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Pharmacology for Graduate Nurses

As per the Revised Nursing Syllabus



2nd Edition

Subrata Sarkar

JAYPEE

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2 CHAPTER

Drugs Act on Autonomic Nervous System

CHAPTER OUTLINE

- Introduction: A short glimpse
- Effects of autonomic stimulation
- Chemistry of a synapse
- Receptors
- Adrenergic drugs
- Adrenergic blocking agents
- Cholinergic drugs

INTRODUCTION: A SHORT GLIMPSE

The nervous system is divided into central nervous system (CNS) and peripheral nervous system (PNS). PNS can be further divided into the somatic and the autonomic nervous system (ANS) **(Fig. 2.1)**.

The autonomic pharmacology is the study of those drugs that affect the autonomic nervous system itself or autonomic receptors on the effector cells controlled by autonomic nervous system (cardiac action, temperature regulation, ionic and fluid balance, metabolism, digestion, excretion). **Autonomic drugs** are used clinically to either imitate or inhibit the normal functions of the sympathetic and parasympathetic nervous systems.

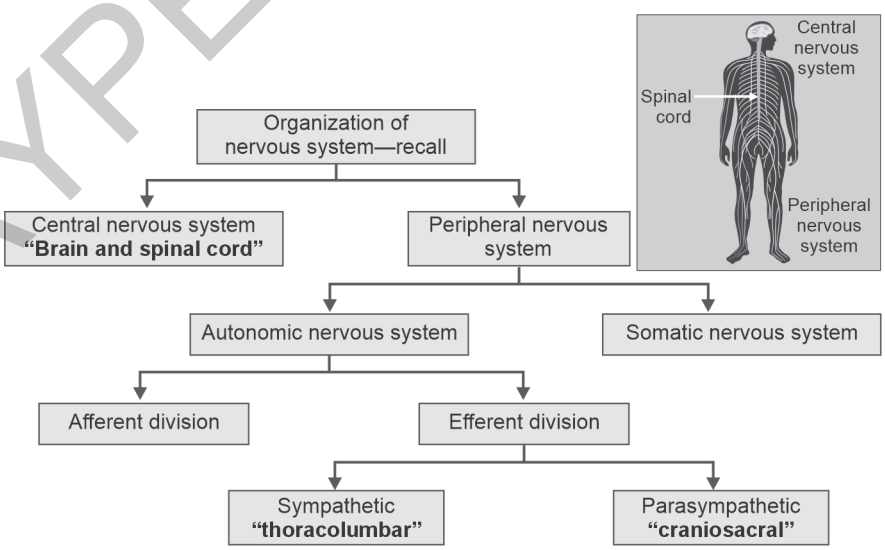

Fig. 2.1: The nervous system.

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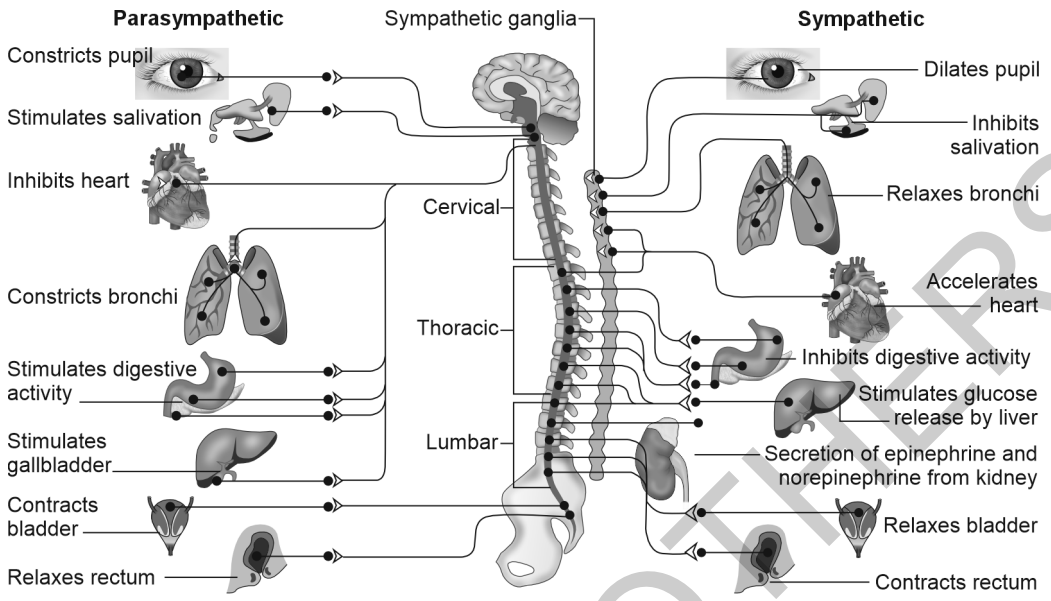

Fig. 2.2: The structures of autonomic nervous system (ANS). Effects of parasympathetic and sympathetic stimulation on various organs.

