AMERICAN JOURNAL OF BIOLOGY AND NATURAL SCIENCES
https://biojournals.us/index.php/AJBNS
ISSN: 2997-7185

Article

Neurovascular Assessment and Peripheral Nerve Involvement in Cases of Fractures: A Narrative Review with Integration of Descriptive Data from Clinical Practice (n = 100)

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Citation: Fanar, S. M., & Ahmed, O. Q. Neurovascular Assessment and Peripheral Nerve Involvement in Cases of Fractures: A Narrative Review with Integration of Descriptive Data from Clinical Practice (n = 100). American Journal of Biology and Natural Sciences 2026, 3(11), 165-178.

Received: 13th Aug 2026
Revised: 28th Sep 2026
Accepted: 15th Oct 2026
Published: 25th Nov 2026

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Abstract: Neurovascular assessment involves a structured clinical assessment of peripheral tissue perfusion and neural integrity distal to an orthopedic injury or immobilization procedure. Neurovascular assessment is a fundamental skill in trauma and orthopedic nursing and requires regular neurovascular assessment in patients with fractures or treated with splints, circumferential casting, or traction. Trauma-related hemorrhage, inflammation processes, and interstitial edema combined with external mechanical compression may increase intracompartmental pressure, disrupt microvascular capillary perfusion, and compress peripheral nerves. Failure to identify any neurovascular deterioration could lead to ACS, ischemic myonecrosis, permanent sensorimotor disorders, Volkmann's ischemic contracture, and amputation of the limb. The purpose of this review article is to integrate current anatomical, pathophysiological, and clinical data on bedside neurovascular assessment, intervals of serial assessment, documentation of findings, and nursing escalation criteria using a clinical descriptive dataset of 100 patients with fractures. The interpretation of the 'Six Ps' (pain, pallor, pulselessness, paresthesia, paralysis, and poikilothermia) as changing signs instead of a checklist is of particular importance. Excessive pain unresponsive to analgesics and intense pain caused by passive muscle stretching are primary signs of ACS development, while pulselessness and paralysis are late, irreversible manifestations of neurovascular compromise. Moreover, this paper describes the indications for monitoring intracompartmental pressure, immediate nursing actions such as neutral limb positioning at heart level, casting bivalving, and structured communication through the SBAR method. Within the associated clinical sample (N = 100), young adults in the age range of 18-28 years comprised 45.0% of the patients, tibial and leg fractures were prevalent (42.0%), 44.0% of the subjects were under cast or splint fixation, and 34.0% had peripheral neuropathies—mostly involving the peroneal nerve (52.9%). It is essential to note that the presence of a palpable distal arterial pulse should not be used to rule out early ACS, and constant nursing assessments and surgery referral are crucial for limb preservation.

Keywords: Neurovascular assessment; Fractures; Splints; Casts; Acute compartment syndrome; Orthopedic nursing; Peripheral ischemia

Introduction

Neurovascular assessment is a process of conducting an organized evaluation of arterial blood supply, venous drainage, and peripheral nervous system function distal to the site of a physical trauma or orthopedic manipulation [1]. In the context of emergency and trauma nursing practice, as well as orthopedic care, the neurovascular assessment can be defined as an essential measure aimed at protecting the patient from complications associated with fractures, soft tissue crushing, closed reduction, skeletal traction, splinting, or circumferential casting [2]. More specifically, the purpose of such an assessment goes far beyond the mere detection of a peripheral pulse; it is focused on establishing a physiological baseline and identifying early signs of pathology or acute compartment syndrome (ACS) [3].

Traumatic bone fractures interrupt the local vasculature and lead to an inflammatory response both locally and systematically [4]. This leads to an increase in microvascular endothelial permeability with fluid leakage and interstitial edema, causing increased pressure in closed myofascial compartments [4]. Compartment syndrome occurs when the injured limbs are tightly wrapped in rigid, non-compliant dressing, splint, or plaster casts, which restrict tissue swelling, thus increasing compartmental pressure [5]. Persistent high tissue pressure leads to capillary beds collapse, which causes hypoxia of peripheral nerves and muscle death [6]. Based on BOA, ACS results from excessively high pressure in the closed myofascial compartment with pain being disproportionate to the clinical injury and passive stretching pain of involved muscles being cardinal signs of ACS [7].

Since nurses are always at the patient's bedside during the acute and subacute phase of trauma, they are best placed to notice changes in neurovascular status [8]. Intense observation, meticulous trend documentation, conservative treatment of high pressure, and multidisciplinary intervention save the patient from permanent neuromuscular disability and amputation of the affected limb [9].

