

Artifactual Hypertension Due to Transducer Cable Malfunction

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Invasive pressure monitoring has become an integral part of the management of critically ill patients. Risks of invasive monitoring include improper clinical decisions based on inaccurate measurements. We present an unusual case of a faulty transducer cable causing an artifactual 50% increase in measured arterial blood pressure.

CASE REPORT

A 70-yr-old man with a recent onset of angina was admitted for elective coronary artery bypass grafting. Preoperative cardiac evaluation included a thallium exercise stress test that was consistent with septal, anterior, and inferior myocardial ischemia. A cardiac catheterization demonstrated a 90% proximal left anterior descending coronary artery stenosis and preserved left ventricular function.

Upon arrival to the operating room, peripheral venous access was established, and the right radial artery was cannulated with an 18-G catheter for arterial pressure monitoring. The arterial catheter was attached to a Spectramed DTx/Plus disposable transducer (Spectramed Inc., Oxnard, CA), which was interfaced with a Marquette series 7010 RA monitor (Marquette Electronics, Inc., Milwaukee, WI) via a Spectramed transducer cable. The system was zeroed to atmospheric pressure, and the calibration was checked against a mercury manometer at 100 and 200 mmHg. The arterial blood pressure waveform indicated a pressure of 130/60 mmHg, which was consistent with previous pressures obtained by cuff sphygmomanometry. Other monitors included an electrocardiogram, pulmonary artery catheter, temperature probe, and pulse oximeter. The patient underwent an uneventful bypass graft of the left internal mammary artery to the anterior descending coronary artery under high-dose opioid anesthesia. In the immediate postbypass period, hypertension was treated with intravenous sodium nitroprusside (SNP).

In preparation for transport to the intensive care unit, the radial artery pressure transducer was connected to a Marquette series 7200 TRAM (Transport Remote Acquisition Monitor) using a second Spec-

tramed cable. The transducer was zeroed, and a digital arterial pressure of 180/90 mmHg was displayed along with a corresponding pressure waveform. Since the pressure displayed with the transport monitor was markedly higher than that obtained only moments earlier with the monitoring system used intraoperatively, the transducer was rezeroed to atmospheric pressure, and the waveform was noted to be consistent with an appropriately damped trace. Also, the SNP infusion was examined and found to be infusing properly. Prior to increasing the dose of the SNP or the administration of additional antihypertensive or anesthetic agents, the transducer was reconnected to the monitor and cable used intraoperatively to check for artifactual hypertension due to transport monitor or cable malfunction. A blood pressure of 120/60 mmHg was now obtained and was confirmed by a blood pressure cuff and a second transport monitor (Protocol Propacq 106, Benvoton, OR) with a new Spectramed transducer cable.

The patient was subsequently transferred to the intensive care unit with the Propacq monitor and the new cable and had a satisfactory postoperative course. Later visual examination of the initial transport cable revealed that a pin from a transducer used during a previous case had broken off and had become embedded in the cable (fig. 1).

Using a Biotek Multiparameter Simulator (Biotek Instruments, Inc., Winooski, VT) to simulate a blood pressure of 120/80 mmHg and a Marquette series 7200 TRAM as a monitor, we tested ten randomly chosen Spectramed transducers to attempt to reproduce our intraoperative experience. When using the defective cable, the measured pressure with all ten transducers was 56% higher than that applied to the transducers by the simulator.

A representative of Spectramed, Inc. was notified of the details of this case. Spectromed, Inc. is currently developing a cable that should eliminate this hazard.

DISCUSSION

Direct arterial blood pressure measurement is accomplished using a system consisting of a mechanical coupling device (cannula and tubing), pressure transducer, cable, and amplifier/display unit (monitor).¹ Arterial pressure

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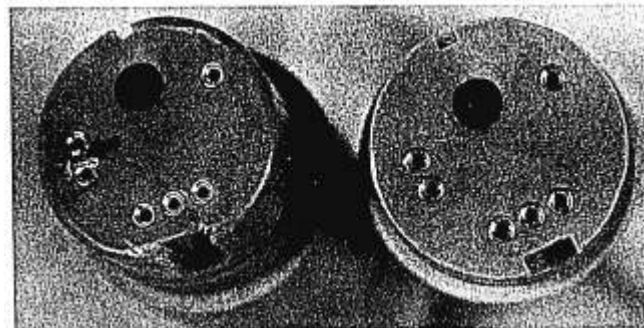


FIG. 1. The defective cable (left) with a transducer pin bridging the two pin sockets (arrow). A new cable (right) is shown for comparison.