The autonomic division of the nervous system is largely independent, and its activities are not controlled by conscious control. The necessary life processes (cardiac output, blood flow to various organs, digestion, elimination) are controlled by ANS.

The other major division of the nervous system is somatic division **(Fig. 2.2)**. It is concerned with consciously controlled functions such as locomotion as well as respiration and posture.

- The autonomic or involuntary part of the nervous system controls the functions of the body carried out 'automatically' means initiated in the brain below the level of the cerebrum.
- The effects of automatic control are rapid and essential for homeostasis. The effector organs are:
  - Smooth muscle (gastrointestinal tract, respiratory and cardiovascular system, urogenital system, intrinsic muscle of eye, various secretory glands)
  - Cardiac muscle
  - Glands
- Effects of autonomic stimulation include:
  - Changes in rate and force of the heartbeat
  - Stimulation and depression of secretion of glands
  - Vasoconstriction or vasodilation
  - Bronchoconstriction or bonchodilation
  - Changes in size of the pupils of the eyes
- The ANS is divided into two divisions:
  i. Sympathetic (thoracolumbar outflow T1–L3)
  ii. Parasympathetic (craniosacral outflow; cranial outflow arises from cranial nerves III, VII, IX, and X; and sacral outflow from S2, S3 and S4 spinal roots)
- Sympathetic activity tends to predominate in stressful situations and parasympathetic activity during rest.
- Each division has two efferent (motor) neurons:
  i. Preganglionic neuron
  ii. Postganglionic neuron

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The postganglionic neuron conducting impulses to the effector organ and **noradrenaline (NA)** is usually neurotransmitter. In general, most of the visceral organs have dual nerve supply, i.e., they are supplied by both the divisions of the ANS, the effects of one system predominate. Most of the blood vessels and sweat glands have mainly sympathetic innervations, and the parasympathetic division controls most of the other systems. Their stimulation usually produces opposite effects on the innervating organ.

The cell body of the preganglionic neuron is in the brain or spinal cord. Its axon synapse with the cell body of the postganglionic neuron and **acetylcholine (ACh)** is the neurotransmitter.

EFFECTS OF AUTONOMIC STIMULATION

1. Sympathetic Stimulation: Cardiovascular System

- Exerts an accelerating effect upon the SA (sinoatrial) node in the heart, increasing the rate and force of the heartbeat.
- Causes dilatation of coronary arteries, increasing the blood supply to cardiac muscle.
- Causes dilatation of the blood vessels supplying skeletal muscles. Thus, increasing the capacity of the muscle to work.
- Raises peripheral resistance and blood pressure by constricting the small arteries and arterioles in skin. Increased blood supply is available for skeletal muscle, heart, brain.
- Constricts the blood vessels in the secretory glands of the digestive system, reducing the flow of digestive juices.
- Blood coagulation occurs more quickly because of vasoconstriction.
- Causes dilatation of airways, especially the bronchioles.

2. Parasympathetic Stimulation: Cardiovascular System

- Decrease the rate and force of the heart beats.
- Causes constriction of the coronary arteries reducing the blood supply to cardiac muscle.
  - *The parasympathetic nervous system exerts little or no effect on blood vessels except the coronary arteries.*
- Produce constriction of bronchi.

Components

In each sympathetic and parasympathetic system, nerves leave the CNS to synapse with other nerve fibers in ganglia (located close to the spinal cord in the case of sympathetic fibers, or near effector organs in the case of parasympathetic fibers).

Both sympathetic and parasympathetic nervous divisions originated in nuclei within the central nervous system and give off preganglionic fibers that exist from the brain stem or spinal cord. The parasympathetic fibers leave the central nervous system and are distributed with certain cranial nerves (especially the III, VII, IX, and X) and with the sacral nerves.

The sympathetic nervous system consists of a chain of ganglia lying on either side of the vertebral column and extending from the cervical to the lumbar vertebrae, they are called paravertebral ganglia.

The relay ganglia of the parasympathetic system are situated peripherally near the organs supplied.

