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Inotropic drugs and their uses in critical care

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ABSTRACT

The aim of this article was to provide a resource for critical care nurses wishing to further their understanding of inotropic drugs used in critical care. The physiology of cardiac output and blood pressure is examined along with an explanation of adrenergic receptors acted on by inotropes. Some common indications for inotropic therapy are discussed, along with essential patient monitoring and dose calculations to ensure safe therapeutic ranges are observed. Some of the most commonly used positive inotropes used in critical care environments are individually explored, providing indications and some of the latest research relating to their uses. Frequently, observed side effects of individual inotropes are also offered, enabling the nurse to maintain patient safety when administering these potent drugs. Some major nursing and professional issues related to inotrope therapy and medicine administration are discussed, as well as some recommended practices in renewing infusions.

Key words: Blood pressure • Cardiac output • Dose range • Inotropes

INTRODUCTION

The use of positive inotropes within the critical care setting is very common, their pharmacodynamics are often utilized to treat haemodynamic compromise for a variety of underlying reasons. From clinical experience, the nursing care of inotropes can be a daunting prospect for the inexperienced critical care nurse, the acquisition of knowledge of the theory relating to inotropes can have a significant impact on the nurses confidence to care for patients receiving inotropic therapy.

The aim of this article was to provide a resource for critical care nurses wishing to further their understanding of inotropic drugs used in critical care. It will provide fundamental information on the physiology of cardiac output (CO) and blood pressure (BP) control. An aspect of physiology that is essential in the understanding of the pharmacodynamics of inotropes. Therapeutic dose ranges will be provided along with the potential side effects of inotropes. Some of these effects will have nursing implications, encompassing practice and professionalism which will be explored, along with practical recommendations for inotrope infusion.

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The need for critical care nurses to have an in-depth understanding of inotropes is vital if safe and effective care is to be delivered. These highly potent drugs can result in catastrophic events for patients if staff are inadequately educated about their use. As mentioned it is important to have an understanding of the physiology surrounding CO and BP, the following section will address this area.

CO AND BP

CO describes the total volume of blood ejected by the heart every minute, normally approximately 5 L/min (Levick 2010). The regulation of CO is dependent on two factors: heart rate (HR) and stroke volume (SV).

SV signifies the volume of blood ejected by the left ventricle during systole, normally 70 mL (Hall 2011). Thus CO can be expressed in the following way:

$$CO = HR \times SV$$

$$CO = 75 \text{ bpm} \times 70 \text{ mL} = 5{\cdot}25 \text{ L/min}$$

SV is influenced by preload and myocardial contractility. Preload is essentially the venous filling of the right or left ventricle, this filling volume must be constant in order for adequate myocardial muscle fibre stretch to occur. The degree of stretch experienced by the myocardial muscle will influence the force of contraction; this concept is detailed in Frank-Starling's law (Levick 2010). In a situation of haemorrhage, preload is reduced because of the diminished volume under filling the heart, and limited stretching of

myocardial muscle fibres reducing the contractile force, leading to low CO. However, contractility can also be affected by cardiovascular disease such as chronic cardiac failure or arrhythmias – atrial fibrillation being one classic example of varying contractility where preload varies from beat to beat and abnormal nervous stimulation varies myocardial contraction (Katz 2011).

HR is regulated by the vasomotor centre located in the medulla. When the HR decreases the vasomotor centre sends impulses along sympathetic nerve fibres to the heart, where they secrete noradrenaline simulating an increase in HR (Hall 2011; Katz 2011). Conversely when HR increases, the parasympathetic autonomic nervous system (ANS) stimulates a decrease in HR via the vagus nerve, where it stimulates vagal cardiac fibres causing hyperpolarization leading to a longer threshold time, thus reducing HR (Levick 2010).

Increased HR can also be achieved under hormonal influences, such as the release of adrenaline from the adrenal gland by sympathetic stimulation, which will also cause vasoconstriction and increased myocardial contractility (Morrill 2000).

