REVIEW ARTICLE

Compartment Syndromes: Concepts and Perspectives for the Anesthesiologist

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Background

Compartment syndromes have been discussed infrequently in literature familiar to anesthesiologists (1–4). Reports of compartment syndromes following vascular punctures in patients receiving anticoagulant therapy (5,6), plus recent allegations by plaintiffs that patient positioning can be a factor in causing postoperative compartment syndromes, establish a need to review the pathophysiology of compartmental ischemia and develop perspectives for the anesthesiologist.

Matsen (7) defined a compartment syndrome as a condition "in which the circulation and function of tissues within a closed space are compromised by increased pressure within that space." Dense osseofascial planes in extremities establish relatively unyielding boundaries that compartmentalize groups of muscles, nerves, and blood vessels. Blood flow through such a compartment can be disturbed by remote perfusion failure (vascular obstruction or systemic hypotension) or by increases in resistance to flow within the compartment itself (Table 1). Compartmental resistance may be increased by (a) an external application of pressure that decreases the effective size of the compartment and compresses its contents or (b) by an enlarged mass of compartment contents that crowds against rigid walls and elevates tissue pressure within the space. If compartmental ischemia is prolonged, it eventually initiates the sequence of events listed in Table 2. Neural dysfunction and muscle injury follow. Usually, prompt surgical decompression of the affected compartments is the only means of minimizing the functional impairment and structural damage of their contents.

Remote perfusion failure (Table 1) disrupts driving pressure whether due to systemic hypotension or some form of vascular obstruction distant from the affected compartments. Reperfusion, after the restoration of systemic pressure or the relief of obstruction, extravasates vascular contents through newly defective walls of the microcirculation, elevating compartment pressure. Perler and associates (8) believe that oxygen-derived free radicals, generated from xanthine oxidase during reperfusion, are the factors that damage the microvasculature. Patman (9) has reviewed compartment syndromes associated with peripheral vascular surgery.

Direct compartmental injuries are frequently associated with fractures, surgery, and multiple trauma. However, a wide range of other causes can elevate compartmental pressures (7) (Table 1). Tight dressings, tight surgical closure of fascial defects, or localized external pressure can decrease compartment size. Body parts, resting either against each other or upon a supporting object for protracted periods, may provide enough local external pressure to initiate the syndrome (10). Increases in the mass of compartment contents can result from elevated capillary pressure, low oncotic pressures, increased capillary permeability, trauma, bleeding, exercise of hypertrophic muscles, or infiltrated infusions.

In the presence of systemic normotension, Reneman (11) found that tissue pressures in normal compartments of the supine lower leg ranged from 9 to 15 mm Hg. In two instances of unilateral anterior compartment syndromes, one patient had a tissue pressure of 45 mm Hg in the hypertensive leg compartment and in the other patient the pathologic value was 67 mm Hg (11). Whitesides et al. (12) considered the normal mean arterial pressure in the microcirculation to be in the same range as systemic diastolic pressure and concluded that ischemia may begin at tissue pressures that are 10–30 mm Hg less than diastolic. Ashton (13) found that tissue circulation stopped when compressive compartmental pres-

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Table 1. Causes of Compartmental Ischemia

- I. Remote Perfusion Failure
  - A. Vascular obstruction
  - B. Systemic hypotension
- II. Increased Compartmental Resistance
  - A. Decrease in compartment size
    1. Constriction by tight dressings or casts
    2. Tight repair of surgical wounds
    3. Local pressure
  - B. Increase in compartment volume
    1. Bleeding and coagulopathies
    2. Increased capillary permeability
       a. Reperfusion edema (postischemic swelling)
       b. Exercise (seizures and eclampsia)
       c. Trauma and burns
       d. Intraarterial drugs
    3. Increased capillary pressure
       a. Exercise
       b. Venous obstruction
    4. Decreased oncotic pressure
    5. Infiltrated infusions
    6. Muscle hypertrophy

Modified from Matsen FA. Clin Orthop 1975;113:8.

Table 2. Stages of Compartmental Ischemia

1. Hypoxic disruption of capillary boundaries
2. Escape of intravascular contents into adjacent tissues
3. Swelling (edema) of compartmental contents
4. Increasing compartmental pressure and perfusion impairment
5. Worsening ischemia and tissue necrosis
6. Myoglobinuria and renal damage
7. Severe fluid and electrolyte upsets
8. Local and systemic infection
9. Remote organ compromise or failure

sures reached 64 mm Hg in the forearm or 55 mm Hg in the calf. Systemic hypotension or arterial spasm reduced the magnitude of tissue pressure increases needed to impair perfusion. Elevation of a limb containing compartments with increased pressures can reduce arterial pressure sufficiently to arrest circulation in the affected compartments (14).