Sensory Fibers

In addition to these clearly defined peripheral motor portions of the ANS, there are large numbers of sensory nerves, which supply the various organs. Afferent nerve fibers are axons of sensory neurons that carry sensory information from sensory receptors to the central nervous

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system. The sensory fibers carry information from the organ to the higher integrating centers (hypothalamus and medulla) to evoke the motor activity carried to the effector's cells by the efferent fibers described earlier.

CHEMISTRY OF SYNAPSE

It is in the synapses of the ganglia and the nerve organ junction that endogenous chemicals play a role in transmitting nerve impulses. Synaptic transmission involves the production, storage, and release of chemical neurotransmitters which stimulate a response in the postsynaptic cell by interacting with the receptors.

When sufficient receptors are achieved an action potential is generalized in the receptor cell, resulting in a second nerve impulse or effector's organ response, such as muscle contraction or glandular secretion.

Released neurotransmitter from the axon terminal is dissipated by at least two processes:

1. Enzymes in the extra cellular space catabolize excess transmitter
2. The axon terminal actively reuptakes part of the chemical.

The drug action is based on this theoretical concept:

- Most drug appear to act at the synapses by processes such as augmenting the amount of neurotransmitter, influencing the action of catabolic enzymes in the synaptic cleft, or interacting with receptors on the postsynaptic membranes.
- There are differences in the chemical transmitters and receptors in various parts of the ANS, different drugs modify automatic response in characteristic fashion.

Acetylcholine

Acetylcholine is the neurotransmitter in ganglionic synapses, postganglionic parasympathetic synapses and certain postganglionic sympathetic synapses **(Fig. 2.3)**. Cholinergic drugs that

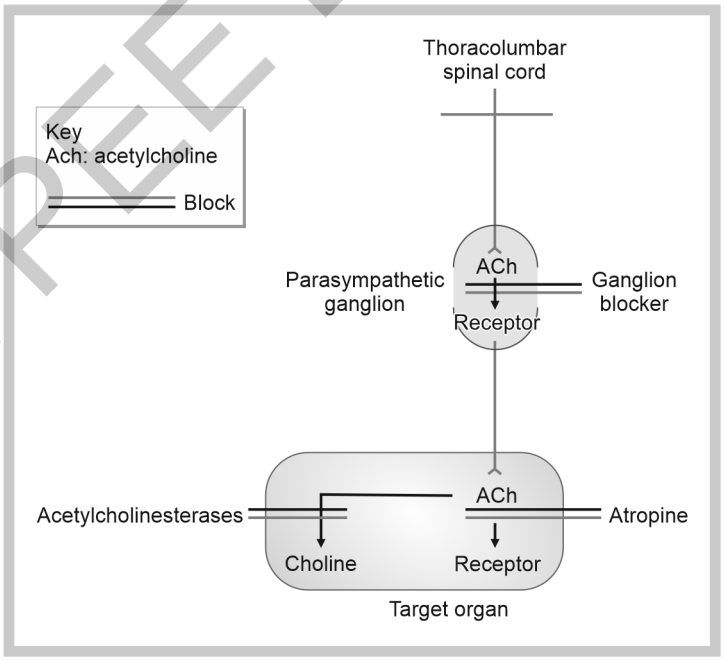

Fig. 2.3: Neurotransmitters and receptors complex of the parasympathetic division of the autonomic nervous system and sites of receptor blocking drugs.

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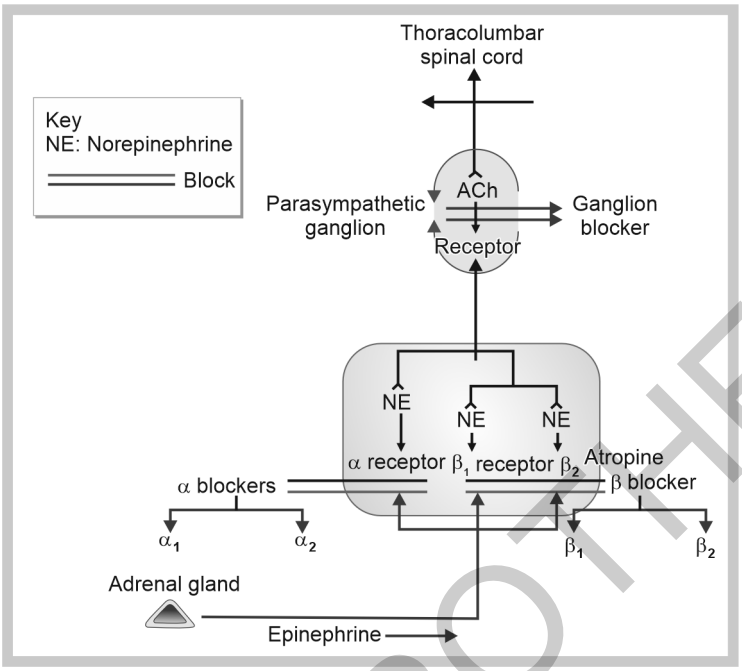

