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NON-RANDOMISED, PROSPECTIVE STUDY OF INOTROPES IN A TERTIARY CARE HOSPITAL

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ABSTRACT

Aim: To conduct non-randomised, prospective study of inotropes in a tertiary care hospital. **Objectives:** To assess the clinical profile of patients requiring inotropes and their outcomes. To analyse the association of inotropes in patients. To assess the indications of inotropes. To assess the mortality rate among individuals who are receiving inotrope therapy. To analyse the length of hospital stay of inpatients. **Methods:** 100 cases were collected for the study, at the Kims - Sunshine Hospital in secunderabad for a six-month period of time. The data was obtained from patient case sheets from ICUs and ER. **Results:** In this study males are predominant over females in distribution of inotropes based on gender that is males are 69(69%) and females are 31(31%). The age wise distribution of the patients represents that most of the patients belongs to the age of 58- 67 years (28%). The major length of hospital stay from 68 patients out of 100 patients was for 6-10 days 48(48.5%). In contrast to recovery rate and mortality rate our studies represents that recovery rate is higher 87(87%) than mortality rate 13(13%) in patients during study period. Incomparison of mortality by monotherapy of inotropes Vs co-therapy of inotropes, mortality is higher in co-therapy of inotropes 7(53%). which indicates that monotherapy of inotrope is safer than combination of inotropes therapy. **Conclusion:** From our Non-Randomised, Prospective Study of Inotropes in a tertiary care hospital concludes the following: Even though inotropes may not be harmful choosing the right drug and knowing all the advantages

and hazards is necessary in every therapeutic situation. As of now there aren't any solid and persuasive studies to back up certain inotropic medication treatment to lower mortality in abnormal BP patients with cardiac shock and poor cardiac output syndrome followed by CAD, Acute HF, undergoing heart surgery.

KEYWORDS: Inotropes, Mortality rate, Length of hospital stay.

INTRODUCTION

Inotropes are substances that alter the heart's contraction force.[1] They raise cardiac contractility which enhances cardiac output, aids in sustaining mean arterial pressure & improves body perfusion.[2]

They have been divided up into two groups. Positive inotropes: These medications allow the heart to squeeze tighter and make each heartbeat more effective at pushing blood through the body by improving cardiac output. Where cardiac output is amount of blood the heart pumps for every minute. Negative inotropes: These are the medications that slow down a person's pulsation and reduce contractility.[3]

CARDIOVASCULAR EFFECTS OF COMMON POSITIVE INOTROPES

Dobutamine

It is relatively a selective beta1 synthetic catecholamine which has a direct positive inotropic effect on the heart leading to increased cardiac output. At low dosages (less than 10 microgram/kg/min), dobutamine often has no chronotropic or vasodilatory effects. Since the infusion has a short half-life of two minutes, elimination occurs quickly after terminating it.[4] It has very minor effects on hypertension, arrhythmia, vasodilation, chronotropic, and cardiac beta-adrenoceptor activation.[5]

The usual dose range is 5-20 μg/kg/min, but doses as high as 200 μg/kg/min have been used safely. Therapy should be driven by hemodynamic endpoints, and not by pre-selected dose rates. It is indicated in treatment of myocardial depression is associated with septic shock, but usually must be combined with a norepinephrine to raise the blood pressure,[6] cardiomyopathies, cardiac surgeries,[7] cardiogenic shock.[4]

Epinephrine

Epinephrine functions via binding to beta and alpha-adrenergic receptors. Three factors contribute to the elevation in blood pressure: peripheral vasoconstriction, an elevated heart rate

(positive chronotropic action), and direct myocardial stimulation that strengthens ventricular contraction (positive inotropic action).[8] Used for hemodynamic support in post CABG patients, anaphylactic shock. Although epinephrine is just as effective in treating septic shock as other catecholamines, its use in this condition has been restricted due to adverse effect concerns.[6]

Nor - epinephrine

A naturally occurring substance, nor - epinephrine has its strongest effects on vascular alpha-1 adrenergic receptors, causing vasoconstriction and raising both the systolic and diastolic blood pressure. Additionally, it has chronotropic and inotropic effects via acting on cardiac beta-1 receptors. Norepinephrine is usually administered withal a more effective inotropic drug in individuals with Acute HF who with cardiogenic shock, based on these characteristics.[9]

In particular, the FDA has authorized its usage as a possible adjuvant to treat serious hypotension along with cardiac collapse and for blood pressure management in certain acute hypotensive conditions.[10]

Milrinone

Increased contractility (inotropy) and enhanced relaxation (lusitropy) are the results of PDE III inhibition in the heart. This enhances cardiac output and improves both systolic and diastolic function. Although less noticeable than the increases in heart rate observed with drugs in the catecholamine class, increased heart rate (chronotropy) nonetheless happens. By stopping the breakdown of cAMP, inhibition of phosphodiesterase III raises protein kinase A activity, which phosphorylates calcium ion channels in the sarcoplasmic reticulum and increases calcium availability in myocyte sarcomere. An increase in cardiac inotropy and chronotropy is a manifestation of the previously noted enhanced calcium availability. Thus, greater calcium absorption into the sarcoplasmic reticulum as a result of Milrinone's PDE III suppression leads to better diastolic function and enhanced cardiac relaxation (lusitropy).[11]

Levosimendan

Levosimendan makes the heart contract harder and promotes better vasodilation. It raises cardiac contractility by making the heart more sensitive to calcium. It works by binding to cardiac troponin C in a calcium-dependent way, which increases the sensitivity of myocytes to calcium. It produces a vasodilatory effect and smooth muscle relaxation by activating adenosine triphosphate-sensitive potassium channels in vascular smooth muscle.[12]

The pharmacokinetics of levosimendan remain mostly unchanged with age, gender, mild hepatic impairment, and other organ failure, and they are similar in healthy individuals and patients with HF. They were discovered to be comparable to those shown earlier in single dosage investigations following a continuous infusion for seven days. Bodyweight affected both the central volume of distribution and the clearance, with weight-adjusting dosages accounting for this in reality. Lanoxin and beta-blocking medications were not among the other factors that had a substantial impact on levosimendan's pharmacokinetics.[13]

Dopamine

Peripheral vasodilation and cardiac contractility are increased when dopamine activates β receptors in the heart and peripheral circulation at modest infusion rates (3–10 μg/kg/min). Used to treat cardiac shock and septic shock, despite the fact that alternative approaches are favoured in these conditions like some mechanical assist devices are preferred for cardiogenic shock, and norepinephrine is preferred for septic shock.[6]