The regulation of BP is dependant on CO and systemic vascular resistance (SVR), which can also be expressed in the manner of an equation:

$$BP = CO \times SVR$$

SVR refers to the resistance experienced by the blood as it is ejected from the heart into the vascular system; this is influenced by the dynamic process that manipulates the diameter of the arteries and arterioles (Katz 2011). When their diameter is reduced it is termed vasoconstriction, while conversely increasing their diameter is described as vasodilation (Levick 2010). SVR is important in cardiac function in relation to afterload. This is the amount of force required by the left ventricle to overcome diastolic pressure and eject blood into the aorta (Hall 2011). An increased SVR will increase diastolic pressure therefore afterload, meaning a greater degree of force will be required to eject blood. The opposite would be true in vasodilation, where afterload is reduced.

Adrenergic receptors

Dynamic changes in HR, SVR and myocardial contractile state are controlled by various adrenergic receptors within the cardiovascular system, these are α, β_1, β_2 and dopamine (DA_1) receptors (Katz 2011). Inotropic drugs are either receptor agonists, where they enhance the actions of the receptor, or antagonist, where they dampen the receptor's actions (Rang *et al*. 2012). It must be noted, however, that these receptors can become desensitized (also termed as 'down-regulation') to the effects of inotropic drugs during prolonged infusion. This occurs as a result of high concentrations of the inotrope saturating the available receptors, reducing the number of available receptor binding sites (Deupree *et al*. 2002). This can be observed in the clinical setting, with the patient requiring higher doses of a drug to achieve the desired effect without a change in clinical condition. The individual receptor effects on the cardiovascular system and their locations are detailed in Table 1.

Table 1 Adrenergic receptor sites

Receptor	Actions	Locations
α	↑SVR	Coronary arterioles
	↑Blood pressure	Vascular smooth muscle
	↓Insulin release	
$\beta 1$	↑Heart rate	Sinoatrial node
	↑Contractility	Myocardium
$\beta 2$	↑Contractility	Coronary arterioles
	↓SVR	Bronchial smooth muscle
		Atrioventricular node
		Vascular smooth muscle
DA_1	Vasodilation	Kidney
		Heart

SVR, systemic vascular resistance.
Adapted from Adam and Osborne (2005), Katz (2011), Levick (2010), Morton and Fontaine (2009).

INDICATIONS

Inotropes are generally used to support the haemodynamics of patients in shock. Before inotropes are commenced the patient should receive fluid resuscitation to ensure optimal response to the drug (Palmer and Pennefather 2009). In the case of hypovolaemia, treatment should always be targeted fluid resuscitation to improve BP and HR, with the primary objective being the repair of any haemorrhage source. However, inotropes can be used to support this process, providing fluid resuscitation is ongoing when time is required to identify a bleeding point.

Although septic patents have not essentially lost fluid, their vascular space has increased because of vasodilation, this increased space must be filled to enable effective inotropic treatment (Raoof 2009). They will also experience capillary leak, meaning fluid will move from the vascular space into the extracellular space through damaged capillary walls. The systemic vasodilation experienced in septicaemia is a result of activated endothelial cells releasing vasoactive mediators (nitric oxide) (Porth and Matfin 2009), this is further compounded by the fact that acidosis associated with

septicaemia decreases myocardial contractility. This is due to the excessive circulating hydrogen ions binding to myosin cross-bridges in the myocardium, inhibiting calcium binding thus reducing contractility (Devlin 2011). Inotropic therapy is usually aimed at vasoconstriction and increasing myocardial contractility that is usually depressed in the presence of this acidotic biochemistry.

Patients in cardiogenic shock on the other hand exhibit low BP and CO because of compromised myocardial contraction and HR, this can be due to myocardial infarction, valve rupture, tamponade or myocarditis (Katz 2011). In these cases fluid should be given cautiously, owing to a risk of fluid overload due to the hearts ineffective pumping abilities. Essentially, the aim of inotropic therapy was to improve myocardial contractility, without adversely increasing myocardial oxygen demand.

MONITORING AND ADMINISTRATION

All the factors within the two equations above are manipulated by inotropes to maintain BP and CO within specified limits. As the result of this action and the potency of these drugs, electrocardiogram (ECG) and invasive BP monitoring must be in place before inotropic infusions are commenced. Advanced cardiovascular monitoring devices such as pulse-induced contour cardiac output (PiCCO) or oesophageal Doppler can be utilized to monitor CO, SVR and intravascular fluid status, enabling detailed and rapid monitoring of cardiovascular condition (Cottis *et al*. 2003). In addition to this, electrolytes should be regularly monitored, particularly serum potassium levels, as β-adrenergic receptor stimulation can cause an intracellular shift of potassium, resulting in hypokalaemia that can lead to arrhythmias (Hall 2011).