Fig. 2.4: Neurotransmitters and receptors complex of the sympathetic division of the autonomic nervous system. The sites of receptor blocking drug action.

augment or mimic the effects of ACh, therefore, increase parasympathetic activity, presynaptic sympathetic activity, and sympathetic stimulation of sweat glands. The drugs act on this chemical process is known as cholinergic (parasympathomimetic) drugs and anticholinergic (parasympatholytic) drugs.

Adrenergic

Most postganglionic sympathetic fibers release norepinephrine (NE) NA; they are noradrenergic fiber (often called simply "adrenergic") **(Fig. 2.4)**. Sympathomimetic or adrenergic drugs produce the same general physiologic effects as sympathetic nerve stimulation.

In addition, sympathetic releases epinephrine (90%) and NE (10%) from the medulla of the adrenal glands, these substances enter the blood stream and produce widespread effects. Norepinephrine and epinephrine produce these effects by combining with specific sympathetic adrenoceptors on the target organs.

Adrenoreceptors

Both sympathetic and parasympathetic systems appear to have two distinct types of receptors. Receptors of sympathetic systems are designated as alpha (α) and beta (β), these receptors are again classified as α_1 and α_2, β_1, β_2 and β_3 subtype receptors.

RECEPTORS (TABLE 2.1)

- **Alpha-1 receptors:** Receptor subtype, postjunctional, occur on target tissues opposite the nerve terminal and are stimulated by NE released at sympathetic nerve endings and by epinephrine. These receptors mediate vasoconstriction, gastrointestinal relaxation, mydriasis, and inhibition of insulin release and lipolysis.

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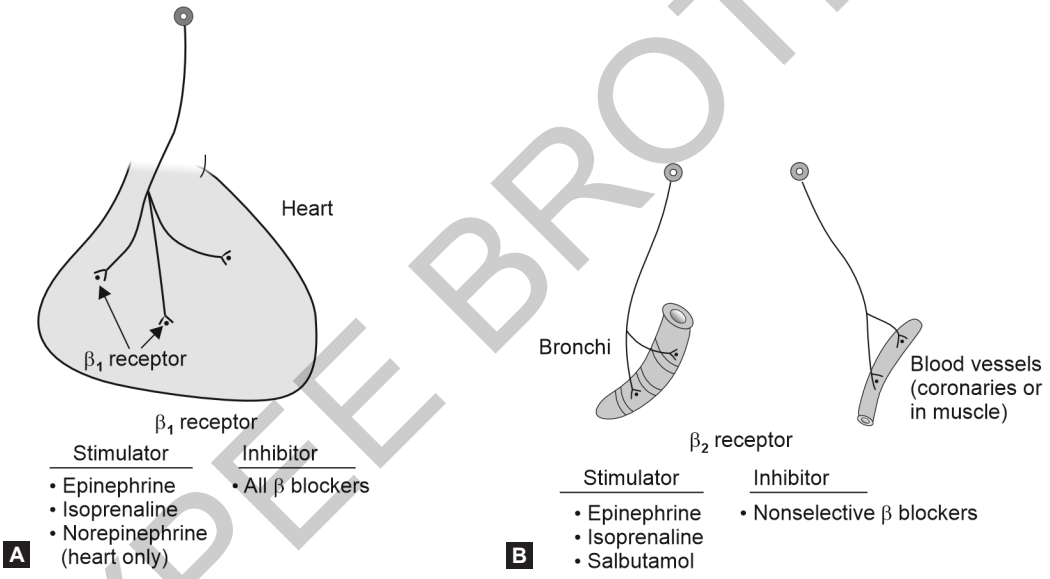
TABLE 2.1: Receptors of autonomic nervous system.