Materials and Methods

Aim and Objectives

Aim

In order to review the anatomical bases, pathophysiology, and evidence-based nursing management principles of conducting a neurovascular examination in fracture patients utilizing splints and casts, especially with an emphasis on limb-threatening complications, in relation to a clinical cohort of 100 hospitalized fracture patients.

Objectives

1. Discuss the anatomy of peripheral arteries, veins, and nerves involved in limb perfusion and neurological testing.
2. Discuss the mechanism of occurrence of post-traumatic swelling, expansion of hematomas, mechanical compression from outside, and ischemia of the microcirculation.
3. Discuss the clinical elements that make up a thorough bedside neurovascular examination which includes capillary refill time (CRT), selective nerve-muscle examination, and the interpretation of the "Six Ps."
4. Determine how often monitoring should be done and how to document the findings using objective parameters and comparing findings bilaterally.
5. Discuss the criteria for diagnosis and surgical treatment of ACS.
6. Define structured interprofessional communication strategy (SBAR), nursing interventions, and patient discharge education regarding safety.
7. Define the profile of a group of 100 patients with fractures in terms of demographics, anatomic location, immobilization, and neurological involvement.

Relevant Anatomy and Physiology

Peripheral Artery Circulation

Peripheral arteries carry oxygen-filled blood and metabolic substrates which are required for cells to survive [10]. In the upper limb, the brachial artery divides at the cubital fossa into radial and ulnar arteries, which connect to give the superficial and deep palmar arches that supply digital arteries [10]. Radial and ulnar pulses at the distal end of the volar and medial surfaces of the forearm, respectively, provide assessment of distal forearm blood flow through palpation [11]. In the lower limb, the popliteal artery is divided into the anterior and posterior tibial arteries [12]. The anterior tibial artery goes on to become the dorsalis pedis artery on the dorsum of the foot, while the posterior tibial artery travels behind the medial malleolus [13]. Vascular assessment requires examination of pulse presence, strength (graded from 0 to 3+), rhythm, and symmetry between the sides [13].

Venous Drainage and Microcirculation

Veins in deep and superficial systems return deoxygenated blood and metabolic products back to the central circulation [14]. Venous flow is compromised with external pressure from tight dressings and with dependence due to increase in hydrostatic capillary pressure and accumulation of interstitial fluids [15]. In an inelastic osteofascial compartment, venous congestion forms a vicious cycle, leading to increased compartment pressure and poor arterial capillary inflow [16].

Peripheral Nervous System

Peripheral nerve trunk consists of myelinated motor and sensory nerves together with unmyelinated autonomic fibers, all sensitive to hypoxia [17]. In the upper limb, radial, median and ulnar nerves innervate sensory dermatomes and muscles of intrinsic and extrinsic hand muscles [18]. Tibial and common fibular (peroneal) nerves, whose divisions are superficial and deep, innervate lower limb plantar flexion, dorsiflexion, toe extension and cutaneous sensation [19]. Sensory myelinated nerves are especially sensitive to ischemic depolarization and paresthesia usually occurs prior to muscle paralysis and absence of arterial pulse [20].

Implications of Anatomical Specificity for Clinical Practice

For a clinically valid neurovascular assessment, a specific nerve distribution and a specific vascular territory should be evaluated beyond the level of injury or immobilization [21]. Asking the patient to "wiggle your toes" and similar non-specific instructions have no clinical validity, since proximal muscle innervation can compensate for the loss of innervation distally. The nurses need to test specific innervations, which include dorsiflexion of great toe (deep peroneal nerve), plantar flexion (tibial nerve), thumb/index opposition (median nerve), and cutaneous sensation in the first dorsal interosseous web space of foot (deep peroneal nerve) [22]. Documentation of an adequate baseline before immobilization is essential to differentiate primary injury from the secondary one [22].

Pathophysiology of Neurovascular Compromise

Neurovascular compromise occurs when local tissue perfusion becomes inadequate for the cell's physiological needs or due to direct compression or ischemic conduction block of peripheral nerves [23]. Following fracture or soft tissue injury, the disruption of endothelium leads to the formation of localized hematoma and release of vasoactive factors (histamine, bradykinin, prostaglandins), causing capillary permeability [24]. There is an outpouring of protein-containing fluid into the interstitium leading to tissue edema [24]. Since muscles are encapsulated by dense and rigid deep fascia and also confined by the circumferential cast/splint, any small volume change results in exponential rise in intracompartmental pressure [25].