It is considered good practice to infuse these drugs via a central venous catheter (CVC) diluted in 5% dextrose or 0·9% sodium chloride using a high risk infusion device (Adam and Osborne 2005). Inotrope infusions are usually goal directed to achieve a specified CO or BP, thus enabling the nurse to titrate the rate to maintain desired parameters. However, the dosage is weight dependent and these should be calculated, as inotropes have a therapeutic dose range (Table 2). Dosage calculations can be carried out as follows:

$$\begin{aligned}\text{Dosage (mcg/kg/min)} = {} & \text{drug (mcg)} \div \text{dilutant (mL)} \\ & \div \text{minutes (60 min)} \\ & \div \text{patient weight (kg)} \\ & \times \text{infusion rate (mL/min)}\end{aligned}$$

For example, an 82 kg patient receiving noradrenaline 4 mg, diluted to 50 mL at 4 mL/h would be:

$$\begin{aligned}4\text{ mg} \times 1000 &= 4000\text{ mcg} \div 50\text{ mL} \div 60\text{ min} \\ &\div 82\text{ kg} \times 4\text{mL/h} \\ &= 0{\cdot}06\text{ mcg/kg/min}\end{aligned}$$

If a patient required dosages above the therapeutic range to achieve specified parameters, the appropriateness of continuing treatment or likelihood of secondary deterioration should be explored.

INOTROPES

Inotropes fall into two broad classifications, those with a positive inotropic effect which increase BP and CO, and negative inotropes which have the opposite effect. In both instances, most inotropes work by influencing intracellular calcium in the myocardium (Palmer and Pennefather 2009). As previously noted, this article will only discuss the positive inotropes commonly used in critical care (summarized in Table 2).

Noradrenaline

Noradrenaline is a naturally occurring hormone that is secreted from the adrenal medulla following stimulation from the ANS (Karch 2010). It primarily acts on α receptors resulting in an increase in SVR, resulting in increased BP. This elevation in SVR causes an increase in afterload, leading to a decrease in CO (Adam and Osborne 2005).

Noradrenaline is indicated in cases of low SVR, primarily septic shock. Noradrenaline's vasocontrictive properties are essential in treating this aspect of septicaemia, with research indicating that early infusion in conjunction with fluid resuscitation improves perfusion and reduces the required effective dose of noradrenaline (Montemont *et al*. 2007). However, noradrenaline is not only used to treat sepsis, it can also be utilized to maintain cerebral perfusion pressure (CPP) in head injured patients, this is essential to maintain blood flow to the brain, as these patients can suffer hypotension due to cerebral events or spinal cord injuries. Thus noradrenaline can be started before intracranial pressure (ICP) monitoring being established in order for CPP to be maintained. Failure to achieve this could lead to secondary ischaemic brain injury (Selladurai and Reilly 2007; Tsang and Whitfield, in press). CPP is calculated by subtracting ICP from mean arterial pressure (MAP) (ICP–MAP = CPP). This equation should be used to ascertain the MAP required, enabling titration of the infusion to achieve the desired CPP.

Table 2 Selected inotropes dilutions and actions

Drug	Dilution	Dose range	Receptor	Effects
Noradrenaline	0·9% sodium chloride 5% dextrose	0·01–1 mcg/kg/min	α	↑SVR
Adrenaline	0·9% sodium chloride	1 mg intravenous in cardiac arrest	$\beta 1$ $\beta 2$ α	↑Contractility ↑SVR ↑HR
Dobutamine	0·9% sodium chloride 5% dextrose	2·5–20 mcg/kg/min	$\beta 1$ ++ $\beta 2$ +	↑Contractility ↓SVR ↑HR
Dopexamine	0·9% sodium chloride 5% dextrose	0·5–6 mcg/kg/min	$\beta 1$ $\beta 2$	↑Contractility ↓SVR ↑HR
Dopamine	0·9% sodium chloride 5% dextrose	1–5 mcg/kg/min 5–10 mcg/kg/min >10 mcg/kg/min	DA_1 $\beta 1$ α	↓Renal SVR ↑Contractility ↑HR ↑SVR
Enoximone	0·9% sodium chloride Water for injection	Bolus: 0·5–1·5 mg/kg over 10–30 min Infusion: 5–10 mcg/kg/min	Phosphodiasterase inhibitor	↑Contractility ↔HR ↓SVR