Type of receptor	*Responses mediated by receptors*
Sympathetic Alpha-1	Vasoconstriction, ureteral and uterine contraction, mydriasis, blinking of eyes, inhibition of the gastrointestinal (GI) tract
Alpha-2	Presynaptic feedback inhibition (limit further release of norepinephrine), form a release control mechanism
Beta-1	Increase in rate and excitability of the heart with increased cardiac output Increased conduction velocity in the myocardium **(Fig. 2.5A)**
Beta-2	Dilation of bronchi and blood vessels. Inhibition of uterine contraction **(Fig. 2.5B)**
Parasympathetic muscarinic	Decreased rate of atrial contraction, selective vasodilation. Increased tone and motility of the GI tract, contraction of gallbladder and biliary ducts. Miosis, contraction of ciliary muscle. Contraction of the detrusor muscle of the bladder, relaxation of the bladder sphincter
Nicotinic	Transmission in the autonomic ganglia



Figs. 2.5A and B: (A) β_1 receptors stimulator and inhibitor agents; (B) β_2 receptors stimulator and inhibitor agents.

❖ **Alpha-2 receptors:** Receptor subtype, prejunctional and postjunctional. Prejunctional receptor activation inhibits neurotransmitter release. When α_2-receptors bind to NE (released in the nerve terminal of sympathetic nerve fiber), they limit further release of NE, thus a release control mechanism is formed. Alpha-2 receptors are target for drugs, selectively to inhibit further NE release, and are therefore useful to treat hypertension.

ADRENERGIC DRUGS

Chemicals that cause an apparent increase in sympathomimetic nervous activity in the body are called sympathomimetic, adrenergic or catecholamine. These catecholamines are important regulators of the activities of organs such as the heart and peripheral vasculature, especially in responses to stress. Drugs that particularly or completely mimic the actions of epinephrine or NE are called sympathomimetic, which would be expected to have a wide range of effects.

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TABLE 2.2: Administration of epinephrine—route/dosage.

Patient	*Clinical condition*	*Route*	*Dosage*
Adult	Anaphylactic reactions or asthma	Subcutaneous/ Intramuscular	0.1–0.5 mg (single dose not to exceed 1 mg). May repeat 10–15 minutes interval for anaphylactic shock or 20 minutes to 4 hours for asthma
-do-	Severe anaphylaxis	Intravenous (IV)	0.1–0.25 mg, may repeat 5–15 minutes interval; may be followed by 1–4 µg/min by continuous infusion
	Cardiopulmonary resuscitation	-do-	1 mg every 3–5 minutes
-do-	Bronchospasm	Inhalation	1 inhalation (125–250 µg) by metered dose inhaler may repeat after 1–2 minutes
-do-	Resuscitation	Intracardiac	0.3–0.5 mg
Child	Severe anaphylaxis	IV	0.1 mg (less in younger children 0.01 mg/kg); may be followed by 0.1 µg/kg/min by continuous infusion in anaphylaxis
Neonates	Resuscitation	IV Intratracheal	0.01–0.03 mg/kg every 3–5 minutes as needed

Sympathomimetic drugs are:
- Epinephrine
- Norepinephrine
- Isoprenaline
- Selective β_2-agonists

Epinephrine

The first of the catecholamines to be used widely in medicine, epinephrine (adrenaline) has long been used in medical emergencies. It is still the best immediate treatment for serious anaphylactic reactions. In anaphylactic reactions, epinephrine causes constriction of blood vessels and thus relieves edema and swelling and stimulates the heart in cardiac arrest **(Table 2.2)**.

Pharmacodynamics

Epinephrine stimulates both α- and β-adrenergic receptors. At low concentration β receptor effect is predominant; at high concentrations α receptor effect is predominant.

At low doses, epinephrine increases systolic blood pressure as a result of positive inotropic and chronotropic effects on the heart (β_1 receptor activation) and decreases total peripheral resistance as a result of dilating effect in the vascular bed (β_2 receptors).

At high doses, epinephrine also will activate α receptors in vascular bed to skeletal muscle with vasoconstriction.

Desired therapeutic responses on the clinical situations (the type and location of the receptors with which they interact), may include mydriasis, decreased peripheral resistance and diastolic pressure, vasoconstriction, increased heart rate and force of contraction and bronchodilation. It is a potent vasoconstrictor and cardiac stimulant. It also inhibits the release of histamine in allergic reactions.

Pharmacokinetics

Epinephrine must be administered parenterally or by inhalation because its protein structure is destroyed by gastric acid. Onset of action of the drugs occurs within minutes and persists

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TABLE 2.3: Time and action profile of epinephrine: bronchodilation.