Increased tissue pressure decreases the arteriovenous pressure gradient of perfusion in the compartment, clinically represented as the compartment perfusion pressure (ΔP = Diastolic Blood Pressure – Intracompartmental Pressure) [26]. With increasing tissue pressure to be close to 10-30 mmHg above diastolic blood pressure, thin-walled capillaries and venules collapse leading to cessation of microcirculation in spite of high arterial pressures sufficient to palpate pulses distally [26]. Continuous hypoxia pushes the cells to adopt anaerobic glycolysis leading to acidosis, depletion of ATP-dependent membrane ion pumps, cellular swelling, rhabdomyolysis and necrosis of the neuromuscular tissue [27].

Result

Bedside Neurovascular Assessment (Complete)

Pre-Procedure and Bilateral Examination

Before carrying out the exam, the nurse needs to confirm the identity of the patient, inform them on the process to lower the sympathetic vasoconstriction caused by anxiety, examine cognitive status and any regional nerve blocks or epidural analgesia which may mask the symptoms [2]. The bilateral approach to the examination offers an important physiological control of the skin temperature, skin color, capillary refilling and baseline pulse amplitude [28].

Interpretation in Motion of the 'Six Ps' Paradigm

Traditionally, the neurovascular compromise condition is described using "Six P's": pain, pallor, pulselessness, paresthesia, paralysis, and poikilothermia [29]. In contemporary orthopedic nursing care, it is important to remember that the six signs presented are on a time continuum and not necessarily concurrent findings that should occur together. If one waits for all six symptoms to appear, it will mean irreparable damage to the tissue [30]. The assessment method for each sign is presented in Table 1.

Table 1. *Clinical Assessment Techniques and Diagnostic Significance of the 'Six Ps' Framework.*

Clinical Indicator	Standardized Assessment Technique	Clinical Interpretation & Diagnostic Timing
Pain	Evaluate intensity (0–10 scale), quality, location, progression over time, response to intravenous opioids, and pain elicited by passive stretching of distal digits [29].	**Cardinal Early Sign:** Severe, escalating pain out of proportion to the injury—or excruciating pain on passive muscle stretch—is the most sensitive early clinical indicator of ischemia and impending ACS [7].
Paresthesia	Assess light touch, pinprick sensation, two-point discrimination in specific nerve dermatomes, and subjective numbness, tingling, or 'pins and needles' [17].	**Early Neurological Sign:** Reflects early ischemic hypoxia of large myelinated sensory fibers. Sensory deficits consistently precede motor paralysis [31].
Pallor	Inspect skin color and nail beds distal to the injury/cast under adequate lighting; compare directly with the uninjured limb [13].	**Variable Perfusion Sign:** Pale, mottled, or dusky/cyanotic skin indicates impaired cutaneous arterial inflow or venous congestion; however, normal skin color does not exclude early ACS [13].
Poikilothermia	Palpate skin temperature distal to the injury using the dorsum of the examiner's hand; compare bilaterally [32].	**Variable Perfusion Sign:** A cool or cold extremity (accounting for ambient environmental temperature) indicates reduced arterial perfusion [32].
Pulselessness	Palpate distal pulses (radial, ulnar, dorsalis pedis, posterior tibial) for presence and amplitude (0 to 3+); utilize handheld Doppler ultrasound if non-palpable [13].	**Late Ominous Sign:** Diminished or absent pulses signify major arterial occlusion or end-stage compartment hypertension. Palpable pulses remain intact in early ACS [33].
Paralysis	Test active range of motion and graded muscle strength across isolated distal nerve-muscle units [18].	**Late Ominous Sign:** Progressive paresis or inability to actively move distal digits indicates advanced neuromuscular ischemia or structural nerve disruption [34].

Microvascular and Tissue Additive Indicators

Capillary Refill Time (CRT): Defined as the application of constant pressure on distal nail bed or digit pulp for 5 seconds until blanching is achieved, followed by assessment of how long it takes before the color returns to its original pink color after releasing the pressure [35]. CRT should be ≤ 2–3 seconds. An increase in CRT (> 3 seconds) would indicate inadequate peripheral microvascular perfusion, but this should be interpreted considering core body temperature, environmental room temperature, and previous peripheral vascular disease [36].

Swelling and Compartment Pressure: Inspect and palpate exposed distal and proximal soft tissues from splints or casts to determine the presence of swelling, taut or shiny skin, and hardness [5]. The 'woody' feeling when palpating a myofascial compartment signifies a high intracompartmental pressure [9].

