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New Aspects in the Treatment of Arterial Hypertension

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The Therapeutic armamentarium began in 1950 and 1951, when Hydralazine, a arteriolar vasodilator, and Reserpine, a sympathetic depressant agent, appeared for the Treatment of Hypertension. In 1953 the mercurial diuretics, appeared for the treatment of Hypertension. Before 1950 the treatments were based on low sodium diet such as the Kempner diet, which were not very effective in the control of hypertension. At that time, severe hypertension and malignant hypertension were condemned to dye from the complications at the target organ levels: Brain, Heart, Kidney and Vessels. This lack of effective drugs in the treatment of hypertension conducted the Scientists to find more effective treatments for this disease.

The appearance of beta blockers in 1962 with pronethalol and later with propranolol, as anti-ischemic drugs, was an important hitus in the therapy. Brian Pritchard at that time, demonstrated that Propranolol was an effective antihypertensive drug along the anti-ischemic properties. The famous paper of William Pettinger in which he demonstrated that the triple therapy bases on Hydralazine, propranolol and diuretics was an effective therapy for severe and resistant hypertension. We also demonstrated that such a combination was also effective in the severe patients with arterial hypertension. During our training at the Emory University, Atlanta, Ga, USA, we had the opportunity to use a nobel compound from Upjohn.

Company, Kalamazoo, Michigan, USA with the name of minoxidil. We began by treating the patients with Minoxidil in doses of 2.5 mg t up to 10 mg combined with furosemide and beta blockers. This triple combination was very effective in severe and malignant hypertension with some secondary problems such as hirsutism and tachycardia. The tachycardia was due to the activation of the adrenergic receptors at the terminal endings. This hyperreactivity on the sympathetic tone was easily counteracted with beta blockers such as propranolol. There was a salt retention on the kidneys and this could controlled with use of Furosemide, however, we noted that furosemide at very high doses induced loss of hearing in the patients, which disturbed the daily life of the patients. We have to recognize that the triple combination of minoxidil, propranolol and furosemide rapidly controlled severe hypertension and resistant hypertension. Pettinger and our Clinical Pharmacology Unit in Atlanta, Georgia, almost at the same time we demonstrated this good alternative therapy to conventional therapy.

When we returned to our Country, Venezuela, in the recent creation of the clinical pharmacology Unit, we continued the research of the pharmacotherapy of hypertension.

For example, we proposed to use clonidine or guanfacine as an alternative agent to counteract the reflex tachycardia induced from minoxidil, although for a different mechanism. By stimulating the alpha adrenergic receptors at the central level, we found that these

compounds such as clonidine and guanfacine, also antagonize the increased sympathetic activity induced by minoxidil. In our experience mostly all patients responded to this effective therapy, that is, minoxidil plus clonidine or guanfacine plus furosemide. However only in one patient who was a woman we have to discontinue minoxidil due to the hirsutism appearing in her face.

Between 1980 until now these new compounds to block the angiotensin aldosterone system and thus angiotensin II, have appeared, first, the direct renin inhibitors among them Saralasin was the most studied, but the problem was the intravenous administration needed to obtain hypotensive action. After Saralasin, we explore numerous compounds such the angiotensin converting enzyme inhibitors or ACEI and also the angiotensin II inhibitors. Among the ACEI we have lisinopril, enalapril, perindopril, cilazapril, etc. and among angiotensin II inhibitors we have telmisartan, losartan, candesartan, irbersartan, etc. For telmisartan, we believe that in addition to the hypotensive properties, it also has antidiabetic action.

It is very important that we have, in addition to Pharmacotherapy for hypertension, we need to use other alternative treatments which they could have beneficial effect, especially in the mild hypertensives, such as low sodium diet, decrease of weight, isometric exercises, yoga, etc. I emphasize that before pharmacotherapy we have to initiate alternative measures for decreasing blood pressure. Sometimes only the alternative measures is enough to control the mild hypertensives.

However we have to precise that if the hypertensive patient is obese or overweighed, the first and important measure to use is the reduction of weight and to ask the patient to decrease the Body Mass Index to 24,9.

Hypertensive syndrome is commonly accompanied by diabetes mellitus, Hyperinsulinism and Blood lipids elevation. We call this syndrome, as metabolic syndrome or cardiometabolic syndrome because in the majority of cases the heart is always affected. The metabolic syndrome is very common, in the general population 25 to 35% suffers from metabolic syndrome, 6 to 8 % suffers from diabetes. And hypertensive disease could reach as high as 45 to 50% of the adult population. When we find a patient with hypertension and diabetes, the Probability of complications on target organs such as brain, heart, kidney and vessels is very high. Only the good the therapy for hypertension and diabetes mellitus could prevent from complicating the organs.

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How we treat a Hypertensive Patient?

If the patient is suffering from respiratory problems, do not use beta blockers. If the patients suffers from prediabetes or hyperinsulinism or diabetes mellitus do not use thiazides diuretics. Right now we count with beta blockers and vasodilatory properties such as carvedilol.

That can be used in the presence of heart failure because the compound induces vasodilatation and the cardiac output usually improves.

Initially we can begin with diuretics or beta blockers or vasodilators including calcium antagonists and compounds antagonizing the angiotensin II receptor such as candesartan, losartan, etc.