HR, heart rate; SVR, systemic vascular resistance.
Adapted from Palmer and Pennefather (2009), Elliott (2006), British National Formulary (2011), Morrill (2000).

As with all drugs, negative side effects must be considered and nursing care should aim to minimize harm to the patient from these effects. One such effect is the reduction in insulin secretion during noradrenaline infusion, posing a significant risk of elevated blood glucose (Adam and Osborne 2005). It is considered good practice to regularly monitor blood glucose, as research has indicated close blood glucose control is linked to more favourable outcomes for septicaemic patients (Van den Berghe *et al*. 2001). However, a larger international study involving 6104 intensive care unit patients concluded that tight glycaemic control increases patient mortality, regardless of the underlying reason for admission. Suggesting that normal glycaemic control (maintaining blood glucose <10 mmol/L) should be the target of treatment (Finfer *et al*. 2009). Haemodynamic side effects of noradrenaline infusion include hypertension, bradychardia and arrhythmias (British National Formulary 2011), emphasizing the need for invasive BP monitoring and ECG. The risks of these effects occurring increase with dosage, with the normal dose range for noradrenaline is 0·01–1 mcg/kg/min (Waldmann *et al*. 2008).

Adrenaline

This endogenous catecholamine is an α, $\beta 1$ and $\beta 2$ agonist (Elliott 2006), resulting in increased SVR, myocardial contractility and vasodilation, respectively. Low doses of adrenaline have greater β effects with additional α effects observed at higher doses (Adam and Osborne 2005). The high numbers of $\beta 2$ receptors in myocardium and skeletal muscle cause the vasodilation associated with adrenaline, its vasoconstrictive properties on the other hand are exerted on tissues with a prevalence of α receptors such as the intestines and skin (Levick 2010). This vasoconstriction shunts blood from these tissues, enhancing circulation to essential organs.

Adrenaline is a well-established drug of choice in the treatment of anaphylaxis (Morrill 2000) due to its rapid onset of action and the ability to administer the drug intramuscularly or intravenously. Adrenaline is also established in the treatment of cardiac arrest, as it increases coronary perfusion during cardiopulmonary resuscitation (CPR) and supports poor CO by increasing HR and myocardial contraction (Resuscitation Council UK 2011). Its bronchodilatory properties also make it an useful addition in the treatment of acute life threatening asthma (Adam and Osborne 2005).

Although adrenaline can be given peripherally in an emergency, it is good practice to administer infusions via a CVC, titrating to achieve the desired effect as there is no recommended maximum dose (Adam and Osborne 2005) (Table 2). Previously tracheal administration of adrenaline during cardiac arrest was recommended when intravenous access was difficult, this route has been shown to reduce BP due to $\beta 2$ effects, and therefore is no longer recommended in the cardiac arrest situation (Resuscitation Council UK 2011).

Close observation should be exercised as adrenaline can cause arrhythmias and hypertension. Furthermore, the increased myocardial workload induced by adrenaline can also produce ischaemic pain because of mixed venous oxygen saturation (SVO_2) and supply mismatch (Morrill 2000); therefore, the nurse should remain vigilant of such occurrences. As with noradrenaline, adrenaline depresses insulin production, thus blood glucose monitoring control must be undertaken to prevent hyperglycaemia (Elliott 2006).