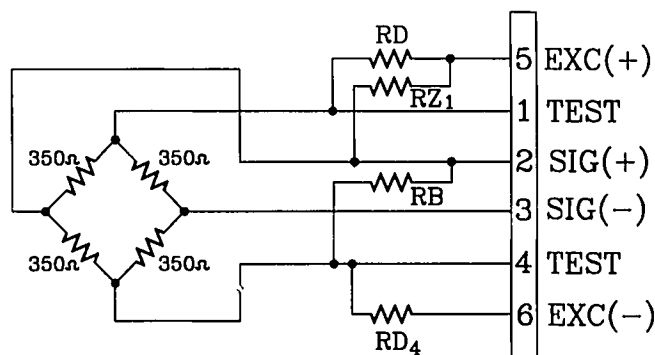


FIG. 2. Transducer circuit. The four resistors that make up the Wheatstone bridge undergo changes in resistance in direct proportion to the applied pressure. The bridge is excited by applying a potential across exc+ and exc-, and the resulting potential difference developed between sig+ and sig- is measured by the monitor and converted to a pressure reading.

is transmitted through the cannula and tubing *via* a solution to a pressure-sensitive diaphragm within the transducer. This pressure displaces the diaphragm, changing the resistances of a series of resistors and consequently the electrical output from the transducer by an amount proportional to the applied pressure (fig. 2). By arranging the resistors into a Wheatstone bridge, a small change in resistance results in a large, easily measured change in output signal to the monitor.^{2,3} The sensitivity of the transducer is defined by the relationship between the applied pressure and the transducer output voltage. The standard sensitivity for biomedical transducers is $5.0 \mu\text{V}/\text{mmHg}$ for each volt used to excite the bridge.[†] Finally, the monitor converts the transducer output voltage to a blood pressure reading based on the assumption that each $5 \mu\text{V}$ per excitation volt corresponds to 1 mmHg.

There are many recognized causes of inaccurate invasive arterial pressure measurements. These include compression, obstruction or spasm of the artery proximal to the point of cannulation, kinks, leaks or air in the pressure tubing, improper transducer zeroing or calibration, and electrocautery interference.⁴⁻⁷

The inaccurate measurement in this case was due to cable malfunction. An embedded pin in the cable end caused a short-circuit between two pin sockets (fig. 1). When the transducer was connected to the cable, pins 4 and 6 (fig. 2) became inserted into these pin sockets and were likewise short-circuited. This eliminated resistor RD_4 from the transducer's circuit. In a properly functioning transducer, resistor RD_4 compensates for variations in bridge sensitivity caused by temperature changes and adjusts the transducer's final sensitivity to $5.0 \mu\text{V}/\text{mmHg}$.

Without resistor RD_4 in the circuit, the transducer's sensitivity increases to $7.7 \mu\text{V}/\text{mmHg}$.

Although it is our practice to check our transducer's calibration against a mercury manometer prior to induction, we do not routinely recheck it prior to transport. Such a check probably would have alerted us to the defective cable, although we found that the short circuit occurred only when the cable and transducer were joined firmly. A loose fit did not result in erroneous readings. It is therefore possible to have artifactual hypertension in a previously calibrated system by simply adjusting the fit between the transducer and defective cable (fig. 3).

Previous reports of erroneous blood pressure readings have involved arterial waveforms that might have been recognized as suspect.⁴⁻⁷ They were damped or had pulse pressures that remained unchanged with large changes in systolic and diastolic pressures. Since the cable in the current case caused a consistent 56% error throughout the cardiac cycle, the shape of the arterial waveform was not affected.

To our knowledge, this is the first report of falsely increased invasive arterial blood pressure measurement secondary to a malfunction of the cable connecting the transducer to the monitor. With the widespread use of invasive monitoring and the potential for significant morbidity from a cable malfunction, we recommend that the

