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BACK TO BASICS: TREATMENTS FOR HEART FAILURE

Sacubitril/valsartan
- Sacubitril inhibits action of the enzyme neprilysin to enhance the beneficial effects of ANP and BNP
- Valsartan, an ARB, blocks angiotensin II receptor sites to limit activation of the RAAS and prevent vasoconstriction and sodium and fluid retention by the kidneys

ANP and BNP counteract negative effects of RAAS to promote vasodilatation and natriuresis, and inhibit fibrosis and hypertrophy

Ivabradine
- Slows impulses from the sinus node in the right atrium to lower heart rate
- Recommended for people with systolic HF and a regular heart beat ≥ 75 bpm, either in combination with standard therapy or if a beta-blocker is not tolerated

Baroreceptors in the arch of the aorta monitor blood pressure. If the BP drops they trigger the sympathetic nervous system to raise the BP again

Liver
produces angiotensin

Kidneys

Diuretics
- Reduce sodium reabsorption by the kidneys to increase loss of sodium and water in the urine
- Lower workload on the heart, and reduce oedema to ease breathing
- Titrate according to need following initiation of other HF therapies

ANP and BNP produced by atria and ventricles respectively in response to myocardial injury/overload

Beta-blockers
- Block the beta-adrenoreceptors in the heart to limit the effects of the sympathetic nervous system
- Slow the heart rate to reduce arrhythmia and BP

Failing heart with BP dropping

Angiotensin

Renin

If the BP drops – even slightly – the kidneys produce renin. Renin sets off the hormonal cascade to raise BP so the kidneys receive sufficient blood to maintain good renal function

As angiotensin I passes through the lungs, ACE converts it into angiotensin II

Lungs

Aldosterone

Spironolactone
- Potassium-sparing diuretic that acts as an aldosterone antagonist
- Prevents fluid retention caused by aldosterone
- Monitor potassium carefully

The sympathetic nervous system stimulates the beta-adrenergic cells, causing the heart to beat faster and stronger to increase the BP

ACE inhibitors
- Inhibit the conversion of angiotensin I to angiotensin II
- Reduce vasoconstriction and production and retention of aldosterone, so reducing BP and the demands on the heart

The sympathetic nervous system stimulates the alpha-adrenergic receptors in the blood vessel walls, causing the vessels to constrict, reducing the volume the blood has to fill, so increasing the BP

Blood vessel

Angiotensin II causes blood vessels to constrict, reducing the volume the blood has to fill and raising the BP

Adrenal glands
Angiotensin II stimulates the release of aldosterone. This travels to the kidneys and increases the retention of fluid, increasing blood volume, and so raising BP

Eplerenone
- Selective aldosterone antagonist that is more specific than spironolactone with fewer side-effects such as gynaecomastia
- Prevents fluid retention caused by aldosterone
- Monitor potassium carefully

Cardiac resynchronisation
- Also called biventricular pacing
- Helps both sides of the heart to contract simultaneously, so improving the pumping ability of the failing heart

Heart transplant
- Indicated for selected patients with advanced HF who no longer respond to other therapies
- Selection based on the patient's clinical need and their capacity to benefit
- Some patients may be 'bridged' to transplantation by a left-ventricular assist device (LVAD)

ARBs
- Block the angiotensin II receptor sites in the adrenal glands and the blood vessels
- Limit activation of the RAAS to prevent vasoconstriction and sodium and fluid retention by the kidneys

KEY
ACE = angiotensin-converting enzyme
ACEI = angiotensin-converting enzyme inhibitor
ARB = angiotensin receptor blocker
ANP = atrial natriuretic peptide
BNP = brain natriuretic peptide
BP = blood pressure
HF = heart failure
RAAS = renin–angiotensin–aldosterone system

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