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Artifactual Hypotension Without Damping, a Hazard of Disposable Diaphragm Domes

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Artifactual hypotension is a complication of intraarterial monitoring of blood pressure. The importance of differentiating false from actual hypotension cannot be overemphasized. Misdiagnosis may lead to unnecessary and potentially fatal therapy. 1,2 The most common causes of artifactual hypotension are external vascular compression, mechanical obstruction of the arterial catheter or line, and leaks in the transducer system. All result in damping of the waveform pressure during electronic recording. Accordingly, falsely low blood-pressure readings can often be deduced by observing the damping or deformation of the pulse tracing on the oscilloscope. The effects of transducer domes on the accuracy and frequency responses of arterial pressure monitors have been studied by Fox et al.3

The following is a report of artifactual hypotension related to the use of a disposable diaphragm dome. The characteristic damping of artifactual hypotension was absent and consequently, the recorded pressures were not recognized as being false. This is a unique hazard of disposable transducer diaphragm domes.

REPORT OF A CASE

A 68-year-old, 78-kg man who had had an acoustic neuroma resected the previous day was brought to the operating room in a comatose state for emergency placement of a ventriculostomy. On his arrival in the operating room, a left radial arterial line, as well as central and peripheral venous lines, remained in place from the first operation. The arterial line was connected to a Gould-Statham® transducer (model P23Db) fitted with a Gould-Statham disposable diaphragm dome (model TA1009D). The pressure was recorded on an Electronics for Medicine® multichannel monitor. A good arterial pressure tracing was obtained on the oscilloscope. After electrical calibration, the blood pressure was found to be 220/120 torr, with a mean arterial pressure of 150 torr. This closely approximated the pressure obtained by use of a blood-pressure

Ten minutes later the surgeons had made their incision. The blood pressure recorded on the monitor had decreased to 160/90 torr, with a mean arterial pressure of 110 torr. Five minutes later blood pressure was 90/25 torr, mean arterial pressure 50 torr. The pulse rate was 90/min and regular, central venous pressure $8-9~\rm cm$ H₂O. Breath sounds were bilateral and equal, and peak inspiratory pressure was unchanged. Arterial blood-gas analysis showed P₀₄ 137 torr, P_{C04} 27 torr, pH 7.48, and a base deficit of 4. The arterial pressure tracing was unchanged in shape. Inspection of the patient's left arm revealed no compression of the brachial or radial arteries. There was no kink or bubble in the arterial line, and no back bleeding was observed; flushing produced no change.

The blood pressure remained in the 80/15-95/30-torr range, with mean arterial pressure 40-55 torr, over the next 5 min. Blood loss had been minimal, urinary output continued, and the extremities remained pink and warm. Despite this good clinical appearance, intravenous fluid administration was increased. Possible causes of the hypotension that were considered included intracranial hypertension, cardiogenic shock, and the combined effects of relative hypovolemia and hydralazine. While a dopamine drip was being prepared, a manual blood-pressure cuff measurement revealed a pressure of 160/95 torr. The transducer recording was 80/15 torr, with an unchanged pulse tracing. Electrical recalibration did not change this value. At that point, one of the anesthetists discovered that the disposable transducer dome was loose. One full turn to tighten the seal between the dome and transducer head did not alter the arterial waveform, but resulted in an elevation of the tracing on the calibrated oscilloscope screen. The blood pressure by the monitor was then 160/95 torr, mean arterial pressure 115 torr. Manual cuff measurement again showed 160/95 torr. By loosening the diaphragm dome, a falsely low pressure could again be reproduced on the monitor, but without any leak of heparinized solution or blood from the transducer and dome. Retightening the dome returned the recorded pressure to its original reading. The remainder of the procedure was uncomplicated.