Evidence-Based Monitoring Guidelines and Frequency

As traumatic swelling and hematomas expand dynamically within 24-72 hours after injury, fracture reduction, or casting, a normal baseline neurovascular observation never excludes any possible further damage [2]. The intervals between the examinations should be selected taking into account the clinical risk, mechanism of injury, and institutional policies:

1. Acute Stage (Up To 1-2 Hours After Injury, Reduction, Traction or Casting Application): Neurovascular assessment every 15-30 minutes up to 1-2 hours [8].
2. Acute Stage (First 24 Hours After Injury): Structured neurovascular observations every 1 hour for the first 24 hours [8].
3. Subacute Stage (24-48 Hours After Injury): Neurovascular observations every 2-4 hours if neurovascular status is stable and pain is under control [8].
4. Stable Stage Before Discharge: Neurovascular assessment every 4-8 hours before discharge from the hospital [8].

MANDATORY CLINICAL PRACTICE: High-risk patients, such as patients with high-energy tibial diaphyseal fractures, forearm fractures, crush injury, vascular repair, circumferential plaster casts, or altered level of consciousness/ regional nerve block, should be mandatorily monitored neurovascularly on an hourly basis for a minimum of 24-48 hours [8].

Acute Compartment Syndrome (ACS)

Pathophysiology and Epidemiology

The Acute Compartment Syndrome can be categorized under the class of surgical emergencies characterized by increased pressure inside an enclosed osteofascial compartment that impairs the blood supply and viability of tissues [7]. Skeletal fractures cause about 75% to 80% of all Acute Compartment Syndrome cases, while the commonest cause of the syndrome among the skeletal fractures is the closed diaphyseal fractures of the tibia, accounting for more than a third of the skeletal injury-related cases, with distal radius and forearm fractures following [9].

Clinical Diagnostics and Measurement of Intracompartmental Pressures

The diagnosis of ACS can be largely clinical, based on disproportional pain and pain during stretching [30]. On the other hand, if the patient is obtunded, intubated, pediatric, or undergoing regional anesthesia such that his pain perception is unreliable, then direct measurement of ICP using a slit catheter or needle manometer becomes necessary [37]. The current consensus within orthopedics states that a perfusion pressure differential (ΔP = DBP − ICP) of ≤ 30 mm Hg indicates critical ischemia requiring urgent fasciotomy surgery [26].

Definitive Treatment: Immediate Fasciotomy

When ACS is diagnosed or when there is strong suspicion of ACS, then surgery is the only definitive option for full-thickness fasciotomies to ensure immediate reperfusion through the skin and fascial layers of all involved compartments [7]. Both experimental and clinical data have shown that necrosis of the skeletal muscle and axonal degeneration is already established after 4 to 8 hours from the onset of ischemia [6]. Any delay beyond 6 to 8 hours poses higher risks of developing Volkmann's contracture, systemic rhabdomyolysis, and kidney failure [38].

Comparison of Expected Symptoms after an Injury and Possible Impeding Compromised State

An important concern in trauma nursing practice is to differentiate between normal inflammation after an injury and the onset of compromised state. Table 2 illustrates the differences between both states.

Table 2. *Clinical Differentiation Between Expected Post-Injury Recovery and Impending Neurovascular Compromise.*

Clinical Parameter	Expected Post-Injury / Post-Cast Findings	Red-Flag Indicators of Impending ACS / Ischemia
Pain Trajectory	Mild-to-moderate, localized to fracture site; gradually subsides; responsive to standard analgesics and limb positioning [29].	Severe, deep, throbbing pain out of proportion to injury; escalates rapidly; refractory to escalating intravenous opioid therapy [7].
Passive Stretch Test	Minimal discomfort or localized mechanical pain directly at the fracture site [7].	Excruciating, agonizing pain elicited by gentle passive stretching of distal digits/muscles traversing the compartment [30].
Sensory Function	Intact light touch and sharp/dull discrimination; symmetrical with contralateral limb [17].	New-onset paresthesia (tingling, burning, numbness) or progressive hypoesthesia within specific distal nerve distributions [31].
Skin Color & Temperature	Warm to touch, normal pink coloration; CRT ≤ 2–3 seconds [36].	Pale, mottled, dusky, or cyanotic skin; cool/cold temperature; delayed CRT > 3 seconds [36].
Motor Strength	Preserved active digital motion within expected pain and mechanical limits [18].	Progressive motor weakness (paresis) or complete paralysis of distal muscle groups [34].
Compartment Palpation	Soft to mildly edematous surrounding soft tissues [5].	Tense, swollen, shiny skin with a firm, 'woody' compartment consistency [9].