Dobutamine

Dobutamine is a drug widely used for the treatment of low CO states. It has a strong agonist effect on β1 receptors within the myocardium, increasing contractility and HR, with a somewhat milder effect on β2 causing vasodilation, reducing afterload (Aitkenhead *et al*. 2001). These effects increase CO and perfusion to vital organs, essential in cases of cardiogenic shock where ineffective contractility can significantly compromise perfusion (Adam and Osborne 2005). However, there is debate regarding the usefulness of dobutamine in cardiac patients. Delaney *et al*. (2010) conducted a literature review of randomized control trials, which suggested that heart failure patients receiving Levosimendan (a recently developed calcium sensitizing drug that increases contractility and vasodilation mainly used in the USA) experienced better survival rates than patients who were treated with Dobutamine. Therefore, research into the benefits of dobutamine is ongoing with emergence of new drug therapies. Dobutamine is also indicated in the treatment of septic shock to treat poor myocardial contractility via β1 agonism. However, its β2 vasodilatory properties can pose a problem in sepsis, where CO and SVR are reduced. This can be resolved by incorporating noradrenaline into the treatment regime to enable an increase in SVR, while dobutamine treats the diminished contractility.

Dobutamine can be infused via a peripheral line if required, but the central route is preferable to ensure rapid effects. Dose ranges are 2·5–20 mcg/kg/min, with the incidence of undesirable effects seen at higher doses (Waldmann *et al*. 2008). These include hypotension because of vasodilation and increased myocardial oxygen demand due to its β1 effects (Adam and Osborne 2005). Therefore, those patients with coronary artery disease must be closely observed during infusion for tachyarrhythmias, and ischaemic pain particularly at higher doses (Palmer and Pennefather 2009).

Dopexamine

This synthetic DA_1 analogue has β2 agonist effect resulting in arterial vasodilation resulting in reduced afterload. Coupled with dopexamine's β1 effects that increases myocardial contractility and HR, an improvement in CO can be achieved (Palmer and Pennefather 2009). Dose range for dopexamine is 0·5–6 mcg/kg/min (Waldmann *et al*. 2008). However at higher doses, the increased HR is at the expense of greater myocardial oxygen demand, making its use in patients with ischaemic heart disease problematic (Elliott 2006).

Dopexamine is primarily indicated in the treatment of chronic heart failure and cardiac surgery-related heart failure (British National Formulary 2011). There are also merits to its use in treating renal and gut hypoperfusion, due to its vasodilatory properties in improving perfusion of arterial beds in these locations (Adam and Osborne 2005). However, the effectiveness of Dopexamine in this area has been questioned by Probst *et al*. (2010) who conducted a randomized control trial on 30 trauma patients. Fifteen patients were given dopexamine and the remaining 15 DA_1, it was concluded that dopexamine increased the inflammatory response, serum lactate was elevated and overall increased patient duration on intensive care. However, the small sample size restricted to patients with blunt force trauma limits the applicability of these results to the wider critical care patient population. As Schmoelz *et al*. (2006) concluded there is no difference in organ perfusion between patients given dopexamine or DA_1. This study sample included 61 patients with septic shock, demonstrating the need for differing approaches depending on the underlying cause of admission.

Dopamine

This endogenous catecholamine acts directly on α, β and DA_1 receptors, with its effects being dependent on dose. At low doses (<5 mcg/kg/min), it has effects on DA receptors in the mesenteric, renal and coronary beds resulting in vasodilation (Waldmann *et al*. 2008). This low dose is sometimes referred to as 'renal dose' DA_1 which has caused a long standing debate as to its effectiveness within critical care in preventing or minimizing renal failure (Dunning *et al*. 2004; Schenarts *et al*. 2006). Mid range doses of 5–10 mcg/kg/min result in β1 stimulation, causing increased cardiac contractility and HR leading to an increase in BP (Raoof 2009). Higher doses (>10 mcg/kg/min) result in α effects causing an increase in SVR and therefore higher BP (Palmer and Pennefather 2009). DA_1 is used at higher doses to treat hypotension associated with sepsis, but research has indicated a higher mortality rate using this strategy, compared with using noradrenaline alone which has vasoconstrictive properties at any dose (Boulain *et al*. 2009).

Undesirable effects surround tachycardia, anginal pain, hypertension and arrhythmias due to increases in myocardial oxygen demand (Elliott 2006). The nurse should be vigilant of these effects, to ensure the patient does not suffer an ischaemic event. Further effects are reported to be decreases in PaO_2, due to increases in pulmonary artery wedge pressures and pulmonary shunt (Hannemann *et al*. 1995).