Route	*Onset*	*Peak*	*Duration*
Intravenous	Rapid	20 minutes	20–30 minutes
Intramuscular	6–12 minutes	Unknown	<1–4 hours
Subcutaneous	5–10 minutes	20 minutes	<1–4 hours
Inhalation	1 minute	Unknown	1–3 hours

for short duration. It is rapidly taken up and metabolized in sympathetic nerve endings. This drug is also metabolized by liver and other tissues [action of monoamine oxidases (MAO)]. Metabolites and small number of unchanged drugs are excreted by the kidneys. Absorption from subcutaneous site in slow because of local vasoconstriction.

Does not cross the blood brain barrier, crosses the placenta and enters into breast milk.

Pharmacological Effects

Following injection, actions become apparent within minutes.

- β_1 receptor-agonists increase in the force and rate of contraction of the heart, so that the patient may report palpitation.
- β_2 receptor-agonists cause dilation of blood vessels of skeletal muscle.
- α receptor agonists constrict vascular smooth muscle of resistance blood vessels (i.e., in the skin and splanchnic beds) causing increased peripheral resistance.
- β_2 epinephrine relaxes smooth muscle including that of the bronchial tree **(Table 2.3)**.
- β receptor agonists increase liver and skeletal muscle glycogenolysis and increase lipolysis in fat cells.
- β receptor-agonists increase and α receptor-agonists decrease insulin and rennin secretion.
- β receptor agonists cause relaxation of the ciliary muscle with some decrease in accommodation.
- α receptor agonists contract the radial muscle of the iris and dilate the pupil (mydriasis).
- α receptor agonists and β receptor agonists decrease intraocular pressure.

Interactions

- Adrenaline should not be given during anesthesia with halothane (risk of arrhythmias). Concurrent use with other adrenergic drugs—will precipitate additive adrenergic side effects.
- Use with MAO inhibitors—may lead to hypertensive crisis.
- Use with β-blockers—may negate therapeutic effect.
- Use with tricyclic antidepressants—enhance pressor response to epinephrine.
- Available drug—injection Adrenaline 1 mg/mL.

Adverse effects: Transient restlessness, nervousness, palpitation, anxiety, tremor, pallor may occur after SC/IM injection of epinephrine. Other adverse effects are nausea and vomiting, marked rise in BP leading to cerebro-vascular accident (CVA), ventricular tachycardia, angina, myocardial infarction are the hazards of large doses or inadvertent IV injection.

Contraindication: Epinephrine is contraindicated in hypertensive, hyperthyroid and angina patients.

Norepinephrine

Norepinephrine is a pressure agent, NE (NA) is chemically closely related to epinephrine and released (as neurotransmitter) from sympathetic nerves at the target organs and tissues. It differs from epinephrine in its effects that it does not stimulate β_2-sympathetic receptors.

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Pharmacodynamics

Norepinephrine produces widespread vasoconstriction and thus a rise in both systolic and diastolic blood pressure (α_1 effect). Although it does increase stroke volume and coronary blood flow, NE does not effectively increase cardiac output, probably because of the increase in peripheral resistance in the vascular tree.

Direct stimulant effect on heart rate is overcome by reflex baroreceptor-mediated vagal bradycardia. Compensatory vagal reflex stimulation slows the heart, and cardiac output may be decreased.

Pharmacokinetics

- Exogenously administered catecholamines are poorly absorbed by gastrointestinal (GI) tract and do not enter the CNS to any appreciable extent.
- Epinephrine and NE are most often administered intravenously (with caution to avoid cardiac arrhythmias or local tissue necrosis).
- Extensive enzymatic metabolism occurs in the liver. Metabolites are excreted by the kidney.

Therapeutic Use

Norepinephrine has been used in the treatment of various forms of shock, which is usually associated with a very low BP.

> **Note:** Use of this agent in shock is controversial. Further vasoconstriction may be harmful, because it reduces the blood flow in essential organs, particularly the kidney, with troublesome results.

Administration and preparations:

- Noradrenaline (NE, levarterenol) 2–4 μg/min IV infusion.
- Action starts declining within 5 minutes of discontinuing infusion.
- *Don't mix with $NaHCO_3$ in the same bottle (rapid oxidation occurs).*
- Prevent extravasation to avoid tissue necrosis. Now it is rarely used as a pressure agent.

Available drugs: Adrenor, Norad, Vascue, Nordrin 2 mg (base)/2 mL ampoule.

Adverse reaction: Restlessness, tremor, weakness, dizziness, pallor, dyspnea, apnea, precordial pain, CNS stimulation in large diseases, rise of lipid profile and glucose level.

The drug can also increase myocardial oxygen consumption (severe peripheral vasoconstriction), making the heart work harder.

