

PERIPHERAL NERVE INJURIES ASSOCIATED WITH ANAESTHESIA*

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NERVE PALSIES following anaesthesia have been observed since shortly after the introduction of ether¹ Early writers blamed the toxic properties of the anaesthetic agent used

Budinger² in 1894 was the first to recognize that the majority of these post-operative neuropthies were due to malpositioning of the patient upon the operating table, with consequent stretching and compression of nerves His findings were soon verified by Krumm,³ Garrigues,⁴ and Horsley⁵

Clausen⁶ in 1942 and Ewing⁷ in 1952 emphasized the importance of stretching in injuries to nerves during anaesthesia

Further publications on this subject have continued to appear to the present time,^{8,9,10 11 12} but these preventable injuries still occur

THE CIRCULATION OF NERVES

Nerves are supplied by nutrient vessels at irregular but frequent intervals, the vascular supply being especially abundant in the region of the joints The vessels at the point of entering a nerve are of arteriolar size These immediately branch into slightly smaller arterioles which ascend and descend longitudinally in the interfascicular spaces, gradually becoming precapillary in type They anastomose with parallel vessels and with more distal and more proximal arterioles Finer capillary branches pass directy from them into the connective tissue septa of the fasciculi These capillary branches tend also to be longitudinally disposed, and they also branch and anastomose repeatedly The intraneural vascular anastomosis is in fact so abundant that long sections of a nerve can be severed from the extra-neural blood supply and yet remain viable¹³

PATHOGENESIS OF NERVE INJURY

Stretching or direct compression of a nerve results in ischaemia If this ischaemia is maintained for a sufficient period necrosis of the nerve occurs

In addition, stretching of the nerve causes rupture of capillary vessels and a consequent haematoma in the nerve bundle If the haematoma is small, the surrounding nerve fibres are merely compressed and may later recover However, if the haematoma is large, the neighbouring nerve fibres become completely necrotic Ultimately exudate and scarring of the fibrous septa of the nerve trunk may develop

We thus see that the ischaemia is of intraneural rather than extraneural origin

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AETIOLOGY

1 *General Anaesthesia*

The principal factor causing the majority of peripheral nerve injuries in anaesthetized patients is ischaemia of the intraneural vasa nervorum^{14,15} Such ischaemia results primarily from stretching of the nerve and secondarily from compression of a nerve rendered vulnerable by stretching

Both stretching and compression of nerves are more apt to occur in the anaesthetized patient for two reasons Firstly, muscle tonus is reduced and so the patient is more susceptible to unphysiological positions⁶ Secondly, with perceptive powers no longer intact, the patient is unable to complain of postural insults which normally he would not tolerate¹

Even in the conscious patient, abduction of the arm to more than 90 degrees in a steep Trendelenburg position becomes painful and intolerable after a few minutes¹¹ and the radial pulse disappears in 83 per cent of such cases¹⁶ Only 30 to 40 minutes of anaesthesia with a patient in an unfavourable position may be sufficient to result in a nerve palsy¹¹

2 *Spinal Anaesthesia*

The low cerebrospinal fluid pressure which may result from a dural leak is probably the cause of the cranial nerve palsies which have occurred after spinal anaesthesia The low pressure is presumed to result in the descent of the medulla and pons and so to cause stretching of the cranial nerves^{17,18 19}

3 *Local Injections*

Nerve palsies can result from the injection of substances into or around a nerve The injury may be due to the chemical irritation of the injected substance such as a local anaesthetic agent, thiopentone, noradrenaline, chlorpromazine, or *d*-tubocurarine chloride A probing needle may directly traumatize a nerve The radial and sciatic nerves have been injured by misplaced intramuscular injections Bacterial contamination at the site of injection may be a causative factor Finally, local haematoma formation from perforation of an adjacent vein may compress a nerve sufficiently to render it ischaemic^{20 21}

4 *Hypothermia*

Hypothermia may be followed by a peripheral neuropathy²² because with present techniques of surface cooling far lower temperatures are recorded peripherally than in the rectum or oesophagus, for example, 4° C in the gastrocnemius versus 28° C per rectum in one particular case²³ The addition of even a minimal pressure such as the rubber straps which hold the E K G electrodes in place probably also predisposes to nerve injury²⁴