Total ischemia has been used experimentally to show that:

1. Neural abnormalities (paresthesias and hypesthesias) begin after about 30 min of ischemia and irreversible functional loss occurs if ischemia exceeds about 12 h (7,15,16)
2. Muscle changes begin after about 2 h, become irreversible after 4 h, and result in contractures after about 12 h (7,17)
3. Permeability of capillary endothelium increases after about 3 h of experimental total ischemia (7)
4. About 4 h of ischemia produces myoglobinuria that reaches its maximum after 3 h of reperfusion and may continue for 12 h (7,18).

Because partial perfusion exists in most clinical situations, the times required to produce changes in human tissue structure and function may be unpredictably longer than values quoted for total cessation of circulation in animals.

As the major dynamics of ischemia occur in affected tissues at the level of the microcirculation, pulses in peripheral vessels, as well as capillary refill in skin and nail beds, are frequently normal despite the clinical presence of a compartment syndrome in that extremity (7). When muscle necrosis is found at fasciotomy, its degree indicates grossly the extent and duration of ischemia. It may also correlate with the subsequent hardships of convalescence and the severity of residual dysfunction.

Compartment syndromes have been recognized in the shoulder, forearm, hand, buttocks, and lower leg. Physical signs depend on the structures involved and include sensory loss, muscle weakness, painful passive movement, and local tissue distention. Early decompression decreases postischemic swelling, tissue devitalization, and the likelihood of infection. Compartment pressure elevations lasting longer than 12 h have been associated with chronic functional defects, including sensory changes, motor weakness, and eventual contractures (19).

Vascular Punctures and Compartment Syndromes

Halpern et al. (5) have reported a compartment syndrome of the forearm that occurred after multiple attempts to obtain a blood sample for blood gas determination from a radial artery of a patient who had received chronic anticoagulant therapy. Neviaser and associates (6) described seven anticoagulated patients in whom brachial artery punctures for obtaining blood gas samples were followed by compartment syndromes in the forearm. Awareness that vascular punctures or cannulation can leak volume sufficient to elevate interstitial pressure (Table 1) and produce a compartment syndrome is essential when anesthetizing anticoagulated patients.

Irritating compounds, injected in or about vessels, have been associated with compartment syndromes. Bortolussi et al. (3) report an antecubital administration of diazepam that was followed by a compartment syndrome of the forearm. Surgical inspection of the brachial artery in the antecubital area showed no discernible puncture wound. Dry gangrene of the digits resulted despite surgical decompression. Doppler evidence of radial and ulnar arterial flow was present during the compartment syndrome.

Table 3. Traits of an "Axillary" Roll

1. *Thick and noncompressible enough* to raise the chest wall off of the surface of the operating table and free the down-side shoulder girdle from medial pressure
2. *Wide enough* to distribute its elevating pressure across the area of several ribs
3. *Long enough* to support the entire anteroposterior expanse of the down-side lateral chest wall
4. *Placed just caudad to the axilla* to avoid compression of axillary neurovascular contents

Patient Positioning and Compartment Syndromes

Upper Extremity

Shoulder. Nambisan and Karakousis (20) describe a young adult patient who remained in the left lateral decubitus position for 9 h during removal of a tumor from thoracic vertebrae. Toward the end of the procedure, the position was gradually converted to the semiprone, apparently without dorsal relocation of the down-side arm (21). When the patient was subsequently turned supine, the left (down-side) shoulder and adjacent neck were found to be extensively swollen. The left arm and hand were not involved, the quality of the radial pulse was good, and the patient could move the fingers. The blood pressure cuff had not been applied to the left arm and no attempts were made to use the left arm, subclavicular area, or neck for intravascular lines. By the following morning the swelling of the shoulder had become massive and the left arm and hand were immobile and numb. Emergency surgical decompression of the area was accomplished. Sensation and movement in the hand and forearm returned by the following day. Swelling of the shoulder subsided within several weeks and 1 yr later all functions of the shoulder, arm, and hand were again normal.