Digoxin

Positive ionotropic: It raises the heart's contraction force by reversibly blocking the myocardial Na-K ATPase pump's activity, which regulates the flow of ions into the heart. Lanoxin stimulates a rise in intracellular sodium, which prompts the heart to pump more calcium and become more contractile. Ventricular fullness decreases as cardiac output increases & It isshown by improved exercise capacity and decreased heart failure hospitalizations and emergency care, while having no or less effect on mortality.[14]

It lowers heart rate and increases beating force. When atrial fibrillation is present, which is characterized by a rapid and erratic heartbeat, the drop-in heart rate is especially helpful. Digoxin users who have an auxiliary atrioventricular (AV) route run the risk of experiencing ventricular fibrillation due to a quick ventricular response. digoxin users with pre-existing sinus node disease and AV block had an increased chance of dying from severe or total heart block.[15]

Isoprenaline

ß-adrenergic agonist isoprenaline is a strong, nonselective agonist. Its favourable inotropic effects enhance the rate of heart and contractility.[16] As it is in use of more indications of heart related diseases it is known to have shorter period of action, cleared fast and having a wide therapeutic index.[17]

CARDIOVASCULAR EFFECTS OF COMMON NEGATIVE INOTROPES

1. BETA BLOCKERS

Metoprolol

Metoprolol blocks adrenergic receptors selectively in the heart.

Metoprolol inhibits beta 2adrenoreceptors found in the bronchial and vascular musculature at g reater concentrations.

Only at plasma concentrations far higher than those needed for betablockade can membrane sta bilising activity be seen, and it lacks inherent sympathomimetic activity. It is indicated and used in the treatment of Hypertension, Angina pectoris, Heart failure. Several potential mechanisms have been suggested for the case of hypertension: (1) a central ef fect that reduces sympathetic outflow to the periphery; (2) a central effect that suppresses renin activity; and (3) competitive antagonism of catecholamines at peripheral (particularly cardiac) adrenergic neurone sites, which results in decreased cardiac output.

In the case of angina pectoris, metoprolol prevents catecholamine-induced increases in heart rate, blood pressure, myocardial contraction velocity and extent, and heart rate, thereby lowering the heart's oxygen demand at any given level of exercise. Because of this characteristic, metoprolol is advantageous for the long-term management of angina pectoris.

In the case of heart failure: It is yet unclear exactly how beta- blockers benefit heart failure patients.[18]

Atenolol

Cardio selective beta-1-adrenergic antagonist, such as Atenolol, selectively binds to the beta-1-adrenergic receptors found in vascular smooth muscles and the heart, there by suppressing the sympathetic nervous system, which is a mechanism by which internalized catecholamines, such as isoproterenol, norepinephrine and Epinephrine, reduce positive inotropic and chronotropic effect. Myocardial contractility is reduced along with BP and HR by this mechanism. Tenormin, however, can raise left ventricular Fibre lengths and end-diastolic pressure in heart failure patients, which increases the demand for oxygen. A second-generation beta-1-selective adrenergic antagonist, Tenormin is prescribed to treat AMI, pectorial angina, and hypertension. managing irregular heart rhythms, prophylaxis against migraines, PSVT, and prevention against subsequent myocardial infarction are among the indications that are not FDA-approved.[19]

Propranolol

Propranolol belongs to the class II antiarrhythmic group and is a nonselective antagonist of beta-adrenoreceptors. It responds by competitively inhibiting the heart's beta-1 and beta 2 adrenergic activation, which is normally brought on by norepinephrine and adrenaline. Heart myocytes, such as the atrioventricular and sinoatrial nodes, have beta-1 receptors. Increased cyclic AMP is the result of these receptors being activated and thus increases intracellular calcium. Muscle Fibers become more contractile in regard to this process. A blockage of beta-adrenergic receptors causes the heart's total workload to diminish, which in turn causes a drop in oxygen demand and cardiac remodelling.[20] The non-selective beta-adrenergic antagonist propranolol is used to treat pheochromocytoma, essential tremor, hypertrophic subaortic stenosis, migraine, hypertension, angina, and atrial fibrillation.[21]

Carvedilol

Carvedilol blocks beta adrenoceptors, which prevents tachycardia brought on by exercise. Because carvedilol relaxes smooth muscle in the vasculature through its effect on alpha-1 adrenergic receptors, peripheral vascular resistance is decreased, and blood pressure is generally lowered. Antioxidant activity and calcium channel blockage are also observed at higher doses.1 Carvedilol's antioxidant action stops low density lipoprotein from oxidizing and from being absorbed into the coronary circulation. Non-selective beta-adrenergic antagonists such as carvedilol is used in clinically stable persons for the treatment of mild to severe CHF, high blood pressure, and LVD following myocardial infarction in clinically stable patients.[22]

2. CALCIUM CHANNEL BLOCKERS

Verapamil

Verapamil is a calcium channel blocker that is non-dihydropyridine based and is used to treat hypertension, arrhythmia, and angina.

The mechanism by which verapamil inhibits L-type calcium channels involves binding to a particular region of the alpha-1 subunit, which is highly expressed on these channels in cardiac and vascular smooth muscle. These channels regulate peripheral vascular resistance and heart contractility. Action potentials required for muscle contraction and the electrical activity of the heart's pacemaker are propagated through these channels when calcium enters the body. Due to verapamil's voltage- and frequency-dependent binding to these channels, affinity increases in regard to both 1) a decrease in smooth visceral muscle membrane potential 2) an excessive

depolarizing stimulation. Verapamil's method of action in the treatment of angina and hypertension is likely attributable to the mechanism outlined above. When calcium influx is inhibited, vascular smooth muscle cannot contract, which relaxes and dilates blood vessels throughout the peripheral circulation. This lowers blood pressure and systemic vascular resistance, also known as afterload. By lowering the force that the heart must push against, this decrease in vascular resistance also lowers the energy and oxygen requirements required by the heart, which relieves angina. Electrical activity through the AV node is crucial for controlling heart rate, and this activity is reliant on the entry of calcium via L-type calcium channels. Verapamil prolongs the refractory period of the AV node and slows conduction by blocking these channels and reducing the inflow of calcium. This helps patients with arrhythmias calm and regulate their heart rates. Although the exact mechanism of action of verapamil in treating cluster headaches is unknown, it is believed to stem from an impact on other calcium channels (such as N-, P-, Q-, or T-type). various targets that verapamil is known to interact with are adrenergic receptors, potassium channels as well as various calcium channels.[23]