5 *Hypotension*

Hypotension, either accidental or controlled, may produce a nerve palsy or aggravate one due to local factors by reducing blood flow to the nerves⁴

6 Tourniquet

A tourniquet has occasionally been known to damage the nerves over which it was applied if the pressure used was excessive or continued for a prolonged period^{4 14,25,26} However, it is not nearly so likely to do so as is malpositioning because the element of stretching is absent, while the compression is dissipated around the entire circumference of the limb and exerted generally through an abundance of soft tissues and not directly against a hard bony point

7 Toxicity

Damage to the nervous system may be produced by direct toxic action of the degradation products of anaesthetic agents or by other impurities contained in them As an example phosgene will be produced from chloroform on exposure to heat, and in the early days of chloroform anaesthesia this undoubtedly occurred in the presence of candles and gas jets In the presence of heat and an alkali such as soda lime, trichlorethylene breaks down to form dichloroacetylene, which is extremely toxic to the central nervous system and this, in turn, is converted by heat to phosgene and carbon monoxide²⁷⁻³⁰

CLINICAL FEATURES

Nerve damage may be apparent immediately after recovery from the anaesthetic or only after several days have passed

The patient complains of a variety of unpleasant sensations, hyperaesthesia, paraesthesiae, coldness, pain, and anaesthesia, in the areas supplied by the affected nerves Tenderness is often noted at the site of the injury itself⁶

Pareses and even frank paralysis, with loss of reflexes, develop in the muscles innervated by the damaged nerves

Evidence of loss of sympathetic innervation is seen in decreased sweating, diminished pilomotor activity and redness, and increased warmth of the skin

If early recovery does not occur, there is eventual muscle wasting, muscle contractures, stiffening of joints, demineralization of bone, diminution of subcutaneous tissue, atrophic shininess, and ulcers of the overlying skin and overgrowth and ridging of the nails³¹

A diagnostic aid useful in differentiating malingerers from those with true palsies is the use of a silver nitrate photographic plate If a normal limb is placed upon such a plate in the dark and the plate is then developed, a picture of the hand can be seen because the chloride in the sweat reacts with the silver nitrate to form insoluble silver chloride This reaction does not occur in denervated areas owing to loss of sweat production

Alternatively the area to be tested can be painted with a saturated cobalt chloride solution in 95 per cent alcohol The area where sweating occurs will change from blue to red³¹

PROGNOSIS

Recovery may be complete within a few days, weeks, or months, or permanent weakness, wasting, and sensory loss may remain

Power usually returns most rapidly in large muscles and slowest in small muscles used for fine movements. Skilled workers are, therefore, generally more handicapped than manual labourers.

The longer the period spent in an abnormal position, the worse is the prognosis.³² The use of muscle relaxants or hypothermia also restricts the prognosis.

Permanent sequellae are more frequent in patients with pre-existing disease of nerves, anaemia, hypovolaemia, arteriosclerosis,⁴ or electrolyte imbalance.³³

SPECIFIC NEUROPATHIES

1 *Upper Limb*

A *The Brachial Plexus*

Aetiology Of all groups of nerves the brachial plexus is by far the most vulnerable to damage from malpositioning during anaesthesia, for two reasons. First, the plexus has a relatively long, mobile, and superficial course in the axilla between two firm points of fixation—the vertebrae and prevertebral fascia above and the axillary fascia below. Second, the plexus lies in close proximity to a number of freely movable bony structures.

Stretching is the chief cause of damage to the brachial plexus,^{5,34,35} with compression playing only a secondary role, acting merely to form a fulcrum or stabilizing point around which the nerves must detour. Stretching of the brachial plexus is produced by any factor which increases the distance between the points of fixation above and below, for example

- 1 Dorsal extension and lateral flexion of the head to the opposite side widens the angle between the head and shoulder tip and so stretches the plexus^{36,5,6,9} (Fig 1)

- 2 Abduction, external rotation, and dorsal extension of the arm on an arm board, especially if it is at an angle of more than 60° to the operating table, or allowing the arm to fall off the side of the table, causes extreme stretching of the plexus^{6,9} (Fig 4)

- 3 Suspension of the arm from an ether screen with the patient in the lateral position (Fig 5)

- 4 Extreme abduction of the arms above the head with the patient in the supine position

- 5 Suspension of a patient by the wrists to prevent slipping in an extreme Trendelenburg position has been stated by some to heighten the tension of the plexus. This will be discussed more fully below.