An "axillary" roll had been placed under the medial wall of the down-side axilla when the lateral position was established. Apparently the roll was either too thin to elevate the chest wall sufficiently to decompress the down-side shoulder or it had moved far enough cephalad in the axilla to have compressed axillary structures. Severe circumduction of the shoulder during the semiprone position may also have led to the ensuing compartment syndrome by compromising perfusion of the shoulder area.

Careful attention should be paid to the characteristics (Table 3) of the axillary roll when the lateral position is established. A stack of wrinkle-free folded towels or sheets usually suffices. Terry cloth bath towels should be avoided as supporting rolls because their rough surfaces may injure the skin at the points of contact. Reusable plastic lifts often become compressible and soon fail as supports.

When the lateral position is shifted to semiprone, the down-side arm should not be allowed to be seriously circumducted across the ventral chest wall lest its suprascapular nerve be stretched enough to produce postoperative shoulder pain (22). A convenient choice is to locate the down-side arm alongside, and slightly dorsal to, the semiprone trunk (21) with the radial pulse carefully checked for sufficiency after posture has been finalized.

Forearm. A century ago Richard von Volkmann recognized ischemic contractures of extremities after compression by excessively tight dressings (23). The entity was one of the earliest forms of what we now regard as a compartment syndrome.

On the volar surface of the dense interosseous membrane that connects the radius and ulna run the anterior interosseous nerve and its associated vessels. The anterior interosseous nerve, a terminal branch of the radial nerve lacking sensory fibers, provides motor supply to muscles of the forearm and hand. Direct injuries to forearms have produced compartment syndromes and dysfunction of anterior interosseous neurovascular bundles (24). A compartmentlike syndrome has been encountered in the forearm of a patient who had been supine during a general anesthetic. The tight binder retaining that supinated forearm on an arm board was alleged by the plaintiff to be compressive enough to initiate ischemia in the anterior interosseous compartment and cause an "anterior interosseous nerve syndrome." Weighed against the thousands of times each month that such a possibility exists in operating rooms in North America, the paucity of reports of such a complication strongly implies that the allegation in question was speculative and without merit. Nevertheless, such a possibility should be kept in mind as an arm is being well padded and retained on an arm board.

Lower Extremity

Exercise history. Intensive use of leg muscles may increase pressures within compartments of the leg to the point of compromising circulation and producing pain. In the acute form, the process leads to ischemic destruction of compartment contents. In the chronic syndrome, exertional pain is relieved by rest and recurs with additional exercise (25). The underlying cause is likely to be hypertrophic musculature within a compartment rather than a poor vascular supply to that compartment. Therefore, a history of recurrent lower leg pain with exercise should alert the anesthesiologist to the possibility of abnormal perfusion of

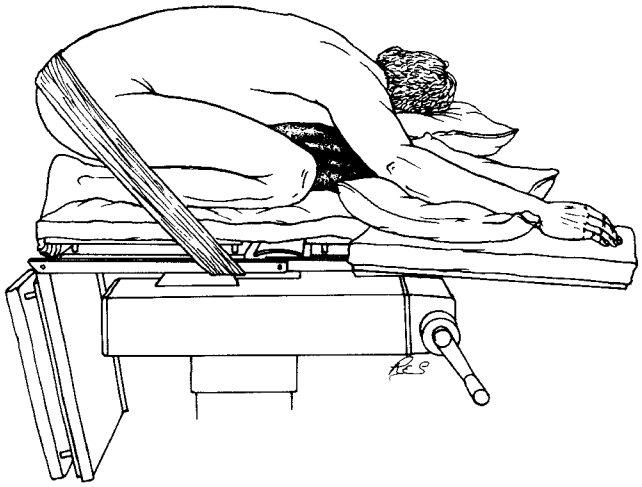

Figure 1. The tuck position. Severe flexion of knees and hips can angulate vessels and produce ischemia of the extremities. (Redrawn with permission from Wayne SJ. The tuck position for lumbar disc surgery. J Bone Joint Surg 1967;49:1195. Dr. Wayne currently uses only a modified tuck position as reported in Wayne SJ. A modification of the tuck position for lumbar spine surgery. Clin Orthop 1984;184:212.)

the limb and the potential for development of a compartment syndrome in the presence of sustained hypotension or extended external pressure on the leg during a surgical procedure.