Enoximone

Enoximone essentially acts by increasing the amount of calcium in myocardial cells, resulting in increased contractility without increasing HR. It achieves this by inhibiting the action of phosphodiasterase, thus increasing the myocardial intracellular levels of cyclic adenosine monophosphate (cAMP). This increase in cAMP enhances calcium uptake and binding to the actin-myosin system resulting in increased contractility and CO without an increase in HR (Gilles *et al*. 2005). Enoximone also has vasodilatory effects by increasing intracellular concentrations of cyclic guanosine monophosphate, which leads to reduced membrane calcium pump action in vascular smooth muscle resulting in vasodilation (Levick 2010).

Enoximone is generally used to wean coronary artery bypass graft (CABG) patients off cardiopulmonary bypass or to increase CO in chronic heart failure. Increasing contractility without an elevation in HR is an useful property when myocardial oxygen demand is important, as may studies have found an improvement in myocardial performance with enoximone infusion in postoperative CABG patients (Bader *et al*. 2010).

There is evidence, however, that enoximone can result in higher incidences of arrhythmias, in particular supraventricular tachycardia and ventricular tachycardia (British National Formulary 2011). The recommended dose range for enoximone is a bolus dose of 0·5–1·5 mg/kg over 10–30 min, followed by a maintenance infusion of 5–10 mcg/kg/min, titrated for therapeutic effect (Gilles *et al*. 2005). Enoximone is supplied in 20 mL ampoules and can be diluted to 40 mL with 0·9% sodium chloride or water for injections, and is incompatible with glucose solutions (British National Formulary 2011).

NURSING AND PROFESSIONAL ISSUES

The administration of all medication must be conducted with the highest standards in mind. This includes ensuring the prescription is correct that clearly indicates: patient identification, name of medication, dose, route of administration and correct reconstitution solution. It is considered good practice to double check these details with another qualified nurse to avoid drug errors (Nursing & Midwifery Council 2010). It is therefore imperative that the nurse be aware of the actions of an inotrope and the correct dose, along with the appropriate diluting solution, in the case of the inotropes discussed in this article 5% dextrose or 0·9% sodium chloride is appropriate (British National Formulary 2011). With the exception of Enoximone, which must be diluted in 0·9% sodium chloride or water for injections (British National Formulary 2011). Research by Tremblay *et al*. (2008) has indicated that noradrenaline undergoes a greater degree of oxidization when mixed with 0·9% sodium chloride compared with 5% dextrose solution. Research of this nature has bought recommendations that inotropes should only be diluted in 5% dextrose solutions.

As the nurse is usually responsible for preparing and connecting infusions it is vitally important for strict infection control guidance be followed. This includes thorough hand washing before preparing the infusion, wearing gloves to prepare and connect the infusion (Macklin 2003). The nurse should also ensure the drug thoroughly mixed within the dilution fluid to ensure even concentration throughout the syringe (British National Formulary 2011). Once the inotrope has been diluted in the syringe, it should be clearly labelled reflecting the prescription. There should also be thorough checks of drug compatibility, if inotropes are to be infused through the same CVC port as another drug. The needleless connection port on the CVC should be thoroughly cleaned with chlorhexidine solution to avoid introducing bacteria into the circulation (O'Grady *et al*. 2011). Furthermore, the administration line should be dated to ensure regular changes are made, with current research indicating lines should be changed every 72 h (O'Grady *et al*. 2011). No form of inotrope should be connected to the central venous pressure monitoring distal port of the CVC, as flushing this port with the transducer set will deliver a bolus of inotropic drug that could have catastrophic consequences for the patient.

In addition to commencing inotropic infusions, the nurse is also responsible for renewing inotropic infusions. This can pose risks to critically ill patients due to their dependence on a consistent dose to maintain haemodynamic parameters. Significant haemodynamic instability can often be observed during infusion renewal due to a reduction in serum levels of the inotrope, as most inotropes have a short half-life of 2–3 min (Trim and Roe 2004). Therefore, it is essential that new infusion syringes are prepared well in advance of the current infusion finishing if catastrophic haemodynamic instability is to be avoided.

