

# Heart Failure and Niacin

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## Introduction

Until the Angiotensin Converting Enzyme inhibitor (ACE) drugs came on the market in the late 1970s, the prognosis for patients with congestive heart failure was bleak. Death would occur within five years at the most.

I explain the mechanism of heart failure to patients in the following terms: The heart consists of two pumps, the right pump which takes the blood from the body to convey it to the lungs to give up the waste gases and pick up oxygen, and the left, which receives the blood from the lungs to pump it around the rest of the body. That the two pumps are joined together is an aspect of the evolution of the heart. In engineering terms, the output of one pump ought to equal the output of the other since the circulation is a closed loop. Reality is not as perfect as that and there is a mismatch for which there are compensating mechanisms. The main one is the lymphatic system.

If, for some reason, the mismatch becomes too great then blood backs up. Whenever this occurs, the water in the blood seeps out into the tissues. If it is the right side of the heart which is not pumping efficiently enough then blood backs up somewhere in the rest of the body, usually the lower limbs (because of gravity), causing swelling. If it is the left side, then the blood backs up in the lungs making the lungs waterlogged. It is the water logging of tissues which is responsible for the symptoms of heart failure. The situation is even worse when both sides of the heart fail. If that cannot be relieved then it imposes an increased load on the heart, worsening the situation and causing death.

The standard drugs -- diuretics (water pills) and digoxin (originally derived from extract of the leaf of foxglove as introduced to medicine by William Withering over a quarter of a millennium ago) -- were useful over the short term, and are still a part of the usual armamentarium, but had no influence over the long term outlook. The diuretics worked by increasing the output of urine from the kidneys, which, in turn, encourages the excess tissue fluid to return to the blood for delivery to the kidneys, lowering the load upon the heart. Digoxin, by mechanisms which are still unknown, promotes increased force of the heart contraction. Neither diuretics nor digoxin reduce the resistance in the arterial circulation to output of the heart, something which we now know, since the coming of the ACE drugs, to be a very important factor in the survival of the patient. The term used is "after-load".

## Case Report

In the late 1970s an eighty-year-old patient presented in my office with the full picture of left heart failure. He complained of shortness of breath, aggravated by exertion. His blood pressure was not elevated but he had a sinus tachycardia. He had little dependent edema; his jugular venous pulse was not elevated and he had no hepatjugular reflux. There were moist crackles at his lung bases.

A family doctor in another community from which the patient had moved had established the diagnosis of left heart failure. The dose of digoxin had been established by regular blood tests, which had also shown no abnormalities in his renal function or electrolyte levels caused by the prescribed loop diuretic Furosemide (Lasix).

Nevertheless it was clear to both the patient and myself that his heart failure

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was not under adequate control. He had also been seen by a cardiologist who had nothing further to offer.

The patient was afraid of dying imminently. Recently I had treated a patient with acute pulmonary edema with transdermal nitroglycerin to induce vasodilatation and unloading of her heart. The result had been a brisk diuresis and relief of her acute shortness of breath within an hour. But the effect had been temporary since I did not know then that nitroglycerin was only effective if limited to twelve hours per twenty-four. The lesson which I had learned from this patient was that vasodilatation helped left heart failure, of which acute pulmonary edema is the most extreme and usually lethal variety.

I also knew that niacin, among its many other benefits, was a vasodilator. I explained this to my elderly patient. He agreed to try niacin in the full one gram dose three times per day after meals. He knew that the niacin flush would occur, and, when it did, he put up with it. Of course it became barely noticeable within a week. Within two weeks his heart rate dropped to within normal limits. The crackles in his lungs no longer were audible. Months went by and then years. At the age of eighty-eight he did die, but it was not because of his heart failure.

## Discussion

Niacin (Nicotinic acid, a possibly prebiotic molecule) has multiple roles in medicine as more than a "mere vitamin" used in small doses in the prevention and therapy of pellagra. It ought to be recognized as the treatment of choice in the dyslipidemias, being demonstrably more cost effective than the popular statin drugs, and certainly much safer with a mortality rate in large doses of zero. Its role in psychiatric illness is well known to the readers of this journal. It is life saving in cholera. Along with ascorbate, and other nutrients, it prolongs survival

and improves the quality of life for patients with cancer. It ameliorates allergies by exhausting the stores of vaso-active substances in mast cells, the basis of the "flush". With prolonged use it heals peptic ulcers. It may have a role in the therapy of migraines.

It does not cause diabetes but may reduce glucose tolerance in diabetic patients. Equally it does not cause gout but can increase the frequency of gouty attacks in patients who do not take Allopurinol. Prolonged use of niacin occasionally causes minor abnormalities of liver function tests, but none of clinical significance, and with a mortality rate due to liver failure of, again, zero.

This case illustrates one application of niacin's vasodilating properties. For example it is a mild antihypertensive agent. It produces significant improvement in patients suffering from peripheral vascular disease whether due to atherosclerosis or Raynaud's phenomenon.

While the ACE inhibitor drugs and the Angiotensin Receptor Blocking (ARB) drugs are very widely used in heart failure to reduce afterload in patients with heart failure, this case has illustrated that the same effect may be achieved by the use of niacin, and more cheaply.