Toxicity means extreme hypertension which can cause hemorrhage including CVA.

Dopamine

Dopamine is the physiologic precursor of NE.

Pharmacodynamics

Activates peripheral β_1 adrenergic receptor to increase contractility and to some extent heart rate. Activates specific pre and postjunctional dopamine receptors in the renal, coronary, and splanchnic vessels to reduce arterial resistance and increase blood flow to kidney, heart and mesentery. Prejunctionally, dopamine inhibits NE release.

- Major neurotransmitter in the CNS.
- Low dose of dopamine has a positive inotropic effect and increases systolic pressure with little effect on diastolic pressure or mean blood pressure.

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Alpha adrenergic receptors are activated at higher doses of dopamine and causes vasoconstriction with a reflex decrease in heart rate.

Pharmacokinetics

Same as epinephrine and NE.

Therapeutic Use

Low to moderate doses of dopamine is effective in treatment of cardiogenic and septic shock. It may be useful as they increase force of cardiac contraction and output, rise of systolic BP, without significant change in heart rate, and diastolic pressure. It is used to treat shock that persists after fluid volume is replaced. Dopamine is used in patients with severe congestive heart failure (CHF) wherein it increases BP and urine flow.

Dopamine stimulates receptors in the renal blood vessels, causing them to dilate and increase both renal blood flow and urinary output. This action is useful since shock often causes a decline in renal function.

Drugs and doses:

- Dopamine, Intropin, Dopacard 200 mg in 5 mL ampoule.
- Adult dose for cardiogenic shock—intravenous infusion is administered into a large vein—initially 2–5 μg/kg/min gradually increased by 5–10 μg/kg/min according to BP, cardiac output, urine output. For seriously ill patients, up to 20–50 μg/kg/min can be administered.
- Children—IV infusion 0.5–3 μg/kg/min has been used to improve renal blood flow.

Note: Use of dopamine in treating children generally not done, because safety is not established.

Precautions: Correct hypovolemia before and maintain blood volume during the treatment.

- Before starting the drug correct hypoxia, hypercapnia, and metabolic acidosis.
- Use low dose in cardiogenic shock due to myocardial infarction.
- Be careful in peripheral vascular disease. Higher doses lead to vasoconstriction, and which may develop gangrene of extremities due to intense vasoconstriction.
- Special precautions for geriatric patients.

Drug interactions: Use with MAO inhibitors, ergotamine, doxapram (respiratory stimulant), guanadrel or some antidepressants result in hypertension.

Hypotension and bradycardia may develop due to use with IV phenytoin. Use with general anesthetics may cause cardiac arrhythmias. Beta blockers may antagonize-cardiac effects.

Adverse effects: Nausea and vomiting, peripheral vasoconstriction, hypotension with dizziness, fainting, flushing, tachycardia, ectopic beats, palpitations, angina pain, headache, dyspnea, hypertension particularly in overdose.

Nursing Responsibilities in Administering Dopamine

1. Assessment

- Monitor BP, heart rate, ECG, pulmonary capillary wedge pressure (PCWP), central venous pressure (CVP), cardiac output, urinary output continuously during administering the drug.
- Significant changes in vital signs or arrhythmias are to be reported. This is necessary for adjusting dose or discontinuation of medication.
- Palpate peripheral pulses and assess appearance of extremities throughout drug administration. If extremities are cold and quality of pulse deteriorates, notify physician.

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- Monitor urine output frequently throughout dopamine administration. Report decreases in urine output promptly.
- Administration rate should be increased if hypotension occurs. If hypotension continues, more potent vasoconstrictors (NE) may be administered.

2. Implementation

This vasoactive drug is potentially hazardous. Checking of the drug order, drug dose calculations and infusion pump setting needs maximum concentration. If needed, second nurse practioner's help can be taken.

Don't confuse dopamine with dobutamine. Store these two drugs separately.

Before starting dopamine therapy, hypovolemia is to be corrected with volume expanders.

Fluid Preparation for Continuous Infusion

Dopamine vials must be diluted before use. About 200–800 mg dopamine can be diluted in 250–500 mL of 0.9% NaCl/5% dextrose solution/5% dextrose saline. Admixed solution is stable for 24 hours. Discard solutions that are cloudy, discolored or contain a precipitate. Premixed infusions are already diluted and ready to use.

Drug Administration

Infusion must be given by infusion pump to ensure precise amount delivered. Rate of administration is based on patient's weight and cardiac condition.