Discussion

False arterial blood-pressure readings during direct intraarterial monitoring can have many causes. These may include an intra-arterial thrombus, an indwelling arterial catheter tip resting against a vessel wall, partial occlusion of the cannula by clot, air or kinks in the heparinized line, leaks in the transducer system, external compression of proximal arteries, intrathoracic compression of the left subclavian artery or ascending aorta, or miscalibration of the monitor. False blood-pressure readings with a normal pulse tracing on the oscilloscope or recorder are almost always due to errors in calibration. The true pressure may be higher or lower. On the other hand, falsely

cuff on the opposite arm. Pancuronium, 4 mg, iv, was administered, and controlled ventilation was begun. Anesthesia was maintained with nitrous oxide—oxygen (4 l/2 l/min) using a semiclosed circle system with carbon dioxide absorption. Hydralazine, 10 mg, was then given iv over a 5-min period.

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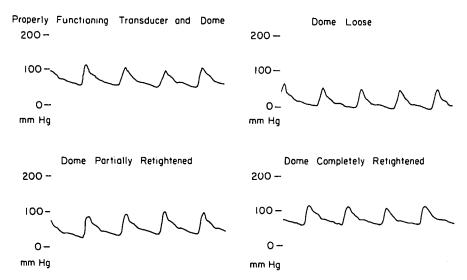


Fig. 1. Artifactual hypotension with a disposable diaphragm dome is simulated in this series of arterial pressure tracings. The left upper tracing is from a properly functioning P23Db transducer and dome. When the dome is loosened (right upper tracing) the tracing falls on the electrically calibrated scale, producing a falsely low blood-pressure reading. Notice the absence of damping. The severity of this artifactual hypotension is proportional to the distance between the transducer diaphragm and dome diaphragm. Thus, when the dome is partially retightened (left lower tracing) the "hypotension" is only partially corrected. Completely retightening the dome (right lower tracing) places the transducer and dome diaphragms in close opposition and returns the blood pressure to its original level.

low readings characterized by damping of the pulse tracing point to vascular compression or problems within the arterial line or transducer. A quick check of the patient's arm, the cannula, the transducing system usually reveals the cause.

Disposable transducer diaphragm domes are a recent development in cardiovascular monitoring. The dome diaphragm prevents direct contact of flush solution or blood with the transducer. Its flexibility permits accurate transmission of the pressure wave to the transducer. The dome diaphragm is in turn separated from the transducer diaphragm by a layer of fluid. Sterilization of the transducer after each use is no longer necessary, since contact between the transducer and the patient's arterial system has been eliminated. This minimizes the risk of patient infection and equipment contamination.

When, during arterial monitoring, the disposable diaphragm dome is loosened, the distance between the dome diaphragm and transducer diaphragm is increased. This results in an inadequate coupling of the dome to the transducer head, and the apparent mean pressure falls as the diaphragm is distended, producing falsely low readings. The difference between the actual and recorded pressures varies directly with the extent of uncoupling. The uncoupling is not sufficient, however, to eliminate the transmission of the pulse waveform to the transducer. Thus, the artifactual hypotension produced is not accompanied by damping of the pulse tracing on the monitor, nor leaking of flush solution from the dome-transducer head connection. The absence of damping and leaking is misleading, and makes detection of an accidentally loosened dome more difficult. Furthermore, the problem is not revealed by calibration against an electric standard. Retightening the dome returns the recorded pressure to its original level. These effects are illustrated by the tracings in figure 1.

We have not attempted to study all available disposable domes for this potential artifact. However, the Bentley Trantec (model D-241) transducer dome was examined, with essentially the same finding: incomplete tightening of the dome affected mean pressure substantially in the absence of waveform deterioration. Perhaps a locking mechanism should be incorporated into the design of these domes to insure a positive coupling between the diaphragm and transducer surface.

In the case reported, artifactual hypotension due to a loose disposable transducer diaphragm dome was not initially detected. The administration of dopamine or other vasopressors might have led to fatal complications in this patient, who had intracranial hypertension. Saka, Liu, and Oka have, in fact, reported a case of artifactual hypotension in which the patient received 5 mg phenylephrine before the cause of the "hypotension" was recognized. All physicians utilizing transducers and electronic monitors for cardiovascular surveillance should be aware of this unique hazard of disposable diaphragm domes.

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