The brachial plexus may be compressed against one or more of a number of bony prominences. For example

- 1 The plexus may be pinched between the clavicle and first rib when shoulder braces used to prevent the patient slipping downward in the Trendelenburg position are not properly placed over the acromio-clavicular joint, but are instead put rather too far medially, where they depress the clavicle downward and backward into the retroclavicular space^{2,6,9}. The plexus must now travel a devious "S"-shaped course, thus in effect increasing the distance between the upper and

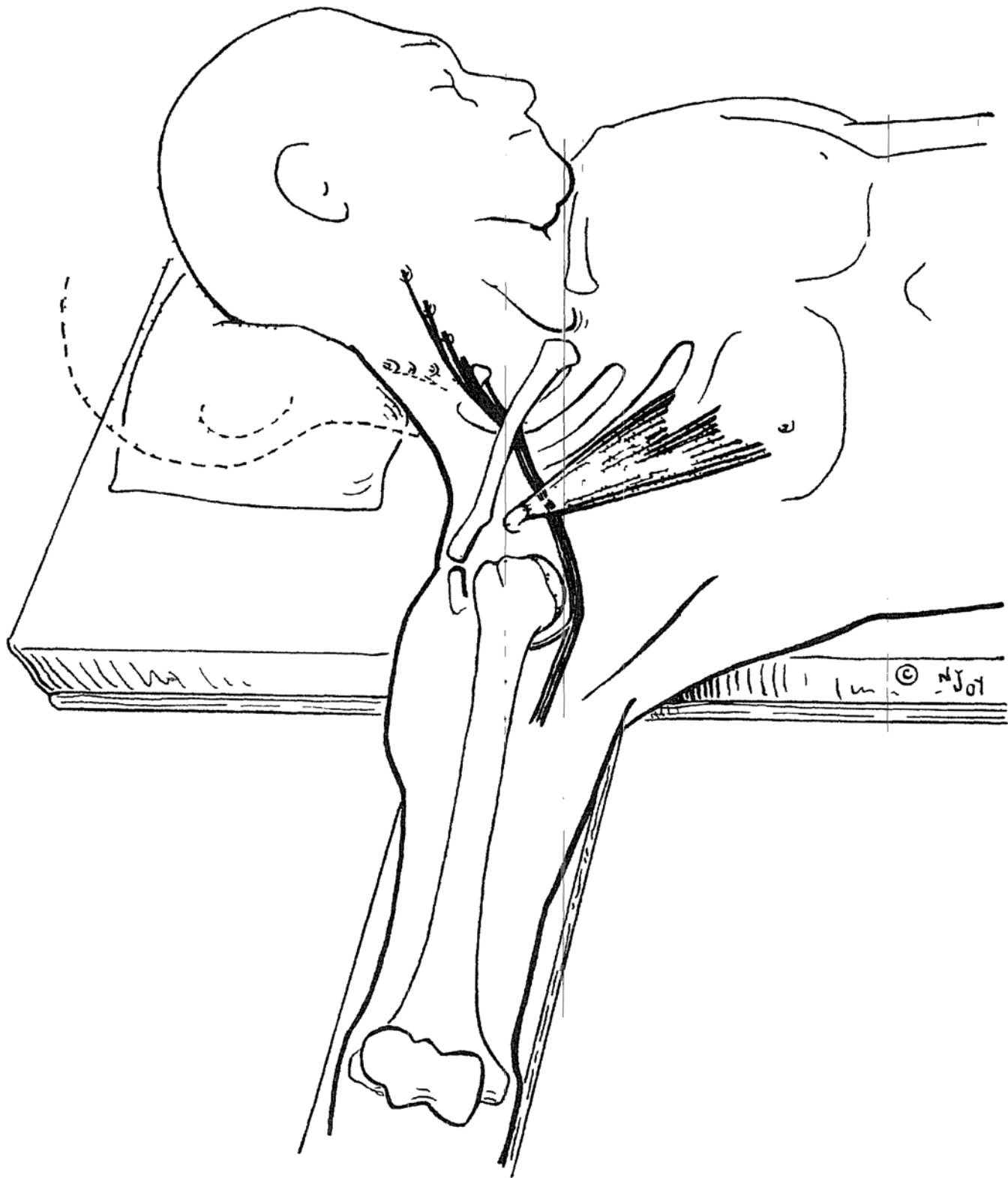


FIGURE 1 The brachial plexus is stretched when the arm is abducted, extended, and externally rotated, and the head is deviated to the opposite side