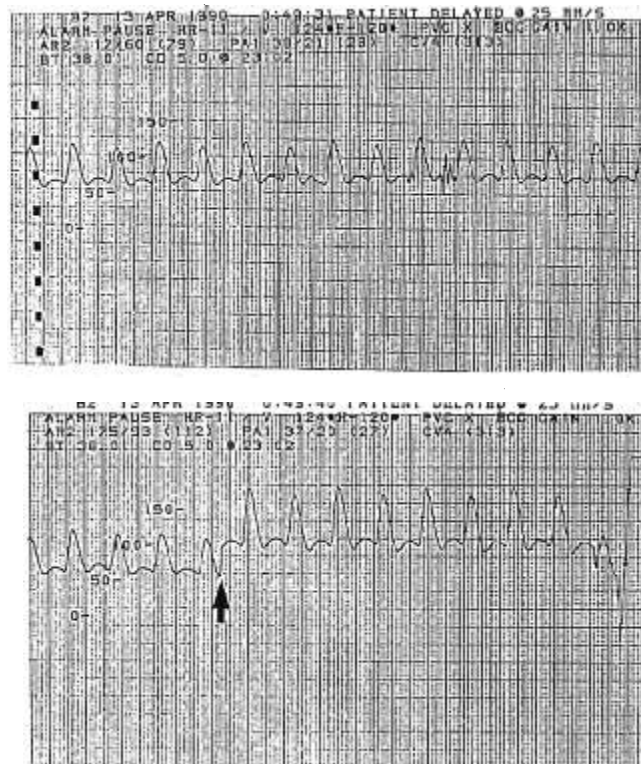


FIG. 3. A continuous blood pressure tracing with the defective cable. An approximately 50% increase in blood pressure reading was achieved by pushing the cable and transducer ends firmly together (arrow).

[†] This is the American national standard for interchangeability and performance of resistor bridge-type blood pressure transducers. Approved November 1986.

following safeguards be routinely used during invasive pressure monitoring. First, the connecting cable should be visually inspected prior to connection to the transducer. Second, after zeroing the transducer to atmospheric pressure, the pressure-monitoring system should be checked against a mercury manometer. Care should be taken to perform the calibration check while the system is not connected to the patient, to avoid air embolus. Finally, the possibility of malfunction of the pressure-monitoring system should always be considered following sudden changes in invasive pressure readings.

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Carbon Dioxide Embolism: Successful Resuscitation with Cardiopulmonary Bypass

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Carbon dioxide embolism is a well-known complication of laparoscopy. We present here a patient who experienced sudden, complete cardiovascular collapse during laparoscopy and hysteroscopy and who was resuscitated with cardiopulmonary bypass. This is the first report in the literature in which an almost certainly fatal CO₂ embolism was treated successfully with cardiopulmonary bypass.

CASE REPORT

The patient was a 28-yr-old woman who presented with a complaint of infertility and who was scheduled for laparoscopy, hysteroscopy, and resection of the uterine septum in our in hospital ambulatory surgery center.

Her past history was negative for any significant medical problems. She had undergone three dilation-and-curettage procedures in the past without any problems. Her only medication was danazol 200 mg three times per day. She had no known drug allergies.

On physical examination, she weighed 78 kg and was 163 cm tall. She had a blood pressure of 120/60 mmHg and a pulse of 85 beats

per min. There were no physical or laboratory abnormalities noted. Preoperative electrocardiogram and chest x-ray were not obtained.

In the operating room electrocardiogram, noninvasive blood pressure (Dinamap[®]) and hemoglobin O₂ saturation (SpO₂) (pulse oximeter) monitoring was established. The patient was preoxygenated with 100% O₂. Following administration of 3 mg *d*-tubocurarine and 100 µg fentanyl, anesthesia was induced with 375 mg thiopental and 150 µg fentanyl. One hundred milligrams succinylcholine was given to facilitate intubation of the trachea, after which an additional 125 mg thiopental was given to blunt the patient's hypertensive response to intubation. End-tidal CO₂ (PETCO₂) monitoring was established, and an esophageal stethoscope and temperature probe were inserted.

Anesthesia was maintained with O₂, N₂O, and isoflurane. Thirty-five milligrams atracurium was given for surgical muscle relaxation. The patient's lungs were mechanically ventilated at a rate of 8 breaths per min and with a tidal volume of 650 ml. Laparoscopy and hysteroscopy were performed simultaneously. The abdomen was insufflated with CO₂ through a Verres needle inserted into the peritoneal cavity. (A Verres needle is a device with a sharp outer needle surrounding an inner blunt tipped cannula with a lateral hole through which gas may be administered.) A 32% solution of dextran 70 in 10% dextrose (Hyskon[®]) via an infusion set (catalog number 003712-901, Cabot Medical Corporation) was used to distend the uterus for the hysteroscopy and resection of the uterine septum.

Approximately 35 min after beginning the procedure and while performing the hysteroscopy, a few minutes after the uterine septum had been divided, the surgeons remarked that the distending pressure of the uterus had decreased and that perhaps the uterus had been perforated. Moments after that, there was a sudden bradycardia, followed by tachycardia of 160 beats per min, followed rapidly in turn by ventricular fibrillation and then asystole. The blood pressure was unmeasurable at that point. At the same time it was noted that there was an abrupt increase in the PETCO₂ (from 31 to 38 mmHg) and a decrease in the SpO₂ to 81%. The peak inspiratory pressure increased

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