Diltiazem

A calcium channel blocker called diltiazem is used to treat hypertension and chronic, stable angina.[24] The calcium channel blocker (CCB) diltiazem is not dihydropyridine-based. A variety of processes underlie therapeutic benefits. Diltiazem stops the entry of calcium ions to the heart muscle during depolarization, which is its main mechanism of action. Arterial dilatation and a drop in blood pressure are the results of smooth muscle relaxation caused by lower intracellular calcium concentrations. High blood pressure: Diltiazem mainly lowers peripheral vascular resistance and relaxes vascular smooth muscle to achieve its antihypertensive action. Blood pressure decrease is correlated with hypertension, therefore people with hypertension receive an antihypertensive effect, whereas people with normotension only see a slight drop in blood pressure. Paroxysmal supraventricular tachycardia and atrial arrhythmia: Diltiazem exhibits both a negative chronotrope (lower rate) and negative inotrope (decreased force). Combination of coronary artery dilation and decreased myocardial oxygen demand can lead to decrease in heart rate.[25]

3. ANTI-ARRHYTHMICS

Amiodarone

It is indicated and used in secondary avoidance of ventricular arrhythmias that might be fatal, AF, Ventricular tachyarrhythmias. It is from class 3 antiarrhythmics. The heart's ability to

repolarize during phase 3 of the cardiac action potential is inhibited by potassium rectifier currents. The lengthened effective refractory time and enhanced action potential duration in cardiac myocytes are the outcomes of this potassium channel-blocking impact. The medication inhibits ectopic pacemaker automaticity, lowers the conduction velocity of the AV node, and decreases the automaticity of the sinoatrial (SA) node. These are the electrophysiological effects of the medication.[26]

METHODOLOGY

Study site and period: The study was performed in the Kims – sunshine hospital, Begumpet for a period of 6 months.

Study design: Non-Randomized prospective observational study.

Study size: A hundred patients were accounted for sample size.

Study criteria

Inclusion criteria-

- Patient with age 18 years and above
- Patients who were initiated on inotropes.
- Gender
- Co-morbidities (HTN, DM and others)
- Social habits

Exclusion criteria-

- Paediatrics
- Pregnant & lactating women
- Neonatal ICU, MICU

Source of data collection

Study resource

- Patient assent form: The patient assent form includes the patient's personal information, the study title, study specifics, and the participants and researcher's signature. Participants must willingly sign the patient assent form to indicate that they are willing to participate.
- Patient profile form: it includes the information about the patient's name, span of life, sex, Date of admittance, date of disgorge, reasons for presenting, nonobjective and non-subjective evidence, medication notes, clinical progress, medications at discharge.

Study procedure

This is a research study which involves group of participants and observe how inotropes are used over a certain period. Research was carried out in Kims- sunshine hospital of Begumpet. After following consent, one hundred patients in all were taken into consideration in the patient profile form (sample data collection form) in 6 months of time period.

The sample was divided based on the age, gender, co-morbidities, anamnesis, past medicines tract record, surgical history, social habits, diagnosis treatment chart (inotrope therapy) and discharge summary. The percentage of each factor was calculated within the sample. MS Excel was used to create the graphical representation of the data that was gathered.

RESULTS

DISTRIBUTION BASED ON GENDER

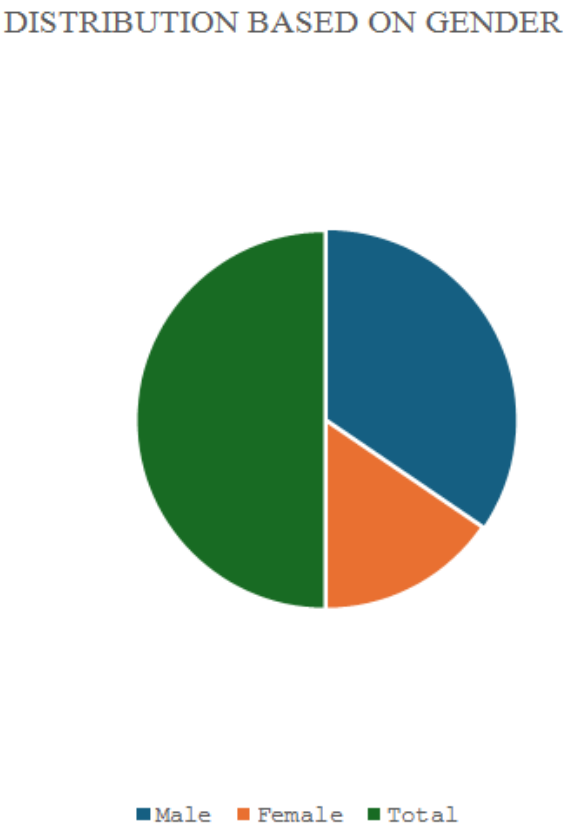

Figure 1: Distribution of inotropes based on gender.

The above-mentioned pie chart displays that 100 patients were initiated on inotropes. Out of which no. of females were 31 and no. of males were 69 which represents 31% of females and 69% of males respectively.

DISTRIBUTION OF INOTROPES BASED ON AGE

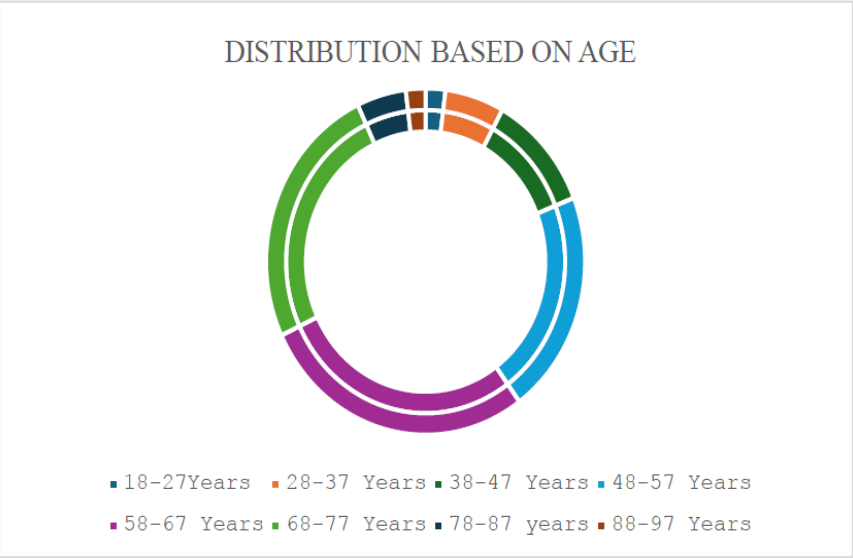

Figure 2: Distribution of inotropes based on age.