Evidence-Based Nursing Interventions

Rapid Bedside Decompression and Positioning

1. Neutral Heart Level Positioning of Affected Limbs: Position the affected limb immediately to neutral heart level [7]. Although raising an injured extremity to above heart level helps to decrease the regular venous edema, in ACS, elevation of the limb decreases the local arterial hydrostatic pressure, reduces the arteriovenous perfusion gradient, and increases the speed of tissue ischemia [7]. On the other hand, dependent positioning of limbs below the heart level impedes venous return and causes interstitial edema [8].

2. Removal of External Mechanical Tissue Compression: Remove the circumferential bandages, and according to the hospital guidelines or urgent surgical clearance, bivalve (cut down longitudinally from the medial and lateral sides) any rigid plaster or fiberglass cast [7]. It is very important that all underlying cotton padding and bloody dressings are cut down to the skin as well because blood-filled gauze acts as a rigid tourniquet. Bivalving and cutting of padding can lead to 50% to 65% reduction in compartment pressure [7].

3. Urgent Surgical Intervention: Call the attending orthopedic or trauma surgery immediately when signs of neurovascular compromise arise [8]. Delaying surgery while trying to see if further pain medications will help is forbidden [7].

Analgesic Stewardship and Clinical Surveillance

Administration of ordered analgesics does not interfere with the ACS diagnosis; rather, persistent ischemic pain despite adequate titration of intravenous opioids is a critical diagnostic indicator [29].

Nurses have to document pain scores before and 15-30 minutes after analgesics administration, and report persistent pain promptly to the surgical team [8].

Objective Documentation and Communication Among Healthcare Providers

Objectivity of Clinical Documentation

The assessment of the neurovascular status has to be documented in an objective, time-specific, anatomical and legal manner [2]. The general phraseology like "intact neurovascular status" or "CSM normal" will not do for a documentation of patient's condition and should never be used. Documentation has to contain an objective assessment of the patient's condition; e.g., "Pulses: right dorsalis pedis 2+, posterior tibial 2+; CRT less than 2 seconds; foot warm and pink; light touch sensation intact in first dorsal interdigital web space and plantar surface; no pain on passive dorsiflexion and plantar flexion of the great toe; pain score 3/10 [8].

Structured Escalation: The SBAR Protocol

When neurovascular compromise is detected, systematic communication with the help of the: The Situation-Background-Assessment-Recommendation (SBAR) technique eliminates confusion and ensures quick surgical intervention [8]:

1. Situation (S): Patient name, ward or bed number, and clinical issue in question ('Mr. X is experiencing suspected acute compartment syndrome in the right lower leg. This is what I am calling about now.') [8].
2. Background (B): Diagnosis, injury mechanism, time of closed reduction or casting, recent analgesia used [8].
3. Assessment (A): Changes in the trend, including such symptoms as 'Pain increased from 4/10 to 9/10 despite 4 mg IV morphine. There is very strong pain in passive flexion of great toe. Now there is paresthesia in first dorsal web space. Capillary refill time increased to 4 seconds. Dorsalis pedis pulse 1+' [8].
4. Recommendation (R): Immediate bedside surgical assessment and preparation for bivalving of complete cast [8].

Case Study

Presentation of Case

A 27-year-old man is presented to the orthopedic trauma service after sustaining an isolated closed right tibial shaft fracture from a high speed motor vehicle accident. After closed reduction, circumferential long leg plaster cast from below the knee was applied. Six hours after immobilization, the patient complained of severe, deep, throbbing pain in the right lower leg of intensity 9/10 in numerical rating scale, which remained unaltered despite two successive IV administrations of morphine. Findings from neurovascular assessment on the bed side included:

1. Severe pain on gentle movement of the right big toe into plantar flexion and dorsiflexion.
2. Recent onset of tingling with reduced sensation to light touch in the web space of the right foot.
3. Capillary refill time of 4 seconds with cold distal toes compared to the left leg.
4. Weak but palpable right dorsalis pedis arterial pulse.