Prone position. Many variations of the prone position have been recommended as methods of avoiding pressure on the abdomen that would congest perispinal vasculature and compromise hemostasis during lumbar area surgery (26,27). Any prone position that requires ventroflexion of the lumbar spine to separate the spinous processes and widen the dorsal aspects of the intervertebral spaces renders the lower extremities dependent and susceptible to pooling of significant amounts of blood. The remedy has been to apply compressive wrappings from foot to midthigh bilaterally before pronating the patient. If tight enough, the wraps fit the description of compressive bandages that decrease compartment size (Table 1). The fact that gravity aids arterial flow into the dependent wrapped areas probably preserves perfusion and accounts for the scarcity of reports of compartmental syndromes after leg wrapping. I am aware, however, of several conversational anecdotes describing wrapped legs that became ischemic after pronation. Care should be taken to see that the tension applied to the wrappings is not excessive and that the proximal limit of the wrap does not act as a tourniquet.

An extreme degree of flexion of the knees and hips (Figure 1) was a component of an early kneeling variety of the prone position variously known as the "carpenter's rule position," the "Muhammedan praying position," or the "tuck position." Reports of postoperative myoglobinemia leading to renal failure began to appear after fairly widespread use of the position (28,29). Although not specified at the time, vascular obstruction in the severely angulated inguinal and/or popliteal regions undoubtedly produced remote perfusion failure and resulted in compartment syndromes in the lower extremities. Apparently the position is no longer extensively used.

Recently, Aschoff and associates (30) reported two instances of compartment syndromes after lumbar disk surgery with the patients in a "genu-pectoral" (knee-chest) position with a pad between the Achilles tendon and buttocks. The result is undoubtedly much tighter flexion of legs on thighs than used in the kneeling position on an Andrews Frame. The ability of their pad to compress calves was not described. One patient was in position for slightly more than 3 h, the other for 4 h. In each case 12 uneventful postoperative hours were followed by the development of compartment syndromes.

Lithotomy position. Instances of compartment syndromes have been reported to follow use of several versions of the lithotomy position, with emphasis being on the considerable length of time that the patient remained in the position (31–33). Each of the several different leg holders available presents both assets and problems (Figures 2 and 3). Most can compress extremity tissue to some degree and all can be elevated enough to create a potentially damaging perfusion gradient. The term "stirrup" should be abandoned because its careless jargonistic use has rendered it nondescriptive.

Perhaps the least complicated holder is either a cloth sling that encircles the instep and heel (Figure 2A) or a cloth boot that encases the foot. It does not compress extremity tissue, but the knee area is not stabilized against medial and lateral pressures.

A "knee crutch" (Figure 2B) consists of an angulated metal trough that supports the popliteal fossa and extends a short distance cephalad on the dorsal thigh and caudad on the calf. The entire foot and the portion of the leg that is beyond the distal edge of the device are unsupported. Consequently, the weight of the foot and lower leg can produce a leverlike action that compresses the calf against the distal edge of the holder. Its ability to compress the calf and popliteal fossa makes the knee crutch a questionable choice as a support for lengthy procedures done in the lithotomy position. Recently, a trial (34) involved bilateral lower extremity compartment syndromes that followed a 10-h gynecologic procedure done with the