The doughnut chart above illustrates that 100 no. of patients were prescribed with inotropes ranging from 18-27 years. The majority of patients treated with inotropes in the age group of 58-67 years were 28 (28%) and the least no. of patients treated with inotropes from the age group of 18-27 and 88-97 years were 2(2 %).

DISTRIBUTION OF INOTROPES BASED ON SOCIAL HABITS

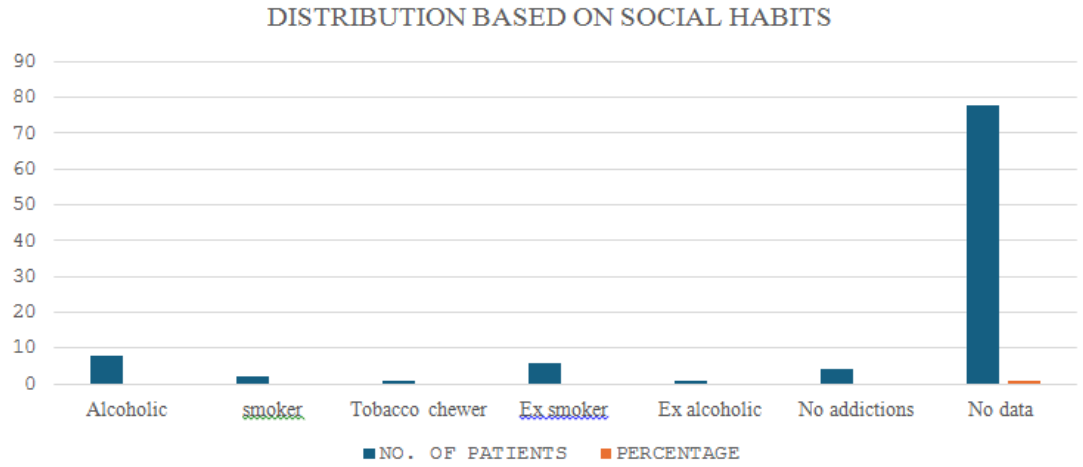

Figure 3: Distribution of Inotropes based on social habits.

The above bar graph represents the various social habits of 100 patient out of which 78% data was not available, and out of 22% the majority of the patients were alcoholic (8%), and minority of the patients were tobacco chewers and ex alcoholic that is 1%.

DISTRIBUTION OF INOTROPES BASED ON COMORBIDITIES

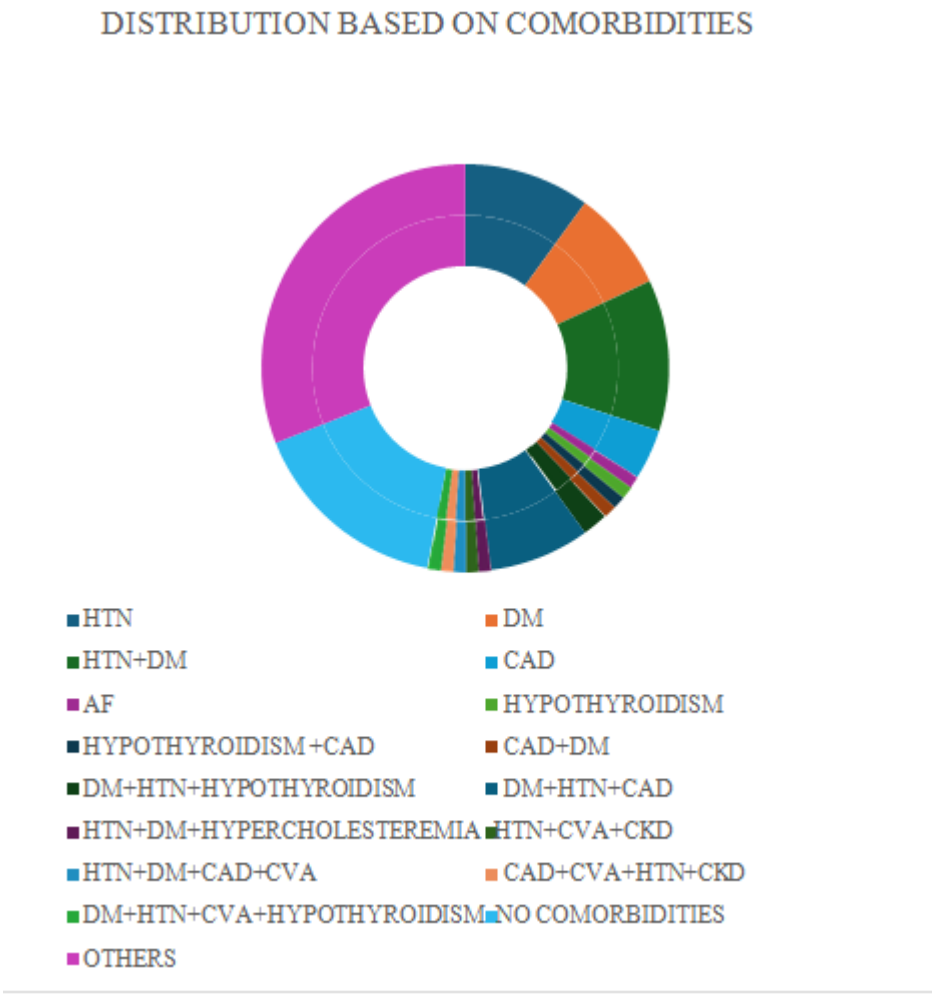

Figure 4: Distribution of inotrope based on comorbidities.