lower points of fixation. If, in addition, the patient's arm is abducted on an arm board as described above, stretching becomes extreme (Fig 2)

2 The plexus may be depressed downward by being stretched over the head of the humerus. This also occurs in the patient in the Trendelenburg position with the arm abducted on an arm board. The shoulder rests are in this case placed too far laterally and actually ride over the humeral head and so drive it downward into the axilla, carrying the plexus with it^{36,7} (Fig 3)

3. The plexus may be deviated posteriorly by the tendon of pectoralis minor or even by the tip of the coracoid process. This is apt to occur in two situations

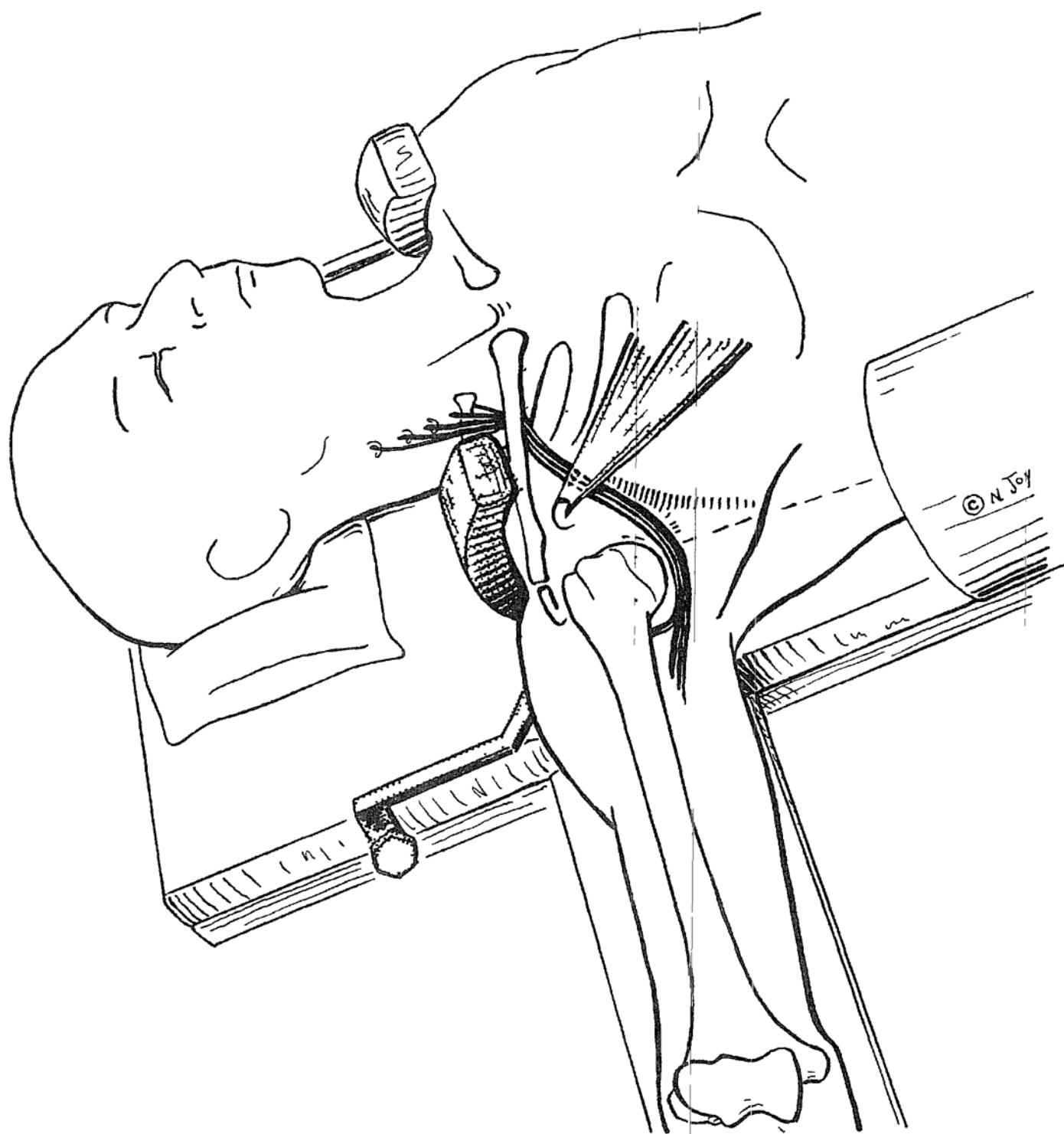


FIGURE 2 The brachial plexus is pinched between the clavicle and first rib by a shoulder rest placed too medially with the arm abducted, extended, and externally rotated

(a) In the rather obese patient undergoing a cholecystectomy in whom a gall bladder rest has been inserted and the head of the humerus on the same side is allowed to sag down off the operating table mattress onto an arm board which is not padded up to the level of the mattress. The entire shoulder girdle is depressed posteriorly and laterally in relation to the rib cage. The attachment of the tendon of pectoralis minor to the coracoid process and with it the brachial plexus are therefore deviated in the same direction¹⁰ (Fig 4)

(b) The same mechanism occurs in the patient lying on the side with the arm suspended from an ether screen⁶. Here the element of gravity accentuates the intraneural ischaemia (Fig 5)