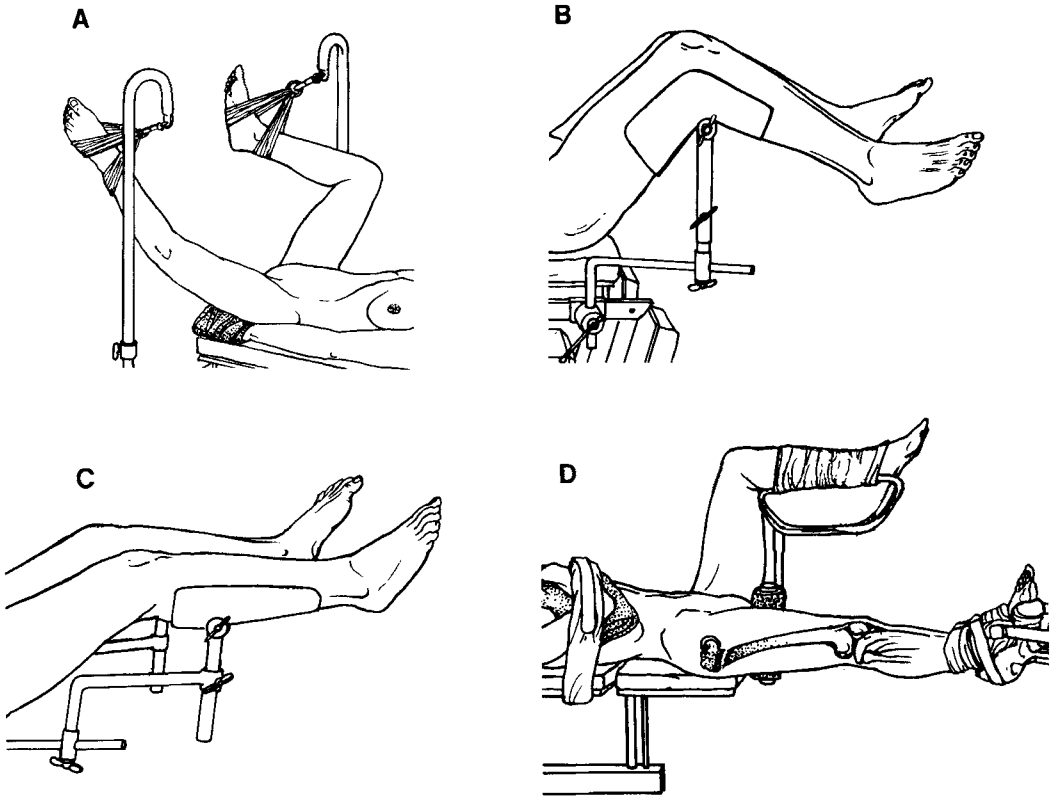

Figure 2. Varieties of lithotomy leg holders (protective padding omitted for clarity). **A.** Cloth slings attached to vertical "shepherd's crook" or "candy cane." (Cloth bootie may replace slings.) **B.** Knee crutch ("Bierhoff" leg holder). **C.** Calf rest. **D.** Calf rest for elevated limb while injured extremity is in surgical traction. [Figures modified with permission from Martin JT, ed. Positioning in anesthesia and surgery. 2nd ed. Philadelphia: WB Saunders, 1987: (A) from Figure 8-1 on page 58, (B) from Figure 7-2 on page 54, (C) from Figure 12-4 on page 120, and (D) from Figure 18-4 on page 227.]

patient in this type of leg holder. Now under litigation from the same institution is a similar problem that was previously reported by Adler and associates (33). Leff and Shapiro (35) warned against use of the knee crutch leg holder in extended procedures.

In the device depicted in Figure 2C, the calf rests in a semicylindrical trough and bears the complete weight of the lower extremity. The unit is used often for prolonged low lithotomy positioning. It is not free of the potential for significant compression of the calf if the extremity is heavy or if the contained limb is used as an arm rest by a standing member of the surgical team.

A variant of this arrangement supports the elevated limb of a patient in the hemilithotomy position on a fracture table (Figure 2D). Two patients developed compartment syndromes in the elevated limb after being in the hemilithotomy position for intramedullary nailing of the femur for 5.5 and 6.0 h, respectively (36).

A foot holder resembles the plantar and dorsal halves of a ski boot with its proximal end flared somewhat (Figure 3A). With this device the weight of the extremity is borne by the holding boot, which needs careful padding between its interior and the heel and sole of the patient's foot. Despite the firmly held limb, no pressure is placed on the popliteal fossa and almost none is placed against the distal calf. This unit obviously requires an intact skeleton in the contained extremity.

A popular device combines the knee crutch (Figure 2B) and the foot holder (Figure 3A) above by using a telescoping rod to connect the knee crutch and the foot holder (Figures 3B, C). It allows good distribution of the weight of the extremity in the holder as well as suitable adjustments of its components to fit the dimensions of the patient. Nevertheless, the device still does not obviate the potential for compression of the popliteal fossa and upper calf by the weight of the supported limb. It may be the holder of choice for the exaggerated lithotomy position (Figure 3C), as its firm support of the elevated extremities stabilizes the position of the symphysis *pubis* for perineal access to retropubic structures.

