FALSE blood pressure recordings result from many causes, including the auscultatory gap, arrhythmias, partial obstruction of the brachial artery, pulsum paradoxus, shock, obesity, and errors in technique.¹

Another cause of false blood pressure readings is Mönckeberg arteriosclerosis of the brachial arteries severe enough to prevent compression by the blood pressure cuff. However, we had never observed this previously. Our search of the literature yielded no references for this entity. Therefore, we report the following case.

Report of a Case
An 82-year-old, alert white man was admitted to the Veterans Administration Hospital, Dayton, Ohio, on May 3, 1973, complaining chiefly of abdominal pain and diarrhea of seven days' duration, and of shortness of breath for 15 to 20 years.

The patient had previously been told that he had high blood pressure. He denied having chest pain, paroxysmal nocturnal dyspnea, or peripheral edema. A hospital summary from an admission in 1968 recorded one blood pressure reading of "300!" and a diagnosis of hypertensive cardiovascular heart disease prior to the time. The patient was discharged to the care of his private physician, who prescribed digitalis and antihypertensive treatment.

On admission, the most striking observation made by the house staff was that the blood pressure could be recorded only by palpation and exceeded 300 mm Hg in both arms. This finding was consistent even though various types of cuffs and manometers were used. Senior house physicians confirmed this phenomenon. Both brachial and radial arteries were easily palpable and pliable. Venous pressure was not elevated. A presystolic gallop and grade III funduscopic hypertensive changes were recorded. Additional positive findings included 2+ pitting edema of both lower legs, bilateral moist basilar rales, and a coarse grade III/V ejection-type aortic murmur with a diminished A₂ sound over the aortic area. Electrocardiogram showed a left anterior hemiblock, right bundle-branch block, and first degree atrioventricular block. Chest film demonstrated generalized cardiomegaly, a densely calcified left pleura, and bilateral interstitial fibrosis with honeycombing. Blood urea nitrogen and electrolyte levels were within normal limits.

On his first night of hospitalization, the patient was treated with furosemide and triamterene because of the tentative diagnosis of hypertensive cardiovascular heart disease, aortic stenosis, and heart failure. The following morning the blood pressure reading noted by the house staff was confirmed by the attending physician.

Roentgenograms of the right and left arms show heavily calcified brachial arteries on both sides. No calcification is detectable at elbow area.

However, by then the lung fields had cleared, the gallop was not detectable, and only a trace of edema persisted. The reported abnormalities in the fundus were not confirmed. Both stethoscopic and sonor blood pressure recording apparatus were tested, but failed to record any pressures. On recommendation of the attending physician, methyldopa (500 mg four times a day) was added to the treatment regimen. However, when he was seen in consultation early that afternoon because of the difficulty the nurses were having recording blood pressures, it was then recognized that all readings were artificial. Blood pressure taken in the thigh, although poorly auscultated, was estimated at about 160/90 mm Hg. Cuffs placed on the forearm or extremely high on the arm recorded pressures of approximately 140 mm Hg by radial artery palpatory. All drug therapy was discontinued immediately.

Five days after treatment had been discontinued, an intra-arterial needle was placed in the right brachial artery and pressures of 130/57 mm Hg were obtained. Simultaneous palpatory pressures were taken when the arm cuff was applied in the routine manner and again were found to exceed 300 mm Hg. Roentgenograms of both upper extremities showed severe Mönckeberg arteriosclerosis involving both brachial arteries (Figure). No calcification was shown at the elbow area where the artery was readily palpable and quite pliable.

Comment
Pseudohypertension due to severe Mönckeberg arteriosclerosis resulting in "pipe-stem" arteries is a predictable anatomical and physiological phenomenon, but we had not encountered it previously. Numerous physicians, including cardiologists, had taken this patient's blood pressure, and all were misled. The deception was enhanced by the readily palpable and pliable brachial artery at the elbow. Artificial hypertension was clearly a mechanical phenomenon, being caused by such severely calcified brachial arterial walls that the cuff could not compress the arteries.

Nonproprietary Names and Trademarks of Drugs

Furosemide—Lasix.
Methyldopa—Aldomet.
Triamterene—Dyrenium.

Reference