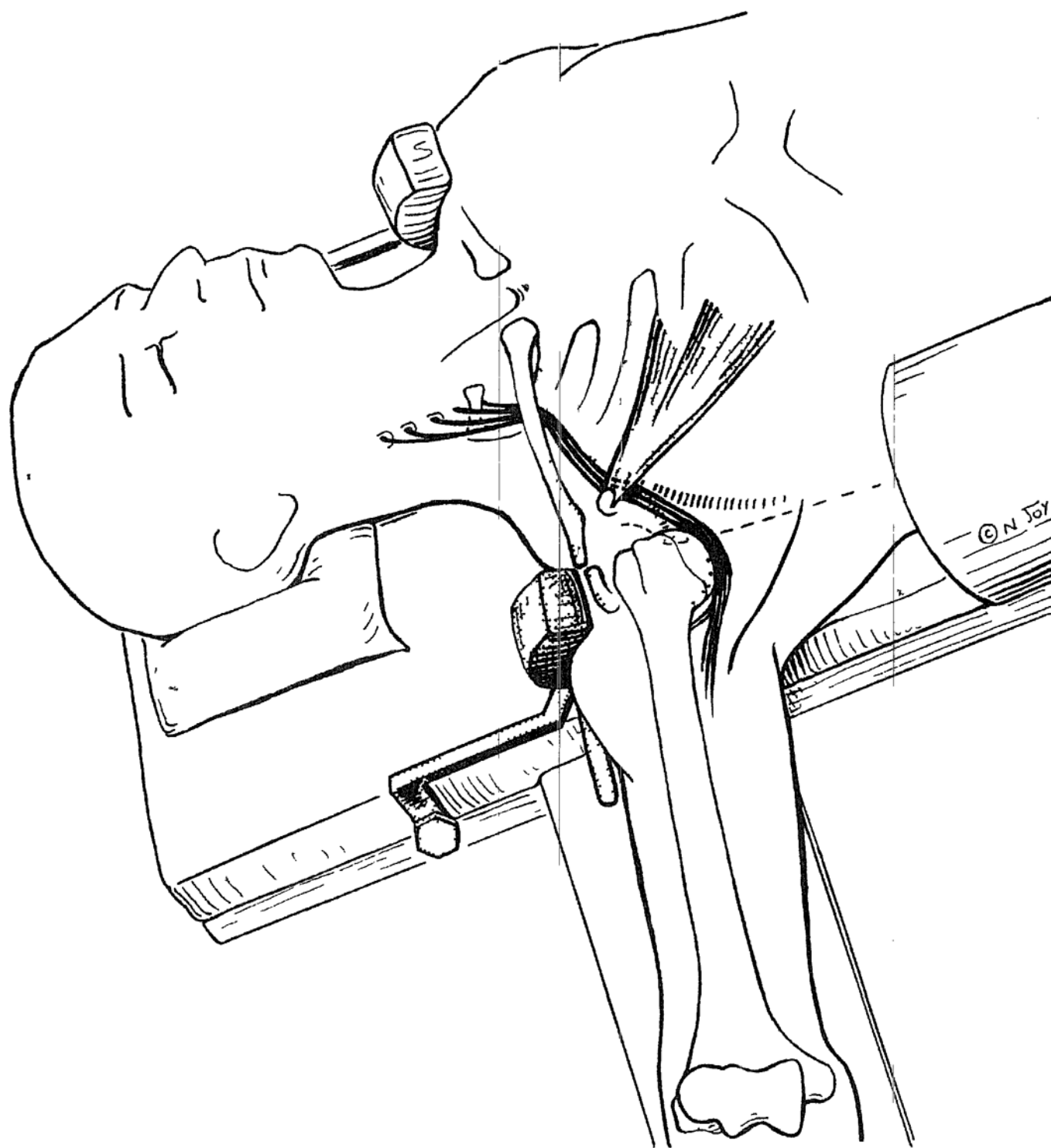


FIGURE 3 The brachial plexus is stretched by the head of the humerus depressed by shoulder rest placed too laterally. The arm is abducted, extended, and externally rotated.

4 Clausen⁶ believed that the first rib became the fulcrum over which the plexus rubbed when downward traction was placed on the adducted arm to prevent slipping in the Trendelenburg position. Ewing⁷ has disputed this and in the author's experience with the dissection of cadavers, there is no significant increase in the tension of the plexus in such a position, nor does the first rib in any way compress the plexus.

Certain congenital anomalies render the plexus more vulnerable to injury. These are

- 1 Hypertrophy of the scalenus anterior^{6, 37, 38}
- 2 Hypertrophy of the scalenus medius¹⁰
- 3 Cervical rib⁶
- 4 Anomalous derivation of the plexus higher or lower than normal⁶
- 5 Abnormal slope of the shoulder¹⁰