A modification of a fabric-covered arm frame has provided a soft sling to support most of the lower leg. The proximal end of the frame is widened somewhat and the unit is usually encased in a tubular "stock-

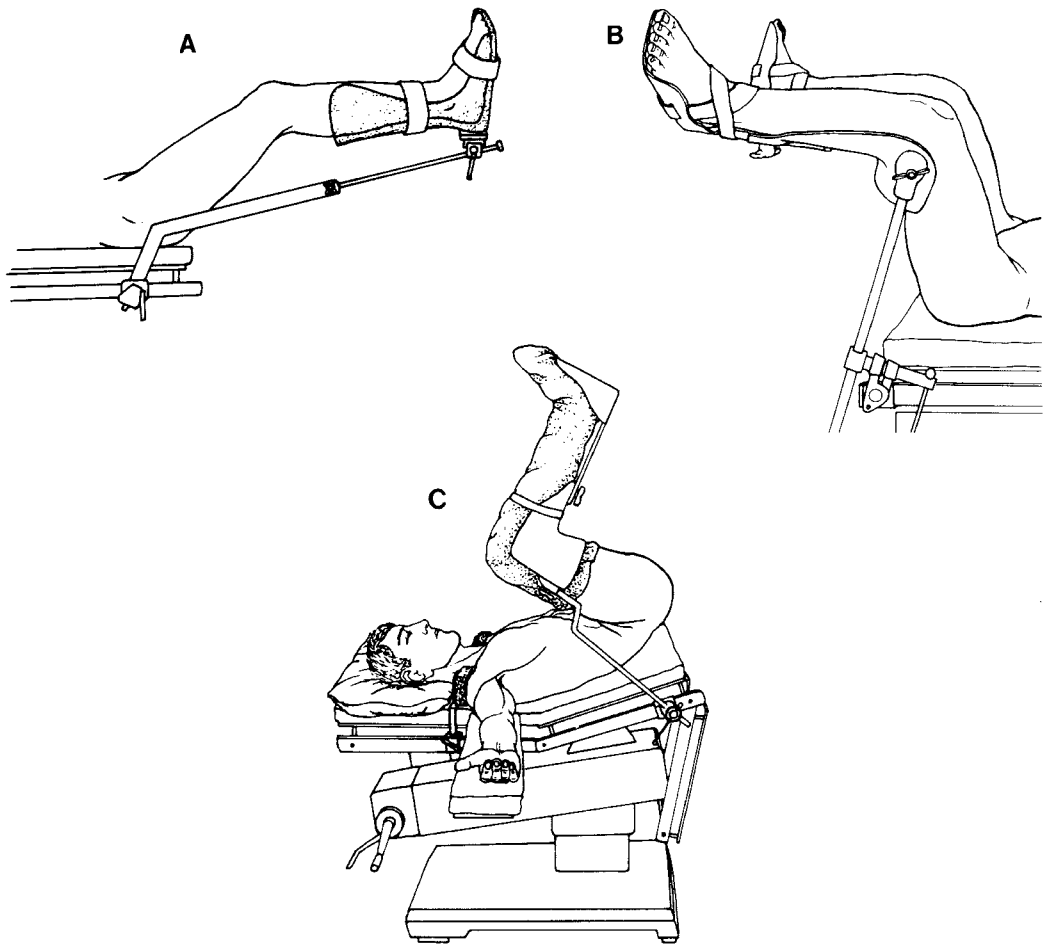

Figure 3. Varieties of lithotomy leg holders (continued) (protective padding omitted for clarity). **A.** Semiboot foot holder that frees popliteal fossa and bulk of calf from pressure. **B.** Knee-crutch-and-boot, distributing weight of extremity between popliteal fossa, calf, and foot. **C.** Knee-crutch-and-boot in exaggerated lithotomy position to firmly retain leg and stabilize tilted pelvis. (Figures modified with permission from: (A) Illustration of item 10001 in brochure of Allen Medical Systems, Bedford Heights, Ohio; (B) Figure 6-4 in Martin JT, ed. Positioning in anesthesia and surgery. 2nd ed. Philadelphia: WB Saunders, 1987:45; (C) Figure 7-3 in Martin JT, ed. Positioning in anesthesia and surgery. 2nd ed. Philadelphia: WB Saunders, 1987:55.)

inette." It distributes the weight of the extremity over the posterolateral surface of the abducted and externally rotated limb (37).

