepinephrine raises blood pressure by narrowing artery blood vessels and stimulating an increase in cardiac output [19, 20]. The action of norepinephrine on the kidneys is to increase salt retention by stimulating the secretion of renin [21]. Norepinephrine stimulates the liver to produce more glucose, which the body uses for energy in most situations [21]. This glucose can be produced by glycogenolysis following a meal or through gluconeogenesis in the absence of recent food consumption. Also, skeletal muscles enhance glucose absorption when norepinephrine is present [21]. Due to its inhibitory action on the enteric nervous system, norepinephrine reduces digestive activity in the gastrointestinal tract by lowering gastrointestinal motility, blood flow, and production of digestive chemicals [22].

Administration

Continuous infusion is the usual method of administering norepinephrine because of its comparatively short half-life of 2.5 minutes. To prevent oxidation and the consequent loss of medication efficacy, the FDA suggests diluting the concentrated norepinephrine in solutions including dextrose before infusion. Use of saline alone as a diluent is strongly discouraged by the FDA [23]. One typical method is to start the infusion at a rate of 8–12 mcg/min and adjust the dosage until the appropriate pressure is reached. Two to four micrograms (mcg) per minute is the typical maintenance dose [24].

Adverse Effects

When alpha1 receptors are activated, norepinephrine's most common side effects occur. In other words, end-organ perfusion can be reduced due to severe vasoconstriction [25].

The baroreceptor reflex can elicit reflex bradycardia as a result of vasoconstriction due to alpha1 activation, and beta1 activity is usually not enough to compensate for this. As a result, even with beta1 agonism, cardiac output could go down or stay the same. Myocardial oxygen demand rises as a result of an increase in workload due to an increase in afterload caused by an increase in systemic vascular resistance [26].

Norepinephrine infusions should preferably be administered by central venous catheters or large-bore peripheral intravenous catheters. For optimal peripheral infusion, it is recommended to use an antecubital vein in the upper extremity; this minimizes the risk of ischemia caused by extravasation. Because occlusive vascular disorders are more common in the lower limbs, it is best to avoid putting pressure on these veins whenever feasible. Significant ischemia and necrosis might result from extravasation into nearby tissues. Stopping the infusion promptly is necessary in the event that extravasation is suspected. Any of the injected medication should be drawn back in an effort to eliminate it. It is recommended to restart the infusion at a different site, preferably in a separate extremity, if it is necessary to continue. The next step is to inject phentolamine into the surrounding region [27].

Monitoring

Whether invasive or non-invasive measurement techniques are available, it is crucial to closely monitor blood pressure if vasopressors like norepinephrine are administered. During initial titration, it is recommended to get values every 2 to 3 minutes using non-invasive measures. After determining the optimum maintenance dose, values should be obtained at least every 5 minutes [28].

Glypressin

Enzymes break down the long-acting inert analogue glypressin (also known as triglycylvasopressin or terlipressin) into the active form of vasopressin, lysine

Physiological effects of vasopressin

V1 receptors mediate the direct systemic vasoconstrictive effects of vasopressin. As a result of its involvement with renal V2 receptors, it is essential for osmoregulation and normovolemia maintenance. Vasopressin promotes the secretion of adrenocorticotropic hormone (ACTH), which in turn aids in temperature regulation, memory, sleep, and hemostasis. Vasopressin mainly regulates water balance under normal physiological circumstances. When the plasma osmolality or volume drops too low, the posterior pituitary secretes it [29].

When used at physiological concentrations and under typical physiological conditions, vasopressin has little to no effect on the vascular control of blood pressure. To induce a considerable increase in mean arterial blood pressure in humans, plasma vasopressin concentrations of about 50 $pg.ml^{-1}$ must be reached beforehand [30].