Pathophysiological and Clinical Interpretation

The clinical picture is characteristic of developing Acute Compartment Syndrome involving the anterior and deep posterior compartments of the leg. Importantly, the fact of retaining palpable dorsalis pedis pulse (1+) is not a contraindication for ACS; the systolic arterial pressure (~120 mmHg) maintains blood flow within the large channels despite the fact that compartmental pressures have risen above the threshold for capillary perfusion (30-40 mmHg) [33]. The combination of unremitting pain unresponsive to opiates disproportionate to injury, severe pain on passive stretching of the extensor

hallucis longus muscle and deep peroneal nerve paresthesia (first web space) requires immediate action before pulselessness or paralysis occurs [31].

Priority Nursing Action Plan

1. Urgently reposition the right leg into a neutral position at the level of the heart (removing any additional pillows placed above the level of the heart) [7].
2. Call an urgent SBAR report to the on-call orthopedic surgeon for urgent bedside evaluation [8].
3. Strip the circumferential plaster cast completely and incise all cotton dressings to the skin layer [7].
4. Keep the patient strictly nil per os (NPO), prepare large-bore IV access for isotonic fluid resuscitation (to prevent myoglobin-induced nephropathy), and obtain baseline laboratory tests (creatine kinase, renal function, coagulation) [38].

Neurovascular Surveillance Clinical Decision Making Process

Figure 3 outlines the sequence of clinical nursing decision making process from risk stratification to preparation for emergency surgery.

Table 3. *Six-Step Nursing Clinical Decision-Making Pathway for Early Detection and Escalation of Neurovascular Compromise.*

Clinical Phase	Core Nursing Actions & Escalation Criteria
Step 1: Risk Stratification	**Identify high-risk orthopedic patients:** long-bone fractures (especially tibia/forearm), crush injuries, post-reduction, traction, splints, or circumferential casts.
Step 2: Baseline Establishment	Perform and document bilateral comparative neurovascular baseline (Pain, Paresthesia, Pallor, Poikilothermia, Pulses, Paralysis, CRT, compartment tension) prior to immobilization.
Step 3: Serial Surveillance	Execute risk-stratified monitoring (q15–30 min × 2h, then q1h for 24h in high-risk limbs); reassess immediately upon any new analgesic request.
Step 4: Trend & Red-Flag Analysis	Evaluate for disproportionate/opioid-resistant pain, pain on passive stretch, new paresthesia, CRT > 3s, tense compartment, or pulse attenuation.
Step 5: Immediate Decompression	Position limb strictly at heart level; completely bivalve cast and split underlying padding per protocol; escalate immediately to surgical team via SBAR.
Step 6: Perioperative Preparation	Maintain NPO status, establish IV access, document exact timeline of interventions and clinical response, and prepare for ICP measurement or emergency fasciotomy.

Discharge Teaching for Patient and Caregivers

Delayed compartment syndrome and pressure ulcers from casts may arise post-discharge as an outpatient; therefore, teaching on discharge is necessary [28]:

1. **Warning Signs of Complications:** Educate the patient to come back to the ED immediately if there is progressive pain not relieved by the oral analgesics prescribed, developing numbness

or tingling sensation, pallor/blueness of the digits, cold digits/toes, and difficulty moving the digits [28].

2. **Casts and Skin Protection:** Instruct the patient to keep the cast free from dirt and moisture, never insert foreign objects inside the cast to stop itching (it leads to breaking of the skin and infection), and should never cut or trim the edges of the cast alone [28].
3. **Dependent Edema and Venous Return:** Teach to elevate the limb at or just above the level of the heart for the initial 48 hours of an uncomplicated course of recovery to reduce the formation of edema, but perform active ROM exercise of the unimmobilized distal joint [14].

Clinical Descriptive Data for Hospitalized Patients with Fractures (N = 100)

Study Design and Methods

For the purpose of applying the findings on bedside neurovascular monitoring techniques reviewed above in the context of clinical orthopedic traumatology, an analysis of a clinical descriptive cross-sectional dataset that consists of 100 hospitalized patients with fractures has been performed. The data has been prospectively gathered from inpatients who were admitted to the orthopedic and surgical departments of Al-Tarmiyah General Hospital and Medical City Hospital during the period from June 17, 2026, to September 6, 2026. The sample included consecutively admitted adult patients (18-50 years) admitted with fractures of bones during the study period, with no missing data from any of the 100 cases. The categorical variables (age group, fracture type, classification, anatomical site, dislocation, fixation technique, and neurological complications) have been presented as frequencies (n) and percentages (%). The percentages have been calculated in relation to the total number of patients in the study group (N = 100), as well as those with neurological problems (n = 34).