The above sunburst portrays the various comorbidities of 100 patients. Out of which 10 (10%) patients have HTN, 8(8%) patients have DM, 12(12%) patients have HTN+DM, 4(4%) Patients have CAD, 1(1%) patient have AF, 1(1%) patient have hypothyroidism, 1(1%) patient hypothyroidism +CAD, 1(1%) patient have CAD+DM, 2(2%) patients have HTN+DM+HYPOTHYROIDISM, 8(8%) Patients have HTN+DM+CAD, 1(1%) patient have HTN+DM+CHOLESTEREMIA, 1(1%) patients have HTN+CVA+CKD, 1(1%) patient have HTN+DM+CAD+CVA,1(1%) Patients have CAD+CVA+HTN+CKD, 1(1%) Patient have DM+HTN+CVA+HYPOTHYROIDISM, 16(16%) of patients have no comorbidities and 31(31%) patients have other comorbidities.

DISTRIBUTION OF INOTROPES BASED ON INDICATIONS

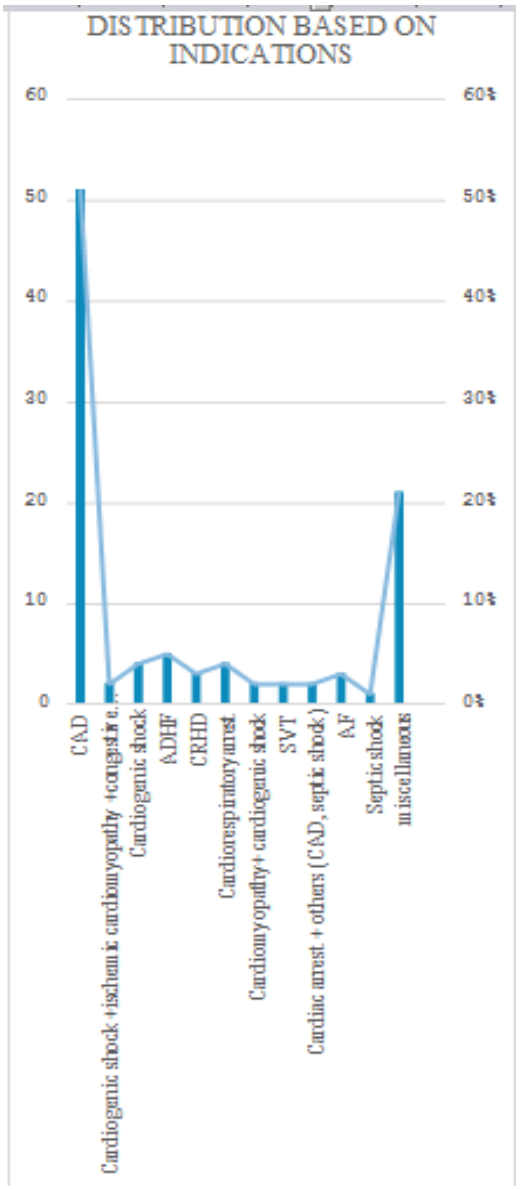

Figure 5: Distribution of inotropes based on indications.

The above graph describes the distribution of inotropes based on various indications in 100 patients taken in a study. Out of which majority of inotropes were used in CAD and minority of the inotropes are used in septic shock.

DISTRIBUTION OF INOTROPES BASED ON DEPARTMENTS

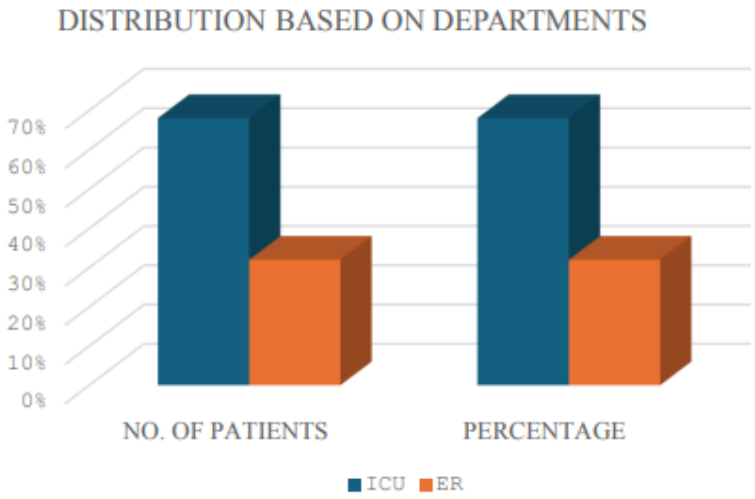

Figure 6: Distribution of inotropes based on departments.

The above graph depicts that majority of inotropes were used in ICU (68%) than ER (32%) during study period.

DISTRIBUTION OF INOTROPES BASED ON LENGTH OF HOSPITAL STAY

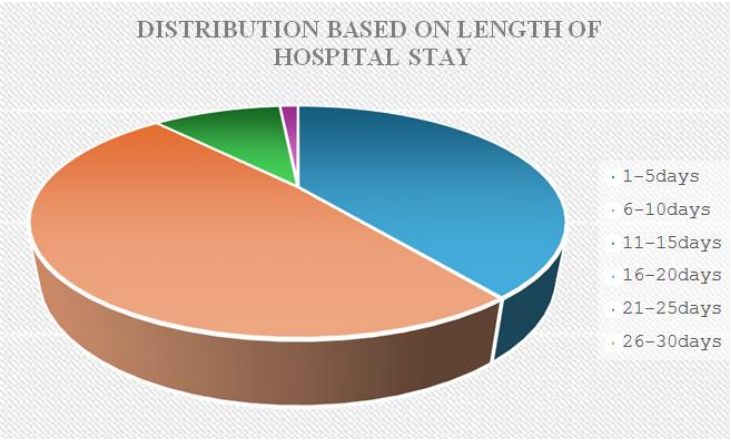

Figure 7: Distribution of inotropes based on length of hospital stay.

The above pie chart and table delineates the length of hospital stay of 68 (68%) patients out of 100(100%). From which majority of length of hospital stay is for 6-10 days (48.5%) and minority was for 21-25 days (1.4%).