Methods such as 'piggybacking' or 'double pumping' involve starting a second syringe of the inotrope

before the first infusion finishes, this ensures serum levels do not drop abruptly. Anecdotal evidence suggests there is a danger of giving an inadvertent bolus during this procedure resulting in hypertension. Furthermore, the nurse should consider the time it takes for the syringe driver to exert pressure on the syringe plunger, one method can be to run the infusion before connecting to the patient enabling the nurse to visualize fluid dripping from the administration set. Trim and Roe (2004) suggest 'piggybacking' is effective in minimizing haemodynamic instability but requires the nurse to be skilled and knowledgeable about inotropes and the infusion devices used to deliver these drugs. The skilled use of monitoring devices such as PiCCO or oesophageal Doppler can assist the nurse in rapidly assessing the haemodynamic effects a patient experiences during infusion renewal (Cottis *et al*. 2003), thus enabling a tailored approach to infusion renewal for each patient.

Once the patient is weaned off their inotrope infusion, the infusion port should be aspirated with an empty syringe until blood is withdrawn, this port should then be flushed with 0·9% sodium chloride in a second syringe. Patient haemodynamics should be continuously observed during this procedure to ensure any inadvertent bolus of inotropic drug is quickly identified.

As the result of the vasoactive properties and circulatory compromise of patient receiving inotropic therapy skin perfusion may be poor. This necessitates vigilance in pressure area care, accurate completion of pressure area assessment tools, with regular position alterations and utilization of pressure relieving devices recommended (Boyle and Green 2001). Consideration should also be given to accuracy of saturation readings on patients at higher doses of noradrenaline, as the vasoconstriction can cause poor peripheral perfusion. In this case, saturations should be measured at a central location; one recommendation can be using a probe on the patient's ear lobe.

CONCLUSION

To deliver safe patient care when administering inotropes it is essential that the nurse has a high level understanding of the physiology of CO and BP control. There is a requirement to know of the various receptors acted on by inotropes and how these influence CO and BP. As the result of the way, different inotropes have differing effects on haemodynamic the nurse must have an understanding of the indications for inotropic therapy, and why a combination of inotropes is sometimes used to achieve the desired parameters. Inotropes have a potent effect on the cardiovascular system, therefore invasive BP and continuous ECG monitoring must be in place during infusion. These are used to titrate infusion rates to achieve predetermined parameters, however, consideration should be given to individual inotropes therapeutic dose range. These should be calculated based on the patient's weight, to determine the patient's escalating or reducing inotropic requirements.

Each inotrope has its own pharmacological properties and side effects; therefore, the nurse should have a good working knowledge of all these factors if they are to deliver safe care to the critical care patient. This also applies to the mixing and administration of the infusion, with application of infection control and safe medicine administration measures essential for patient safety. As the result of a critically ill patients dependence on a consistent dose of inotropes to maintain stable haemodynamic parameters the procedure of renewing the infusion can pose risks to patient stability. Methods such as 'piggybacking' or 'double pumping' can minimize this instability. However, the nurse must be knowledgeable about the inotrope, individual patient response and the infusion device being used.

The use of inotropes is an integral treatment method in critical care; therefore, the nurse has a duty to acquire a high level of knowledge on indications for inotropes, and their effects if high quality patient care is to be achieved. This article provides support for the nurse in achieving this goal, but research in this area of treatment is ongoing, exploring effectiveness of different inotropes on varying conditions and evaluating the significance of newer inotropic drugs in critical care.

KEY LEARNING POINTS

- Inotropic drugs have an effect on myocardial contractility and SVR by either causing vasoconstriction or vasodilation.
- They exert these effects by acting on α and β receptors, with dopamine affecting DA receptors.
- Enoximone increases myocardial calcium thereby enhancing contractility without causing tachycardia.
- Inotropes are administered at a rate to achieve desired haemodynamic parameters, but the majority of inotropes have a therapeutic dose range calculated in mcg/kg/min.
- Inotropic therapy is tailored to meet the requirements of individual patients, as differing conditions require different approaches to ensure patients benefit from the infusion.

- Thorough preparations should be made for timely renewal of infusions, as failure to do so could result in catastrophic haemodynamic instability.
- Vigilance and high quality nursing assessments should be exercised when infusing these drugs, due to their potent nature and detrimental adverse effects.

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