Demographic and Clinical Data

According to Table 3, the most frequent demographic category was that of young adults from the age of 18 to 28 years (45.0%, n = 45), followed by the categories of 29- to 39-year-old (35.0%, n = 35) and 40- to 50-year-old (20.0%, n = 20) patients. When considering fracture morphology, Table 4 shows that the most common type of fractures is transverse (32.0%, n = 32), followed by oblique (28.0%, n = 28), spiral (18.0%, n = 18), comminuted (16.0%, n = 16), and mixed (6.0%, n = 6) types. The proportion of closed fractures was 68.0% (n = 68), while 32.0% (n = 32) was made up of open fractures, which were most common among 18- to 28-year-old patients (18/45; 40.0% in age group) (Table 6).

Table 4. *Age distribution of hospitalized fracture patients (N = 100).*

Age Group (Years)	Frequency (n)	Percentage (%)
18–28	45	45.0
29–39	35	35.0
40–50	20	20.0
Total	**100**	**100.0**

Table 5. Fracture pattern stratified by age group (N = 100).

Fracture Pattern	18–28 yrs (n)	29–39 yrs (n)	40–50 yrs (n)	Total (n)	Percentage (%)
Transverse	13	13	6	32	32.0
Oblique	12	8	8	28	28.0
Spiral	10	6	2	18	18.0
Comminuted	8	5	3	16	16.0
Other/Mixed	2	3	1	6	6.0
Total	**45**	**35**	**20**	**100**	**100.0**

Table 6. Fracture classification (closed vs. open) stratified by age group (N = 100).

Classification	18–28 yrs (n)	29–39 yrs (n)	40–50 yrs (n)	Total (n)	Percentage (%)
Closed	27	25	16	68	68.0
Open	18	10	4	32	32.0
Total	**45**	**35**	**20**	**100**	**100.0**

With respect to anatomical location (Table 6), the fractures that were most frequent were those involving the tibia and/or lower leg (42.0%, n=42), followed by fractures of the forearm/hand (28.0%, n=28) and foot/ankle (18.0%, n=18), whereas upper arm and femoral shaft fractures constituted 12.0% (n=12). Of significance is the fact that tibial/leg and forearm/hand fractures—being the two anatomical sites which carry the greatest clinical risk of developing acute compartment syndrome—accounted for 70.0% (n=70) of all cases combined. In addition, 62.0% (n=62) of the fractures observed had been displaced (Table 8). Immobilization in casts or splints was the main mode of management for 44.0% (n=44), followed by internal fixation using plates and screws (26.0%, n=26), intramedullary nailing (20.0%, n=20), and external fixation/traction (10.0%, n=10) (Table 9).

Table 7. Anatomical distribution of fractures stratified by age group (N = 100).

Anatomical Site	18–28 yrs (n)	29–39 yrs (n)	40–50 yrs (n)	Total (n)	Percentage (%)
Tibia/leg	20	14	8	42	42.0
Forearm/hand	12	10	6	28	28.0
Foot/ankle	8	7	3	18	18.0
Other (arms/thigh)	5	4	3	12	12.0
Total	**45**	**35**	**20**	**100**	**100.0**

Table 8. Fracture displacement status stratified by age group (N = 100).

Displacement Status	18–28 yrs (n)	29–39 yrs (n)	40–50 yrs (n)	Total (n)	Percentage (%)
Displaced	30	22	10	62	62.0
Non-displaced/minimally displaced	15	13	10	38	38.0
Total	**45**	**35**	**20**	**100**	**100.0**

Table 9. Immobilization and surgical fixation modalities stratified by age group (N = 100).

Modality	18–28 yrs (n)	29–39 yrs (n)	40–50 yrs (n)	Total (n)	Percentage (%)
Cast/splint	18	16	10	44	44.0
Plate & screws	12	10	4	26	26.0
Intramedullary nail	11	6	3	20	20.0
Other (external fixation/traction)	4	3	3	10	10.0
Total	**45**	**35**	**20**	**100**	**100.0**

The presence of clinically significant peripheral nerve involvement, in the form of paresthesia or irritation of peripheral nerves, occurred in 34.0% (n=34) of the patients while 66.0% (n=66) had normal neurological function (Table 9). Patients with neurological deficits occurred mostly among younger age group of 18-28 years (18/34; 52.9% of total neurological deficit cases). From the total of 34 patients who were affected with neurology (Table 11), the most affected nerve trunk was the common peroneal nerve (52.9%, n=18), followed by the median and ulnar nerves (29.4%, n=10) and others (17.6%, n=6), reflecting the higher anatomical prevalence of tibial and lower leg fractures.