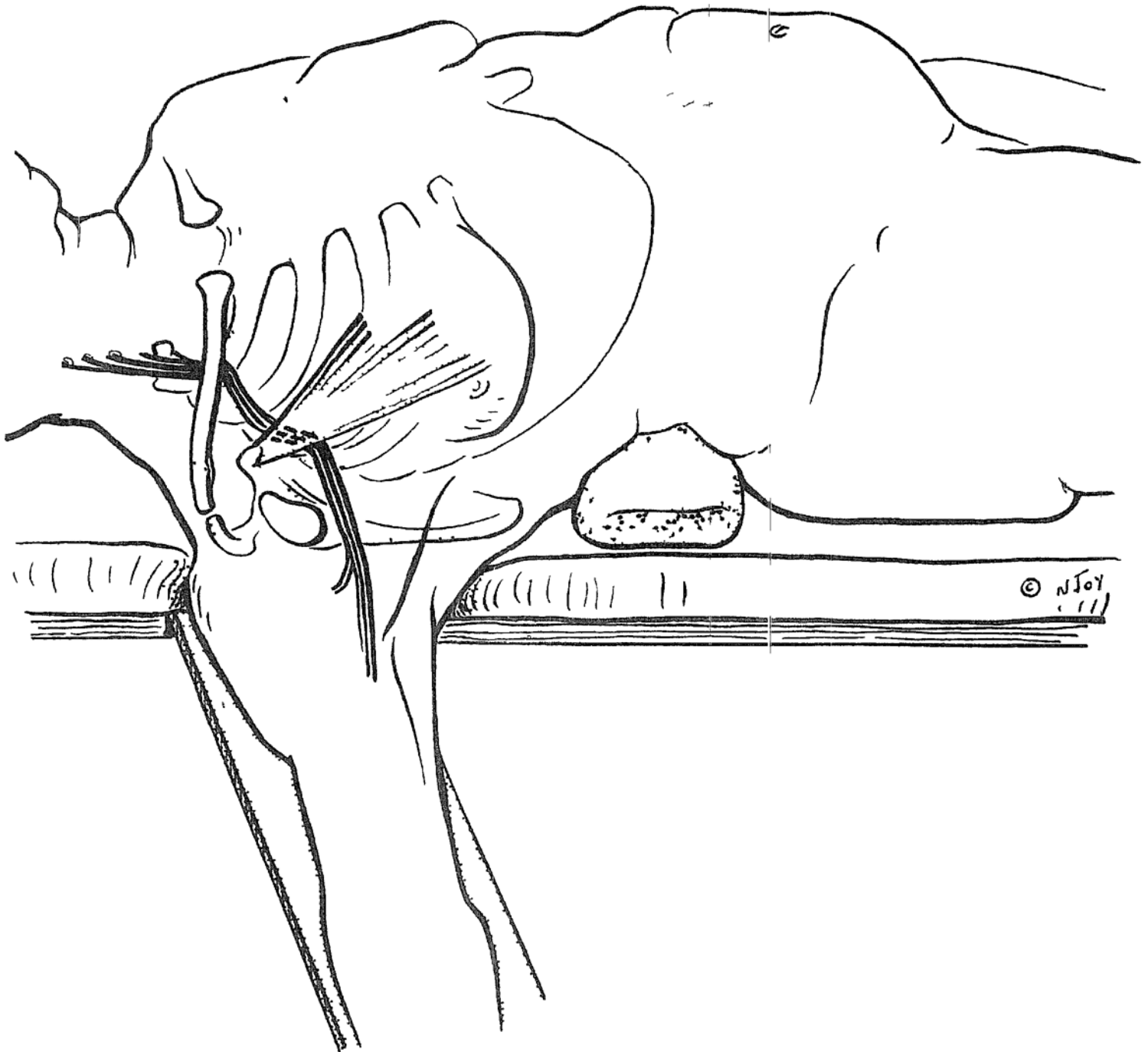


FIGURE 4 Stretching of the brachial plexus around the tendon of pectoralis minor when the shoulder girdle falls back with the arm abducted, extended, and externally rotated

Clinical Features Characteristically, there is shoulder pain and tenderness in the supraclavicular area one to several days postoperatively

The entire plexus may be involved, in which case the arm hangs flaccid and the skin of the whole limb is numb. The upper roots (C5, 6, and 7) only may be injured, with consequent internal rotation of the arm, extension of the forearm, and pronation of the hand (Erbs palsy). More rarely the lower roots (C8 and T1) alone may be affected with loss of flexion of the fingers, paralysis of the hand muscles, and perhaps Horner's syndrome (Klumpke's paralysis).

Involvement may be chiefly confined to one of the cords. With posterior cord damage there is loss of abduction of the arm and paralysis of the extensors of the elbow, wrist, and fingers. With lateral cord involvement there is paralysis of the flexors of the elbow and wrist. With medial cord injury the lesion is similar to that affecting the lower roots.