Plasma vasopressin levels rise during hypotension due to hypovolemia, which is essential for maintaining perfusion pressure. In animals that have their autonomic nervous system intact, vasopressin shows mild vasopressor characteristics. The heart rate-arterial pressure baroreflex curve is shifted to the left by its action on brain V1 receptors [31]. In addition to its effects on the skin, skeletal muscle, fat, pancreas, and thyroid, vasopressin is a powerful vasoconstrictor in other tissues as well. In comparison to catecholamines, it has a milder effect on vasoconstriction in the brain and coronary arteries. Also, the vasculature is made more sensitive to norepinephrine by vasopressin [32].

Adverse effects of vasopressin

Several side effects are linked to vasopressin administration. It is recommended against peripheral administration of low-dose vasopressin infusions due to the serious skin necrosis that can occur after extravasation of this medication in cases of catecholamine-resistant septic shock [33]. Vasopressin raises the risk of thrombosis by activating endothelial cells' V2 receptors, which in turn produce endothelial von Willebrand factor, which improves platelet aggregation [34]. Hyponatremia, anaphylaxis, bronchospasm, urticaria, and gastrointestinal ischemia are among the other side effects linked to vasopressin [35]. One study found that patients administered terlipressin had a significantly lower oxygen delivery index compared to those given norepinephrine [36]. This suggests that vasopressin may reduce cardiac index and heart rate, which in turn may affect oxygen delivery and utilization. The therapeutic relevance of the additional finding—an increase in pulmonary vascular resistance—is unknown at this time [36]. Potential side effects of vasopressin include abdominal cramps and heart arrhythmias [37]. The development of hypokalemia in cirrhotic individuals was suggested to be caused by secondary hyperaldosteronism with renal potassium wasting, according to one study of a small number of patients who took terlipressin [38]. Reduced serum potassium concentrations due to intracellular shift may also be expected as a result of acidosis correction in septic shock patients. Terlipressin has not been associated with any major medication interactions [39].

Terlipressin

A synthetic version of vasopressin, terlipressin is also known as tri-glycyl-8-lysine vasopressin. Lysine vasopressin (LVP) is a 12-amino acid peptide that differs from arginine vasopressin (AVP) due to the substitution of lysine for arginine at position 8. Terlipressin is a prodrug of LVP that is converted to LVP in the bloodstream when endothelial peptidases break the N-triglycyl residue. After the glycyl residues are cut, the active metabolite LVP is released gradually over several hours, while terlipressin is removed from circulation at an average rate of 24 minutes [30].

Pharmacodynamics of Terlipressin

Terlipressin, when given externally, activates vasopressinergic V1 and V2 receptors through intrinsic action, leading to the release of lysine-vasopressin. Simplified form of the signal transduction diagram resulting from activation of vascular V1 and renal V2 receptors. Terlipressin has a more selective V1 agonistic action (V1:V2 ratio = 2.2:1), in contrast to arginine vasopressin, which has a V1:V2 ratio of 1:1 [40].

Side effects

Ischemic side effects are linked to terlipressin because of its

action mechanism. But using a continuous infusion instead of bolus doses could reduce these negative effects [41-43]. The most recent RCT, the CONFIRM research, found one unanticipated side effect. Compared to the placebo group, terlipressin patients were more likely to experience respiratory failure [44] The majority of these cases occurred in patients with grade 3 acute-on-chronic liver failure (ACLF) according to the EASL-Chronic Liver Failure Consortium. Thirty percent of the terlipressin group and none of the placebo group experienced respiratory failure. The volume expansion caused by albumin and the cardio-suppressive actions of terlipressin are thought to be the culprits behind this phenomena.

Several pretreatment characteristics increase the likelihood of respiratory failure: a high international normalized ratio (INR), a low oxygen saturation of less than 90% as determined by pulse oximetry, and a high mean arterial blood pressure. Hence, terlipressin should only be started with great caution in patients with grade 3 acute cardiogenic shock (ACLF) who also have renal failure. Patients on terlipressin should be closely watched for signs of respiratory failure [45].

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