

## Pseudohypotension in a Patient with Malignant Hypertension

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Key words: pseudohypotension, malignant hypotension, shock

IMAJ 2000;2:484-485

Pseudohypotension is a syndrome in which indirect blood pressure measurement by sphygmomanometer overestimates true intraarterial blood pressure, due to arterial medial sclerosis and calcification [1]. In pseudohypotension, non-invasive measurement of the peripheral blood pressure underestimates true central pressure due to obstructive arterial disease. We report a case in which malignant hypertension was masked by pseudohypotension.

### Case Description

A 46-year-old man was referred to the emergency room because of severe generalized weakness and confusion that had intensified during the previous several hours. The patient was a heavy smoker and suffered from chronic obstructive lung disease, non-insulin-dependent diabetes mellitus, coronary artery disease, peripheral vascular disease, and end-stage renal disease necessitating regular hemodialysis. His regular medications included metformin, glibenclamide, bisoprolol, aspirin, furosemide and erythropoietin.

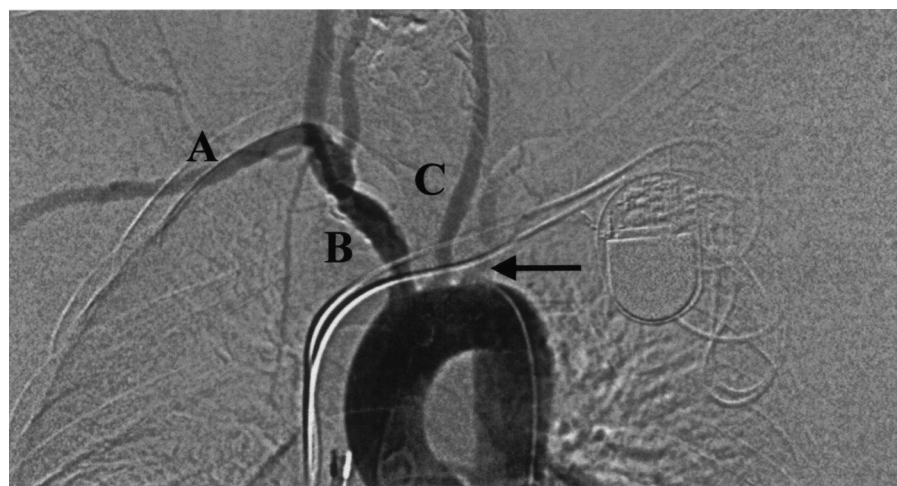
On examination the patient was somnolent, afebrile and not in respiratory distress. The heart rate was 30 beats/minute, and a weak arterial pulse could be palpated over the dialysis shunt in the right antecubital fossa, the left brachial artery and over both femoral arteries. The peripheral pulses were absent and the extremities were cold and cyanotic. Blood pressure in the left arm was 60 mm Hg, without pulsus paradoxus. Blood pressure in the right arm was not measured with a sphygmomanometer

to avoid damage to the shunt. Bruits were heard over both carotid and femoral arteries. The jugular venous pulse was elevated 8 cm above the sternal angle, inspiratory crackles could be heard over both lung bases, and the heart sounds were distant. The examination, including a neurological assessment, was otherwise unremarkable.

The electrocardiogram showed a nodal rhythm of 30 beats/minute and left ventricular hypertrophy. The chest X-ray revealed some prominence of interstitial markings. The serum sodium was 139 mmol/L, potassium 5.6 mmol/L, glucose 95 mg/dl (5.3 mmol/L), blood urea nitrogen 50 mg/dl (17.7 mmol/L), creatinine 2 mg/dl (177  $\mu$ mol/L), carbon dioxide 20 mmol/L, arterial pH 7.36, arterial  $pO_2$  80 mmHg, and hematocrit 35%. A pulmonary ventilation/perfusion scan revealed no segmental perfusion defects.

The patient was treated with intravenous fluids, atropine, dopamine and norepinephrine. He became unresponsive to verbal commands. The pulse could no longer be palpated in the left arm, but was felt weakly over the shunt. Echocardiography revealed a left ventricle with normal size and function. The right ventricle was enlarged with severe tricuspid regurgitation. A small pericardial effusion was demonstrated without evidence of tamponade. A balloon flotation catheter revealed a systolic blood pressure of 100 mm Hg in the right ventricle and pulmonary artery. The pulmonary capillary wedge pressure was 20 mmHg. An arterial line inserted into the femoral artery revealed a systemic blood pressure of 300/100 mmHg. The cardiac output estimated according to the Fick method was 1.5 L/min.

Vasopressor therapy was stopped and the intraarterial blood pressure gradually decreased to 200/70 mm Hg,



Aortic arch angiogram demonstrating the right subclavian artery [A], the innominate artery [B], and the left common carotid artery [C]. The left subclavian artery (arrow) has a subtotal occlusion at its origin from the aorta.

while the patient's neurological status improved. The bradycardia persisted and non-invasive measurement of the blood pressure in the left arm showed 80/40 mm Hg. Angiography of the aorta and distal vessels revealed a subtotal occlusion of the left subclavian and severe stenosis of the right subclavian arteries [Figure]. Both the lower limb vessels were severely stenosed beyond the common femoral arteries.

### Comment

The patient's complaints and physical findings produced a clinical picture indistinguishable from shock. The patient was mistakenly diagnosed as hypotensive because the blood pressure measured in the left arm did not reflect his systemic blood pressure. Treatment with vasoconstrictors worsened the central hypertension and pe-

ripheral hypoperfusion, and caused further neurological deterioration.

The invasive hemodynamic study revealed the correct diagnosis of malignant hypertension [2]. Severe arterial occlusive disease can result from a number of causes, such as Takayasu's arteritis [3], and is associated with hypertension. In view of the patient's risk factors, it is likely that his condition was caused by cardiovascular atherosclerosis. The patient probably had chronic unrecognized hypertension, perhaps due to renovascular stenosis. The etiology of the bradycardia was not discovered, and a permanent dual-chamber cardiac pacemaker was later implanted after hemodynamic studies demonstrated a doubling of the cardiac output during temporary pacing.

The entity of pseudohypotension is probably under-recognized and should

be kept in mind when evaluating the hemodynamic status of patients at risk for occlusive arterial disease.

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