Recently, an elderly gentleman, who had a stenotic carotid artery, intermittent claudication, hypertension on medication, and an old history of a coronary artery bypass procedure that was followed by an angioplasty of a lower extremity, developed a pelvic tumor that needed an extensive abdominopelvic resection. All of the monitors required to control his cardiovascular status during and after the operation were placed before an uncomplicated induction of combined general and epidural anesthesia. His legs were wrapped with compressive dressings to "prevent pooling of blood," then further wrapped into padded leg holders (probably the type shown in Figure 3C) for the low lithotomy position, and the table was tilted about 15° head down. The difficult surgical procedure lasted approximately 10 h. Hypotension that followed the induction of anesthesia was allowed to continue during the first 4 h to minimize surgical bleeding. Otherwise, the monitoring and management of the anesthetic was thoughtful and excellent. Twelve hours after anesthesia, compartment syndromes were recognized in both lower extremities. Fasciotomies were performed and subsequently needed revision. Muscle necrosis was extensive. After a stormy, septic 5-mo postoperative course that included renal and pulmonary failure, he suffered a sudden cardiac arrest from which he could not be resuscitated.

By retrospective calculation, the final operative position placed his feet about 20 in. above the level of his heart (Figure 4). In the presence of systemic arteriosclerosis and treated hypertension, the driving pressure needed to perfuse sclerotic vessels in his

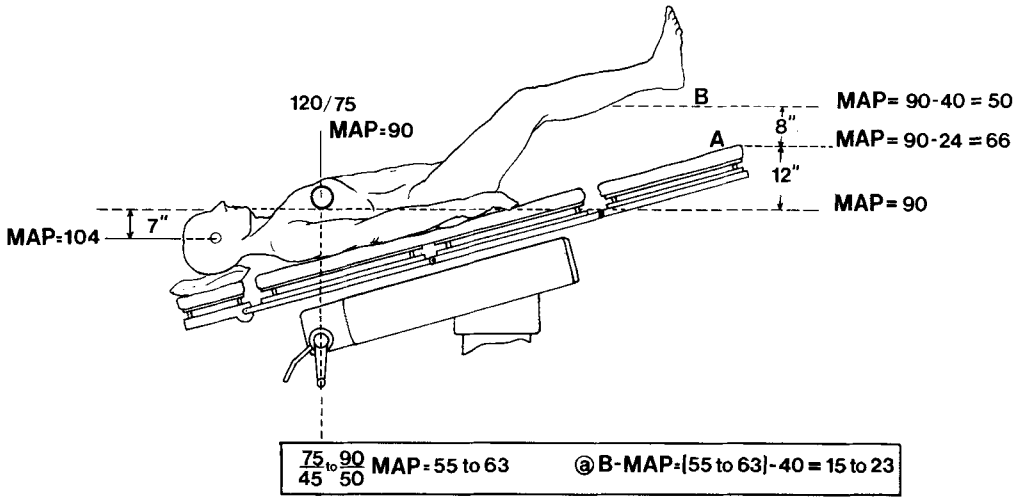

Figure 4. Calculated effects of gravity on perfusion pressure in elevated lower extremities. Pressures vary 2 mm Hg per vertical inch (2.5 cm) (38). (Mean arterial pressure [MAP] estimated as 0.33 [SBP − DBP] + DBP, where SBP = systolic blood pressure and DBP = diastolic blood pressure.) Rectangle displays effects of systemic hypotension on calculated perfusion pressure in distal calves. Sclerotic vessels and external pressures from leg rests or leg wrappings would increase the ischemic potential. (Note: the elevated single leg in the hemilithotomy position [Figure 2D] would be about 18–20 in. above heart level with MAP at the calf being approximately 36–40 mm Hg less than cardiac MAP.)

legs was increased by (a) compressive leg bandages, (b) the uphill gradient to his legs in low lithotomy, (c) additional wrappings to secure his legs in the holders, and (d) adding head-down tilt to the lithotomy position. During the hypotensive period, mean arterial blood pressure (MAP) at the heart ranged between 55 and 63 mm Hg. Simultaneously, because MAP varies 2 mm per vertical inch (38), the MAP in the elevated, wrapped calves (Figure 4) would have hovered around 20 mm Hg in structures accustomed to the chronically high MAP of hypertension. Vasodilation from epidural anesthesia was probably not a useful factor in aiding leg perfusion because of the simultaneous presence of external compression from the leg wrappings. A significant issue was the 10 or more hours of lithotomy position. Extensive myonecrosis at fasciotomy indicated that the perfusion deficit in affected compartments had been severe and lengthy.

