BETA-BLOCKING AGENTS

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Beta blockers prevent the action of catecholamines on beta receptors by competitive inhibition. Adrenoreceptors are of two types-alpha and beta. Type 1 beta receptors are in the heart muscle and type 2 in bronchial and vascular smooth muscles. They are situated in the cell membrane and perhaps belong to adenyl cyclase system.

Beta blockers are found to be very effective lor anginal pain in most of the cases but 20% patients do not respond to these drugs perhaps due to alpha vasoconstriction (Opie, 1980). Heart rate should be 50-60 beats per minute for these drugs to be effective. This can be obtained by combining beta blockers with nitrate vasodilators. In Prinzmetal's variant angina beta blockade may even be dangerous because of unopposed tone maintained by alpha receptors in large coronary arteries (Yasue et al., 1978).

Beta blockers have no place in the treatment of myocardial infarction but if the patient is already on them, the therapy should be continued except in the presence of extreme bradycardia and low cardiac output to avoid the risk of withdrawl rebound. In post infarction period Aloprenolol 200 mg twice a day has been found to be effective by Wilhelmson et al (1974) but proctalol which reduces the risk of sudden death cannot be used indefinitely because of its side effects. The value of these agents in reducing J the infarction rate is uncertain.

Beta blocking agents are particularly suitable for treating young hypertensives with associated angina, only 20% of older patients with hypertension respond to these therapeutic agents. Combination of beta blockers, vasodilators, me-thyldopa and alpha blocking agents have been found fairly effective in - the management of hypertension. Treatment with a beta blocker or a diuretic should be started first but if the side effects of the former appear the latter or a vasodilator should be added so that the close of the beta blocker can be reduced.

Beta blockers are also used for supraventricular and ventricular tachycardias, cardiomyopathies and congenital heart disease and in thyrotoxicosis alone or with antihypertensives, or radioine before surgery (Zonszein et al., 1979).

Somatic manifestations of anxiety like palpitation and sinus tachycardia respond to beta- blockers and they also inhibit platelet aggregation and are thus beneficial for angina at rest (Opie, 1980).

Beta blockers arc contraindicated in left ventricular failure heart blocks and acute myocardial infarction unless facilities for careful monitoring are available. Their use should also be avoided in severe bronchospasm, depressive states, gangrene, skin necrosis and intermittant claudications. These drugs should be cautiously used in diabetes mellitus, renal failure, liver disease, pregnancy, hypertension, surgical operations and in elderly subjects. Bradycardia due to beta blockers can be controlled by the intravenous use of 1-2 mg of atropine or slow administration of orciprenaline. Infusion of glucagon (2.5-7.5 mg/hr) stimulates the formation of cyclic AMP without affecting beta receptors. Dobutamine in doses of 15 ug/kg per minute overcomes the beta blockade. In the absence of ischaemic heart disease infusion of isoprenalinc can also be used (Opie, 1980).

References

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