Titrate to patient response, i.e., BP, heart rate, urine output, peripheral perfusion, presence of ectopic activity, cardiac index.

Decrease rate gradually to prevent marked decreases in blood pressure. Extravasation may cause severe irritation, necrosis, sloughing of tissue.

Following steps are needed to avoid this complication:

- Administer dopamine into a large vein
- Frequently assessment of infusion site. If extravasation occurs, affected area should be infiltrated liberally with 10–15 mL of 0.9% NaCl containing 5–10 mg of phentolamine.

Toxicity and Overdose

Infusion should be decreased or temporarily withdrawn if excessive hypertension occurs. Usually, blood pressure comes down with this action, phentolamine may be administered if hypertension continues.

Education to the Patient and Family

- Explain to patient the reason for administering this drug and need for frequent monitoring.
- Instruct patient to inform nurse immediately if chest pain, dyspnea, numbness, tingling or burning of extremities occurs.
- Instruct patient to inform nurse immediately of pain or discomfort at the site of administration.

3. Evaluation

- Blood pressure increased
- Urine output increased
- Peripheral circulation increased

Dobutamine

Dobutamine is a synthetic catecholamine related to dopamine. It is a derivative of dopamine, but not a D_1 and D_2 (dopamine) receptor agonists. It has relatively selective effect on β_1

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receptor. Its prominent action is to increase force of cardiac contraction and cardiac output, without significant change in heart rate, peripheral resistance and BP. Larger doses may increase heart rate and BP. It is used as an inotropic agent in pump failure accompanying myocardial infarction, cardiac surgery and for short term management of severe congestive heart failure.

It is less arrhythmogenic than adrenaline. Administered by IV infusion because of short life (2 minutes). It should be infused via a central vein to minimize peripheral vasoconstriction.

Dobutamine exerts vasodilator effects on both afferent and efferent arterioles and increases renal blood flow. Increases in renal plasma flow and a increase in glomerular filtration rate. It shows dobutamine has a beneficial effect on renal function.

Dobutamine is preferred when there is a need to improve low cardiac output. It is relatively cardio selective at dosages used in clinical practice, with its main action being on beta-1 adrenergic receptors. receptors. Unlike dopamine, it does not have any effect on specific dopaminergic receptors.

Drugs and doses: Cardiject 50 mg/4 mL and 250 mg per 20 mL ampoule. Dobutrex, dobustat 250 mg vial.

Clinically employed dose is 2–8 μg/kg/minute by intravenous infusion. Drug adjustment is done in accordance with the patient's cardiovascular response.

Adverse effects: Sympathetic nervous system (SNS) stimulation of the cardiovascular systems which include cardiac arrhythmias (ectopic heart beats and increased rate), angina, palpitation, and hypertension. Dobutamine increases the risk of cardiac arrhythmias during general anesthesia.

Precautions and contraindications:

- Volume correction is needed before administration of dobutamine.
- Use of this drug is usually limited to coronary care units (CCUs) where ECG, vital signs, cardiac output and pulmonary wedge pressure can be monitored.
- Patients with arterial fibrillation should be digitalized before dobutamine used.
- Extreme caution is necessary to use this drug in patient with recent myocardial infarction.
- It is contraindicated for patients with idiopathic hypertrophic subaortic stenosis.

Nursing Responsibilities in Administering Dobutamine

1. Assessment

- Monitor BP, heart rate, ECG, PCWP, CVP, cardiac output, urinary output continuously during administering of drug.
- Significant changes in vital signs or arrhythmias are to be reported. This is necessary for adjusting dose or discontinuing medication.
- Palpate peripheral pulses and assess appearance of extremities throughout drug administering. If extremities through out drug administration. If extremities are cold and quality of pulse deteriorates, notify physician.
- Monitor urine output frequently throughout dopamine administration. Report decreases in urine output promptly.
- Administration rate should be increased if hypotension occurs. If hypotension continuous, more potent vasoconstrictors (NE) may be administered.

Lab Test

- Monitor potassium assess concentrations during therapy, because hypokalemia may occur.
- Monitor electrolytes dobutamine, blood urea nitrogen (BUN), creatinine and prothrombin time weekly during prolonged therapy.

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Subrata Sarkar PhD was Principal of Government College of Nursing, RG Kar Medical College and Hospital, Kolkata, West Bengal, India, has broad working experience with nursing students in different clinical fields and community settings. She has been reinforcing the philosophy of rational use of drugs among her students and counsels them for safe administration and recording of the drugs. She desires to remain involved in action-oriented research and services in the field of nursing and public health.

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