DISTRIBUTION BASED ON TYPES OF INOTROPES

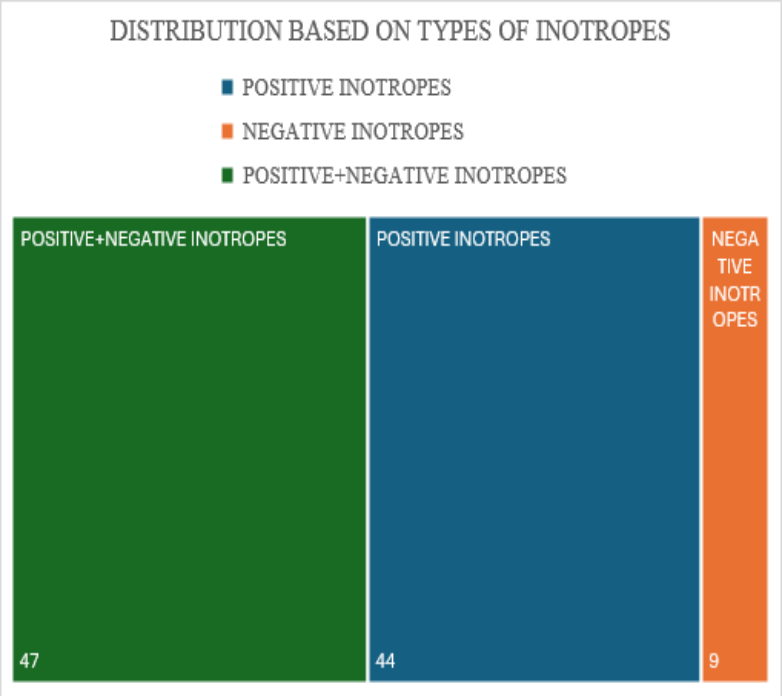

Figure 8: Distribution based on types of inotropes.

The above tree map demonstrates the distribution based on types of inotropes. Amidst positive inotropes, negative inotropes and positive + negative inotropes, positive +negative inotropes were mostly used in the patients during study period.

DISTRIBUTION BASED ON MONO INOTROPE THERAPY Vs COMBINATION INOTROPE THERAPY

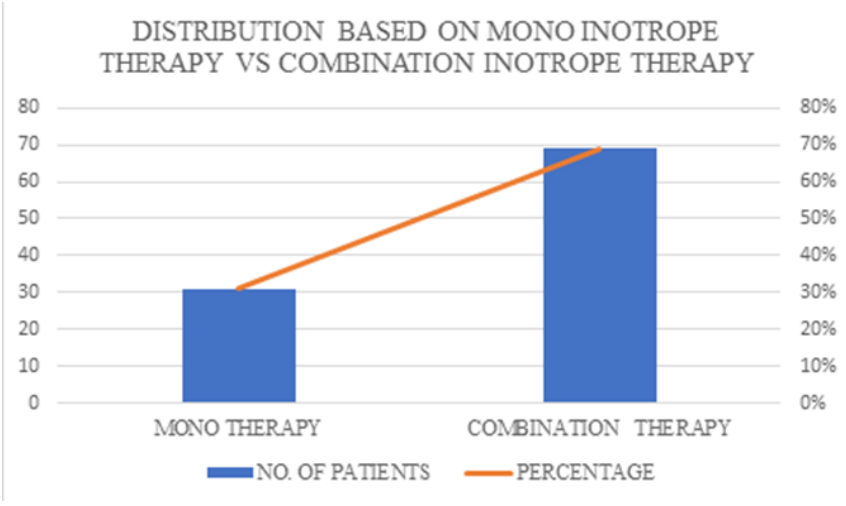

Figure 9: Distribution based on mono inotrope therapy Vs combination inotrope therapy.

The above graph concludes that combination of inotropes therapy was widely used over mono inotrope therapy.

DISTRIBUTION BASED ON POSITIVE INOTROPES Vs NEGATIVE INOTROPES Vs POSITIVE +NEGATIVE INOTROPES

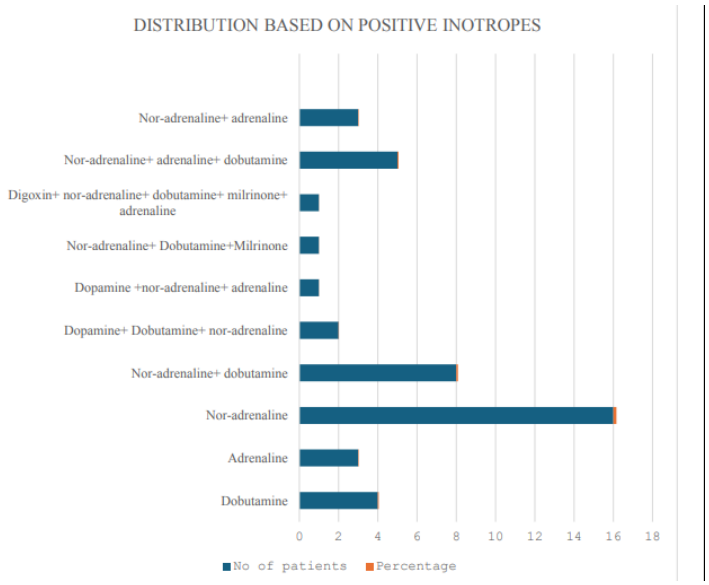

Figure 10: Distribution based on positive inotropes.

The above graph interprets that, from positive inotropes Nor -adrenaline is widely used.

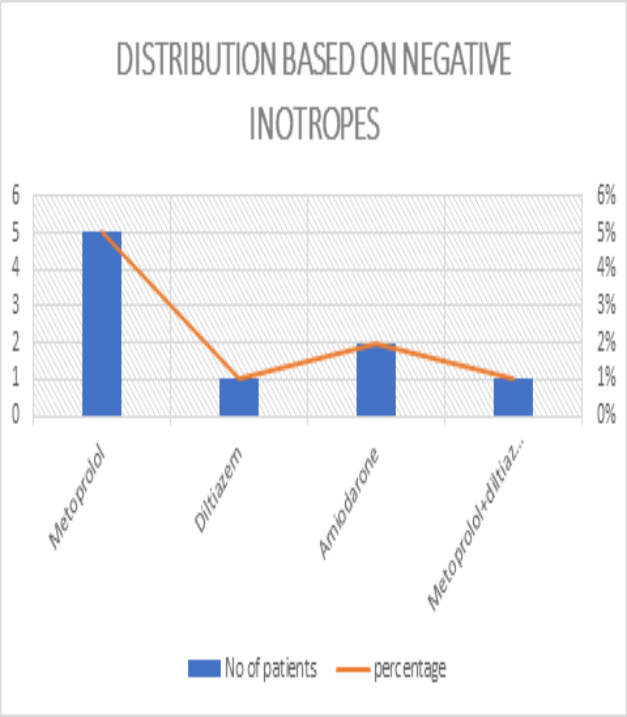

Figure 11: Distribution based on negative inotropes.