Table 10. Peripheral neurological involvement stratified by age group (N = 100).

Neurological Status	18–28 yrs (n)	29–39 yrs (n)	40–50 yrs (n)	Total (n)	Percentage (%)
Paresthesia/nerve irritation	18	11	5	34	34.0
Neurologically intact	27	24	15	66	66.0
Total	**45**	**35**	**20**	**100**	**100.0**

Table 11. Distribution of affected peripheral nerves among patients with neurological involvement (n = 34).

Affected Peripheral Nerve	Frequency (n)	Percentage of Neurological Cases (%)
Peroneal nerve	18	52.9
Median/ulnar nerves	10	29.4
Other peripheral nerves	6	17.6
Total	**34**	**100.0**

Discussion

This review, bolstered by empirical evidence of 100 patients hospitalized for bone fractures, demonstrates the crucial importance of nursing surveillance to prevent severe neurovascular injury in orthopedic trauma patients [8]. In our clinical sample, young adult patients between ages 18-28 represented nearly half (45.0%) of all admissions, while high-risk anatomic sites including tibia/lower leg (42.0%) and forearm/hand (28.0%) represented 70.0% of injuries. Additionally, 44.0% of patients were immobilized using casts or splints, and 34.0% had peripheral paresthesia or nerve irritation that affected peroneal (52.9%) and median-ulnar (29.4%) nerves. The clinical profile of the current study sample is consistent with previous epidemiologic literature, where young adult males and tibial or forearm fractures represent the main risk factors for post-traumatic neurovascular injury [3].

There is one myth in clinical practice that palpable distal pulse means sufficient tissue perfusion. According to pathophysiological and clinical research, microcirculatory failure and axonal ischemia develop at significantly lower intracompartmental pressures compared to the systolic arterial pressure [33]. For this reason, using pulselessness and paralysis as indications of the need for treatment leads to unacceptably delayed diagnosis after the 4-8 hours period of viable tissues [6]. On the contrary, considering the high frequency of early sensory signs of compartment syndrome that was recorded in clinical practice, nursing practice should be focused on examination of specific dermatomes (for example, sensitivity of the first dorsal web space for deep peroneal nerve) and pain assessment—pain disproportionate to the injury and passive muscle stretching [30].

In addition, EBN-based nursing interventions act as direct influencers on compartment hemodynamic physiology. Whereas limb elevation is the conventional method of treating uncomplicated post-

traumatic edema, elevating a limb suspected to have ACS above heart level results in reduction of local mean arterial pressure and worsening of hypoxia [7]. Neutral placement of the extremity, prompt bivalvation of circumferential casts, and cutting of any underlying padding is crucial immediate intervention that will safely take the patient to surgery [7]. Routine documentation of observation periods, elimination of subjective documentation through recording of objective parameters, and SBAR communication protocols are important elements of interprofessional response that will ensure that the patient does not develop Volkmann's ischemic contracture, neuropathy, and amputation [8].

However, limitations to the clinical data set must briefly be outlined. Since the patient group relied on a cross-sectional descriptive study design conducted within an inpatient setting without longitudinal intra-compartment pressure measurements or functional follow-up after discharge, it is impossible to make any causal connections between early paresthesia and confirmed surgical fasciotomy.

Conclusion

Neurovascular assessment in an organized manner is absolutely necessary in caring for patients with fractures, splints, and casts. Synthesis of literature and clinical information gathered from 100 hospitalized fracture patients demonstrates that there is a high prevalence of lower leg and forearm injury, immobilization by cast/splint, and early involvement of peripheral nerves such as the peroneal, median, and ulnar nerves. Nurses will be able to identify ischemic injuries by assessing for the patient's arterial flow, capillary return, relevant dermatomes of the peripheral nerve, muscle strength, and pain profile compared to a previously established baseline. Identifying the presence of disproportionate pain and pain with passive stretching, keeping the limb at a heart level position, alleviating the pressure from the cast, and performing an SBAR surgical referral will help nurses maintain neuromuscular integrity.

Declarations

Author Contributions: Saif Mohammed Fanar conceived the study and review topic, coordinated clinical data collection, performed the literature synthesis and descriptive statistical analysis, and drafted the manuscript and clinical pathway. Ohood Qahtan Ahmed contributed to data validation, critical revision, clinical nursing framework verification, and final manuscript approval. Both authors read and approved the final version of the manuscript.

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