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Hypertension Management and Pathophysiology

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Introduction

A major cause of morbidity and mortality is arterial hypertension, which is linked to renal disease, cerebrovascular disease and coronary heart disease. The outcome is influenced by how much the target organs—the heart, brain and kidneys—are affected. Hypertension is the primary cause of 5,00,000 strokes (which result in 2,50,000 deaths annually) and 1,00,000 myocardial infarctions (which result in 500,000 deaths annually), according to North American research.

National studies continue to demonstrate that hypertension is frequently undertreated and frequently misdiagnosed. It appears that only 25% of hypertensive patients control their blood pressure. One good illustration of this is isolated systolic hypertension. On the other hand, isolated systolic hypertension becomes more prevalent with age. In fact, isolated systolic hypertension, as opposed to systolic and diastolic hypertension [1], affects 20% of people under the age of 40, 80% of those 60–69 and 95% of those over 80. As systolic blood pressure is an excellent predictor of coronary and cerebrovascular risk, particularly in the elderly, the risk of systolic hypertension is becoming more widely recognized. Systolichypertension treatment, with its wide heartbeat pressure, is powerful as far as pulse control and decreased grimness, particularly in older people with a high gamble profile [2].

Description

The treatment of hypertension has evolved over the last decade, with the awareness that there is no safe level of blood pressure below which health is jeopardised. Recent guidelines, such as those issued by the British Hypertension Society, emphasise the need of treating isolated systolic hypertension as well as systolic and diastolic hypertension. Hypertension should now be treated if it rises above 140/90 mm Hg to avoid long-term consequences. Treatment of isolated systolic hypertension (systolic 140–159 mm Hg, diastolic 90 mm Hg) lowers the prevalence of left ventricular hypertrophy, which is a predictor of future morbidity and mortality in Stage 1 hypertension. There is also a 42 percent lower risk of stroke and a 42 percent lower chance of dementia [3].

According to the Hypertension Optimal Treatment (HOT) study, the goal of treatment is to lower blood pressure to 140/85 mm Hg. High normal blood pressure (130–139/85–89 mm Hg) develops to Stage 1 hypertension (>140/>90 mm Hg) in >37 percent of those under the age of 64 and >49 percent of people over the age of 65. The British National Formulary advises taking the following steps: Blood pressure >220/>120 mm Hg: rapid treatment; blood pressure 200–219/110–119 mm Hg: confirm for 1–2 weeks, then treat; or blood pressure 160–199/100–109 mm Hg: confirm for 3–4 weeks, then treat Over a 10-year period [4], the cumulative incidence of first cardiovascular events in patients with high blood pressure is 10% in males and 4.4 percent in females. Even high blood pressure in the normal range is linked to an increased risk of death from coronary or cerebrovascular events. It's unclear whether treating high-normal blood pressure will prevent cardiovascular events [1-3].

Blood pressure control is important. Blood pressure regulation is a complicated topic that will only be briefly discussed. Control of the nervous system The nucleustractus solitarius in the dorsal medulla (baro-receptor integration), the rostral section of the ventral medulla (pressor area) and other centres in the pons and midbrain make up the vasomotor centre. The arterialbaroreceptors increase afferent impulse activity in response to vessel wall distension. This, in turn, reduces efferent sympathetic activity while increasing vagal tone. Bradycardia and vasodilation are the end results [4,5].

Conclusion

The renin-angiotensin system is a hormone that regulates blood pressure. Angiotensin is cleaved by the enzyme renin into the inactive peptide angiotensin I. The angiotensin-converting enzyme converts the latter into an active octapeptide called angiotensin II (ACE). Though the renin–angiotensin system is found throughout the body, the juxtaglomerular apparatus of the kidney is the primary source of renin. The renal perfusion pressure and salt concentration in the distal tubular fluid are sensed by this equipment. Renin release is also increased by b-adrenoceptor activation and inhibited by a-adrenoceptor stimulation. A negative feedback loop occurs when high levels of angiotensin II decrease renin secretion. Angiotensin II causes smooth muscle contraction and the release of aldosterone, prostacyclin and catecholamines via acting on particular angiotensinAT1and AT2receptors. The renin–angiotensin–aldosterone system is involved in the regulation of arterial pressure, as well as the sodium balance.

Acknowledgement

None.

Conflict of Interest

None.

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