The above graph depicts that, from negative inotropes Metoprolol is widely used.

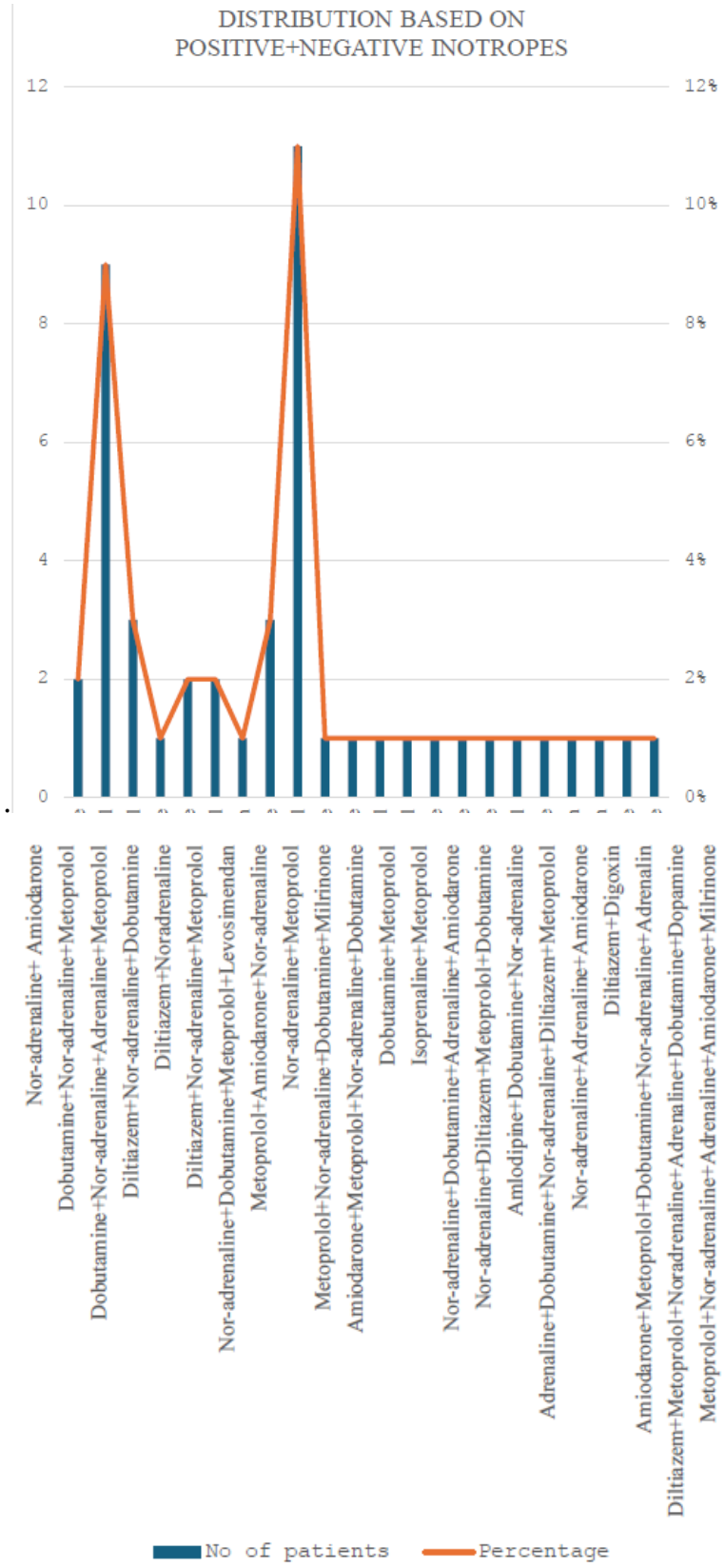

Figure 12: Distribution based on positive + negative inotropes.

The above Clustered column depicts that, from positive + negative inotropes Nor- adrenaline + Metoprolol was widely used.

DISTRIBUTION BASED ON MORTALITY RATE Vs RECOVERY RATE

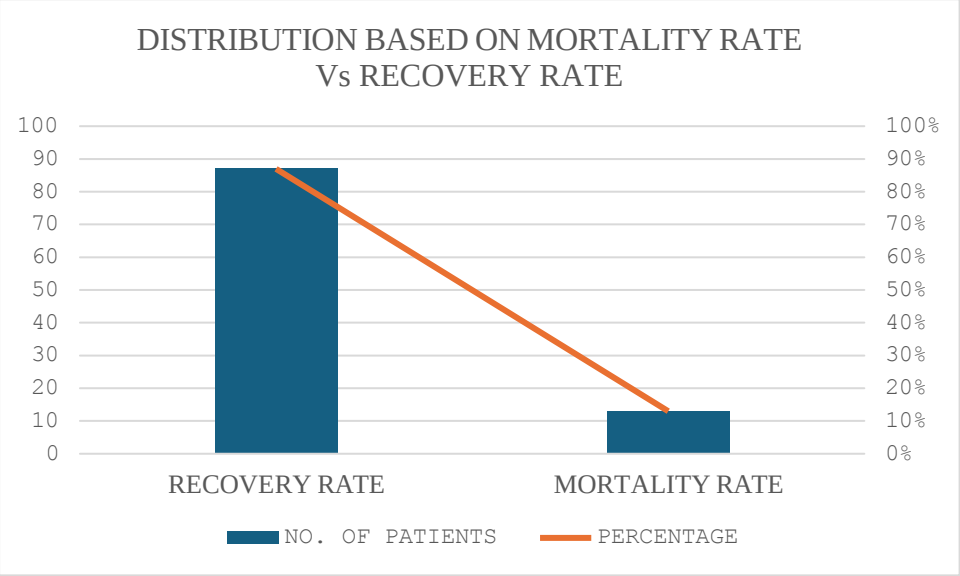

Figure 13: distribution based on mortality rate Vs recovery rate.

The above graph illustrates that recovery rate is more than morality rate.