Varying opinions exist about the ability of postoperative analgesia to impede recognition of a compartment syndrome. Based on evidence that was open to serious question and involved only one patient, Strecker and associates (39) asserted that a continuous infusion of epidural bupivacaine for postoperative analgesia masked the development of compartment syndrome pain in a leg from which a donor bone graft had been taken. While that syndrome was induced by invasive surgery and not by patient posture, their implication indicted epidural analgesia in all compartment syndromes. Recently, by contrast, Montgomery and Ready (4) reported two patients in whom, after use of the lithotomy position for prolonged genitourinary procedures under combined general anesthesia and epidural lidocaine, unilateral compartment syndromes requiring fasciotomies were "readily diagnosed" despite the presence of otherwise effective postoperative epidural morphine analgesia accomplished by intermittent injection of functioning catheters. Reinjecting the catheters did not relieve the development of excessive limb pain in either of their patients, suggesting a sympathetic perivascular pathway for the pain. When the potential for a compartment syndrome exists, they recommend a high index of suspicion plus vigilant clinical evaluation to detect its onset and institute treatment. Additional experience is needed to resolve the controversy, but data holding epidural analgesia responsible for obscuring the diagnosis of a compartment syndrome are sparse and the evidence is flimsy at best.

Perspectives

Because the entity of compartment syndromes still may be unfamiliar to some practitioners who require use of the lithotomy position (32), the anesthesiologist should bear in mind the following considerations:

1. The duration of lithotomy position is apparently a matter of concern. Compartment syndromes have been associated with several different types of leg holders when lithotomy position time has ex-

ceeded about 5 h (31–34). Long procedures that require lithotomy for only a part of the operation can often be planned so that either much of the procedure can take place before establishment of lithotomy or the patient's position can be changed to an alternative posture for the balance of the operation when lithotomy is no longer needed.

2. For a prolonged procedure, a leg support that allows pressure either in the popliteal fossa or on the calf should be avoided despite seemingly adequate padding. An exception is the necessary use of present models of fracture tables with the hemilithotomy position; however, the table manufacturers should be asked to make design modifications in the holder for the elevated leg so that the calf is freed from compression.

   Use of the popliteal support, or knee crutch, which allows the lever action of an unsupported foot to intensify the pressure of the distal lip of the crutch on the patient's calf, should be limited to brief procedures and the crutch must always be very carefully padded.
3. Throughout the procedure, blood pressure should be maintained carefully within ranges that are normal for that patient. Loss of driving pressure for blood flow in elevated extremities allows compartmental ischemia to occur at lesser levels of increased compartment pressure. Hypotension used to conserve blood loss and replacement in the multiple-trauma patient may intensify existing vascular injuries in extremities. The risks posed by vasoconstrictor drugs that increase peripheral resistance and decrease compartment blood flow should be borne in mind. Of major importance are the warnings of Matsen (7) and Ashton (13) that, once a compartment syndrome has been diagnosed, elevating the involved extremity in an attempt to promote venous drainage only lowers driving pressure into that limb and further diminishes perfusion of its tight compartments.
4. Wrapping elevated legs to prevent blood from pooling therein is thoughtless and counterproductive.
5. Detecting the onset of compartment syndromes during anesthesia is apt to be impossible. Distal pulses in an extremity, as well as capillary refill in the ipsilateral nail beds, remain essentially unchanged despite the development of the syndrome. Reperfusion is needed to produce the tissue edema that initiates a compartment syndrome; usually this adds a time factor to the development of the syndrome that may extend its earliest recognition to well beyond the termination of anesthesia. Thus, measuring compartmental tissue pressures during anesthesia and surgery may be neither practical nor informative. More prompt indicators of an incipient compartment syndrome should be sought as experience accumulates.
6. The choice of anesthesia for lengthy procedures to be done in the lithotomy position has little bearing on the potential for diagnosis of a compartment syndrome at or after the end of the case. Recent evidence (4) indicates that postoperative epidural analgesia did not prevent early recognition of the development of the syndrome.

Data about the association of compartment syndromes with vascular punctures and patient positioning are sparse, anecdotal, and not susceptible to statistical analysis. However, published reports and recent litigation confirm distinct hazards reliably enough to compel attention. Although compartment syndromes associated with anesthesiologic maneuvers are admirably infrequent, they do occur. The uncomfortable conclusion is that some may be preventable. Familiarity with the circumstances that encourage development of a compartment syndrome will allow the anesthesiologist to cooperate effectively with other members of the surgical team in avoiding this distressing cause of major postoperative morbidity.

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