Noninvasive blood pressure monitoring is widely employed in the critical care environment for those patients not requiring vasoactive drug titration. All existing devices use a self-inflating bladder and a bladder pressure-sensing (oscillometric) pulse transducer. By sensing the absence of pressure oscillations, the equipment automatically inflates to a suprasystolic pressure. As the cuff automatically deflates, the instrument senses oscillating signals and estimates systolic and diastolic pressures for display or printout. Reported complications in adults have been related to lack of accuracy [1–3], ulnar nerve compression with resulting transient paresthesia [4] and superficial phlebitis [5–7]. We report on a patient with hyperactivity and tremor secondary to lithium toxicity in whom, in the absence of other forearm trauma, volar and dorsal muscle necrosis developed after application of an oscillometric automated sphygmomanometer that apparently malfunctioned. No prior reports of compartment syndrome arising from such a cause were found, although this entity has been reported after the use of tourniquets, constrictive dressings, antishock garments, and pneumatic splints [8,9].

CASE REPORT

A 42-year-old man with a long history of a psychiatric disorder attempted suicide by swallowing 120 g of lithium about six hours before admission. Initial treatment included the administration of activated charcoal and forced diuresis with mannitol and hydration. The patient became increasingly lethargic and began to convulse. The lithium level was 7.0 mmol/L (therapeutic level, 0.5 to 1.4 mmol/L). He was tracheally intubated and mechanically ventilated, and hemodialysis was instituted. The patient was transferred to the intensive care unit, where his blood pressure was initially moni-
tored with the help of an arterial catheter. The arterial catheter was replaced with an automated, self-inflating sphygmomanometer (Sensomat BP, Biochem International, Milwaukee, WI) applied to the right upper arm when his hemodynamic and respiratory status improved. During this period, sporadic seizure activity and nonspecific hyperactivity persisted. The patient was examined daily by the house staff, and on the sixth day after admission, the right forearm was found to be tense, edematous, cool, and cyanotic. The blood pressure cuff was found to be inflated and did not spontaneously deflate during the period of time required to remove it. The audible alarms had been turned off. A period of hyperactivity had been noted by the nursing staff just before this finding. The cuff was immediately removed. It was not possible to determine how many faulty cycles had occurred or the occlusion time, but the monitor had functioned properly at the end of the previous shift about six hours earlier.

The patient’s radial and ulnar pulses were palpable, and Doppler examination of the palmar arch and digital arteries revealed nothing abnormal. Venous sounds over the antecubital fossa were markedly decreased. A venogram showed several regions of thrombosis in the veins of the forearm, extending to the medial aspect of the elbow. There was extensive abnormal collateral circulation but normal filling of the brachial and axillary veins. Pressures were measured with the direct manometric technique by inserting an 18-gauge needle connected with a water manometer into the dorsal and volar compartments of the right forearm. The manometer was adjusted to zero at the level of the forearm. The threshold infranascial pressure at which fasciotomy is usually recommended is 30 to 40 cm H2O. The pressure in the dorsal compartment was 34 cm H2O, and that in the volar compartment was 60 cm H2O. (The pressure in the anterior thigh compartment was less than 10 cm H2O.) Immediate fasciotomies of the right forearm were performed; upon release of the fascial compartments, bulging of the muscles and improvement of the distal circulation were evident. The patient required multiple debridements of necrotic muscle in the deep compartment and had residual paresthesias and limitation of hand motion.

**DISCUSSION**

Compartment syndrome occurs when pressure within a fascially enveloped muscle compartment compromises capillary perfusion. The increase in pressure can be due to a decrease in the size of the compartment or an increase in the fluid content of the compartment [2,10–12]. Trauma, especially that associated with fractures, burns, hemorrhage, posts ischemic edema, limb compression, strenuous exertion, and rattle snake bites, may initiate the pathophyslogic mechanisms that eventually lead to nerve injury and muscle necrosis [12,13]. Drug overdose is often reported as a predisposing factor, usually because of limb compression by other body parts while the patient is obtunded [10–12]. Altered sensorium may also delay detection of compartment syndrome. Usually, diagnosis is based upon the presence of the classic “five Ps”: pain, increased pressure, paresis, pulselessness, and paresthia. Pain, paresis, and paresthia cannot be appreciated if the patient is obtunded, and absence of pulse is a very late sign. Diagnosis rests solely on a high index of suspicion and on compartment pressure measurements.

Review of the operation manual and electromechanical schematics accompanying this particular device indicates an appreciation by the manufacturer that extended periods of inflation may be detrimental. The safeguards incorporated include limitation of cuff inflation time (30 seconds), limitation of cycling time (3 minutes), and limitation of time to deflate (120 seconds). Visible and audible alarms for pressure limits and excess inflation time are provided. In the lethargic patient and the patient under anesthesia, clinical observation and function alarms are mandatory. If within the busy critical care environment alarms are used as an aid, then equipment in which alarms may be permanently silenced is not acceptable. Most equipment permits short-time or one-time silencing of alarms for convenience. In this reported case, however, the equipment allowed permanent bypassing of the audible alarms.

Even in the presence of heightened clinical surveillance, users often mistakenly rely on automated backup of fail-safe systems. In this instance, we theorize that the sensing system of the device permitted hyperinflation or rapid recycling in response to artifacts induced by motion of the patient’s arm. This could have occurred as a result of the fail-safe system, which falsely interpreted movement as continued bladder oscillation and therefore increased bladder inflation pressure and deflation time in an effort to sense systolic and diastolic levels. As a possible consequence of the faulty sensing cycle, recycling was initiated and may have contributed to functional venous occlusion combined with increased muscle oxygen consumption secondary to hyperactivity.

In an attempt to confirm this theory, we applied the device to five normal volunteers who simulated minor arm movement (waving, biceps flexing, rotation, circumduction). Each subject wore a fingertip pulse oximeter taped in position. In all subjects, erratic pressure readings and extended inflation, deflation, and cycling times occurred. Average decreases in saturation from a