DISTRIBUTION BASED ON MORTALITY BY MONOTHERAPY OF INOTROPE Vs MORTALITY BY COMBINATION THERAPY OF INOTROPES

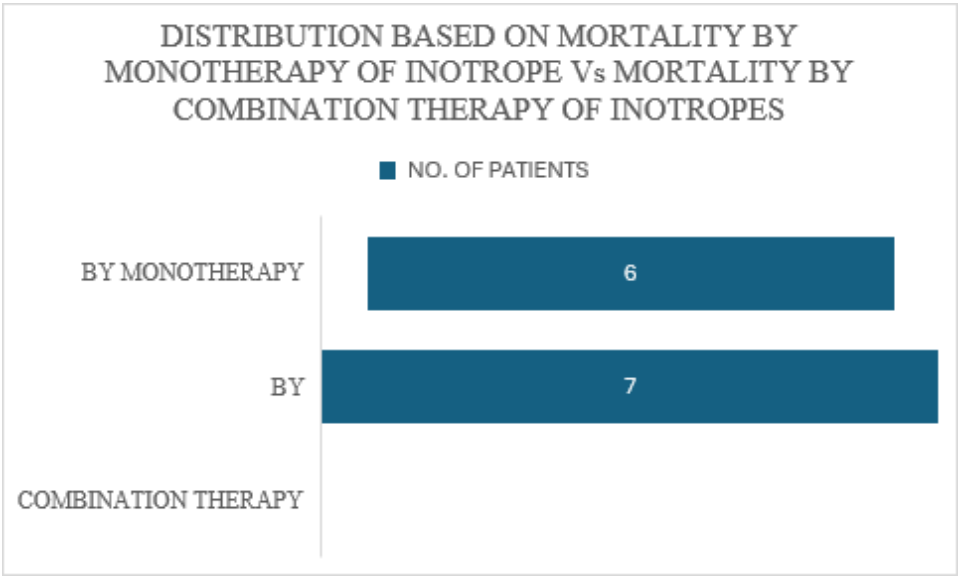

Figure 14: Distribution based on mortality by mono inotrope therapy Vs combination inotrope therapy.

The above funnel graph depicts that mono inotrope therapy is safer than two or more than two inotrope therapy.

DISCUSSION

A total hundred number of persons were incorporated in the study, which assess the clinical profile of patients requiring inotropes and their outcomes. It is a prospective observational study, conducted in a tertiary care hospital for a period of 6 months. Objective of this research is to assess the use of inotropes based on indications, length of the hospital stays, mortality rate, and types of inotropes being used.

Based on gender wise distribution males are predominant over females. out of 100 patients no. of male patients were 69 (69%) and females were 31(31%). During study period, Distribution based on age indicates that majority of inotropes are indicated among the age of 58-67 years was 28(28%) and the least no. of patients treated with inotropes belongs to the age range 18-27 and 88-97 years were 2 (2%).

Based on available data, distribution of inotropes in patients with social habits (like consumption of alcohol, tobacco chewing, smoking) was prevailing in alcoholic patients 8 (8%).Among various comorbidities of 100 patients 10 (10%) patients have HTN, 8(8%) patients have DM, 12(12%) patients have HTN+DM, 4(4%) Patients have CAD, 1(1%) patient have AF, 1(1%) patient have hypothyroidism, 1(1%) patient hypothyroidism +CAD, 1(1%) patient have CAD+DM, 2(2%) patients have HTN+DM+HYPOTHYROIDISM, 8(8%) Patients have HTN+DM+CAD, 1(1%) patient have HTN+DM+CHOLESTEREMIA, 1 (1%) patients have HTN+CVA+CKD, 1(1%) patient have HTN+DM+CAD+CVA,1(1%) Patients have CAD+CVA+HTN+CKD, 1(1%) Patient have DM+HTN+CVA+HYPOTHYROIDISM, 16(16%) of patients have no comorbidities and 31(31%) patients have other comorbidities Distribution of inotropes based on indications was high in patients suffering with CAD 51 (51%) and was found least in patients suffering with septic shock 1 (1%).

Out of 100 patients 32 % cases were from ER and 68 % were from ICU. The major length of hospital stay from 68 patients out of 100 patients was for 6-10 days (48.5%). And minor was for 21-25 days (1.4%). Amidst positive inotropes, negative inotropes and positive + negative inotropes, positive +negative inotropes (47%) were mostly used in the patients during study period. According to S Lavanya, G Oliver et al., the medication that was prescribed the most was positive inotropes. The average number of prescription medicines was high. Reducing the

number of medications prescribed and increasing the degree of variation between positive and negative inotropes could enhance the prescribing pattern.[1]

Out of positive inotropes Nor -adrenaline16 (16%) is widely used, and from the negative inotrope's met-xl 5 (5%) is mostly used in comparison to other negative inotropes. Whereas in positive +negative inotropes Nor-adrenaline + met-xl 11 (11%) is widely used during study period. In contrast to recovery rate and mortality rate our studies represents that recovery rate is higher 87 (87%) than mortality rate13 (13%) in patients during study period. In comparison of mortality by monotherapy of inotropes with combination therapy of inotropes mortality is higher in individuals with co- therapy of inotropes 7(53%) and lesser in individuals with mono therapy of inotrope 6 (46.1%).

According to Juhani rossinen, et al. Administering inotropes to individuals suffering from acute heart failure is associated with a higher risk of death. However, using just one inotrope while in the hospital seems to be safe.[27]

The institution, the surgeon, and the patient are all responsible for variations in inotrope usage. The need for more quantitative and qualitative research to comprehend how variations in inotrope use impact results and create patient-centred, evidence-based inotrope therapy is suggested by heterogeneity throughout institutions and physicians.

CONCLUSION

From our Non-Randomised, Prospective Study of Inotropes in tertiary care hospital concludes the following

That males are predominant over females and wide use of inotropes in patients was amidst the age group of 58-67 years. The most common comorbidity in patients requiring inotrope was HTN+DM and according to our study the recovery rate (87%) is higher than mortality rate (13%).

Even though inotropes may not be harmful choosing the right drug and knowing all the advantages and hazards is necessary in every therapeutic situation.

As of now there aren't any solid and persuasive studies to back up certain inotropic medication treatment to lower mortality in abnormal BP patients with cardiac shock and poor

cardiac output syndrome followed by CAD, Acute HF, undergoing heart surgery.

FUTURE DIRECTIONS

Existing inotropic medications have not demonstrated positive benefits in individuals with heart failure beyond brief haemodynamic improvement. To overcome these limitations, it is necessary to design new drugs that targets novel system.

Agents having a favourable safety profile and improved cardiac performance are desperately needed in a clinical practice.

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