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Perspective

OVERVIEW OF ANTIHYPERTENSIVE DRUGS

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INTRODUCTION

Antihypertensive medications fall under a number of chemical classes and are used to treat, regulate, or prevent hypertension. Antihypertensive medication types differ from one another both structurally and therapeutically. Due to the fact that they are frequently administered to the general public and that hypertension affects 31% of people worldwide, they are crucial in the practice of anesthesia. Examples of these include ACEIs for heart failure and -blockers for thyrotoxicosis. Therefore, the medicine and its indication both have an impact on how anesthesia is administered. Most people with hypertension have primary (or essential) hypertension, which is hypertension without secondary causes. Controlling arterial pressure, preventing end-organ damage (to the cardiovascular, renal, and cerebral systems), and lowering the risk of early demise are the goals of management. Reducing cardiac output, systemic vascular resistance, or both can lower arterial pressure.

Through altering vascular compliance and reactivity, drugs that manipulate SVR may potentially result in clinical improvement. As an illustration, the -blockers carvedilol and atenolol both

lower arterial pressure when the patient is at rest, but carvedilol is linked to better vascular compliance. By location of action or mechanism, drugs can be categorized.

DESCRIPTION

There are numerous medications in each class, each with slight structural and pharmacological differences that affect the therapeutic and side effects in different ways. Many substances act on many routes rather than having a single, "clean" mode of action. Antihypertensives can be categorized into two major categories. The first category includes medications that either directly or indirectly block the renin-angiotensin system (RAS), such as ACEIs, ARAs, DRIs, and to a lesser extent -blockers. Despite the fact that these medications work through a variety of methods, vasodilatation is their main side effect. The second class of medications reduces intravascular volume by increasing sodium and water excretion or by inducing vasodilation through non-RAS routes, for as with diuretics and calcium channel blockers (CCBs). Through negative feedback, the acts of this second group boost RAS activity, which has the effect of potentiating the effects of medications that target and inhibit the RAS. The right antihypertensive medication depends on which drug classes are most likely to be successful in regulating arterial pressure and avoiding problems such end-organ damage [1-3].

According to current NICE recommendations, RAS-targeting medications should be started as first-line therapy for patients under the age of 55. As these latter patient groups often experience low-renin hypertension, patients over 55 and black persons of African or Caribbean ancestry are initially treated with medications that work through non-RAS pathways. Additionally, there can be strong personal indications or prohibitions for using a specific drug class. Three medication types specifically target RAS pathway nodes. They work to lessen angiotensin II receptor binding or its peptide hormone synthesis. When AT1 G-protein-coupled receptors are activated by angiotensin II, the resulting rise in arteriolar tone and SVR occurs.

Additionally, it results in sympathetic nervous system activation, increased pituitary secretion of adrenocortical aldosterone and antidiuretic hormones. Inhibiting the RAS pathway lowers arterial pressure and SVR. A decrease in aldosterone secretion, which leads to a decrease in renal

salt and water retention, amplifies this effect. When there is negative feedback, the juxtaglomerular apparatus releases more renin. Synthetic, orally taken ACEIs were created as a result of the finding that the Brazilian pit viper's venom, which significantly lowers arterial pressure, inhibits the angiotensin-converting enzyme (ACE).

A metallopeptidase enzyme known as ACE is found mostly in the pulmonary vasculature. The peptide hormone angiotensin I's conversion to angiotensin II is decreased when ACE is inhibited, as does the metabolism of bradykinin into inert molecules. The majority of the therapeutic effects are brought on by the lowering in angiotensin II. Bradykinin builds up, which offers some therapeutic benefits through vasodilatation but also causes a dry cough in susceptible people. As a further concern in renal artery stenosis, ACEIs can induce renal impairment by lowering renal efferent arteriolar tone and, consequently, effective renal perfusion pressure. Agranulocytosis, skin rashes, taste disturbance, and hyperkalemia brought on by decreased aldosterone secretion are among other adverse effects. Angio oedema with possible upper airway obstruction can result from a rare idiosyncratic reaction to ACEIs and can develop years after the start of ACEI therapy. Since ACEIs are linked to birth abnormalities, they should not be used during pregnancy [4,5].

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