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Pathophysiology and Treatment of Hypertension

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Introduction

Because of its links to coronary heart disease, cerebrovascular disease, and renal disease, arterial hypertension is a major source of morbidity and mortality. The extent to which the target organs (heart, brain, and kidneys) are affected influences the outcome. According to research conducted in North America, hypertension is a primary cause of 5,00,000 strokes (2,50,000 fatalities) and 1,00,000 myocardial infarctions (500 000 deaths) per year.

National studies continue to show that hypertension is frequently undiagnosed and, when diagnosed is frequently undertreated. Only 25% of hypertensive patients appear to have their blood pressure under control. Isolated systolic hypertension is a good example of this. Isolated systolic hypertension, on the other hand, becomes more common as people get older. Indeed, the proportion of people suffering from isolated systolic hypertension (as opposed to systolic and diastolic hypertension) rises from 20% in the under 40s to 80% in the 60–69s, and to 95% in those over 80. The risk of systolic hypertension is becoming more widely recognised, as systolic BP is an excellent predictor of coronary and cerebrovascular risk, particularly in the elderly. Systolichypertension treatment, with its wide pulse pressure, is effective in terms of blood pressure control and reduced morbidity, especially in elderly individuals with a high risk profile.

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.

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, 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

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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.

Conflict of Interest

None

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