Determination of exactly which area of the skin is numb may be very difficult,

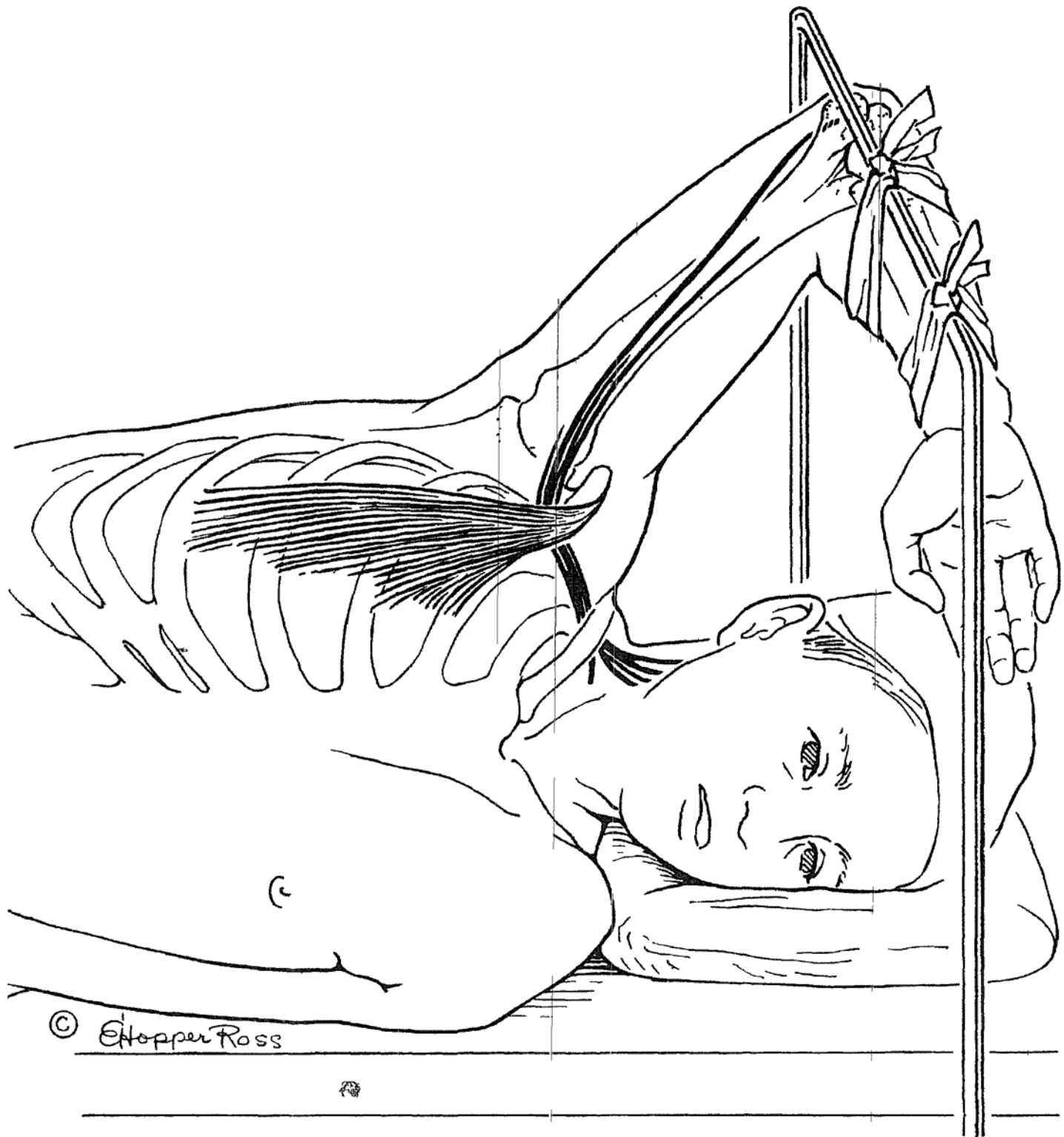


FIGURE 5 Stretching of the brachial plexus around the clavicle and tendon of pectoralis minor by fixation of the abducted arm to the frame of an "ether screen"

because of the extensive overlapping in the distribution of the cutaneous nerves. A simple method is to test the dorsum of the first web space for posterior cord damage, the palmar pad of the distal phalanx of the index finger for lateral cord nerve palsy, and the palmar pad of the distal phalanx of the little finger for medial cord anaesthesia.

B *The Radial Nerve*

The radial nerve may be injured as it traverses the brachium if the arm is permitted to sag off the side of the operating table. If the patient is horizontal the

plexus rubs against the table edge. If the patient is in the Trendelenburg position the arm tends to be pushed up against the ether screen as illustrated in Figure 6, thus pinching the nerve between the screen and spiral groove.⁹

Clinically there is wrist drop and inability to extend the metacarpo-phalangeal joints because of paralysis of the extensor muscles of the forearm. Weakness of abduction of the thumb results from paralysis of the abductor pollicis longus. The dorsal surface of the lateral three and one-half fingers and adjacent hand show varying degrees of numbness, dryness, increased warmth, and redness.

For medicolegal purposes this injury should be differentiated from an intramuscular premedication given below rather than into the deltoid. An injection given into such a location may easily damage the radial nerve in the spiral groove.

Infusion of thiopentone into the vein passing up the lateral side of the wrist may damage the underlying superficial radial nerve,³⁰ with resulting numbness on the dorsum of the thenar web.

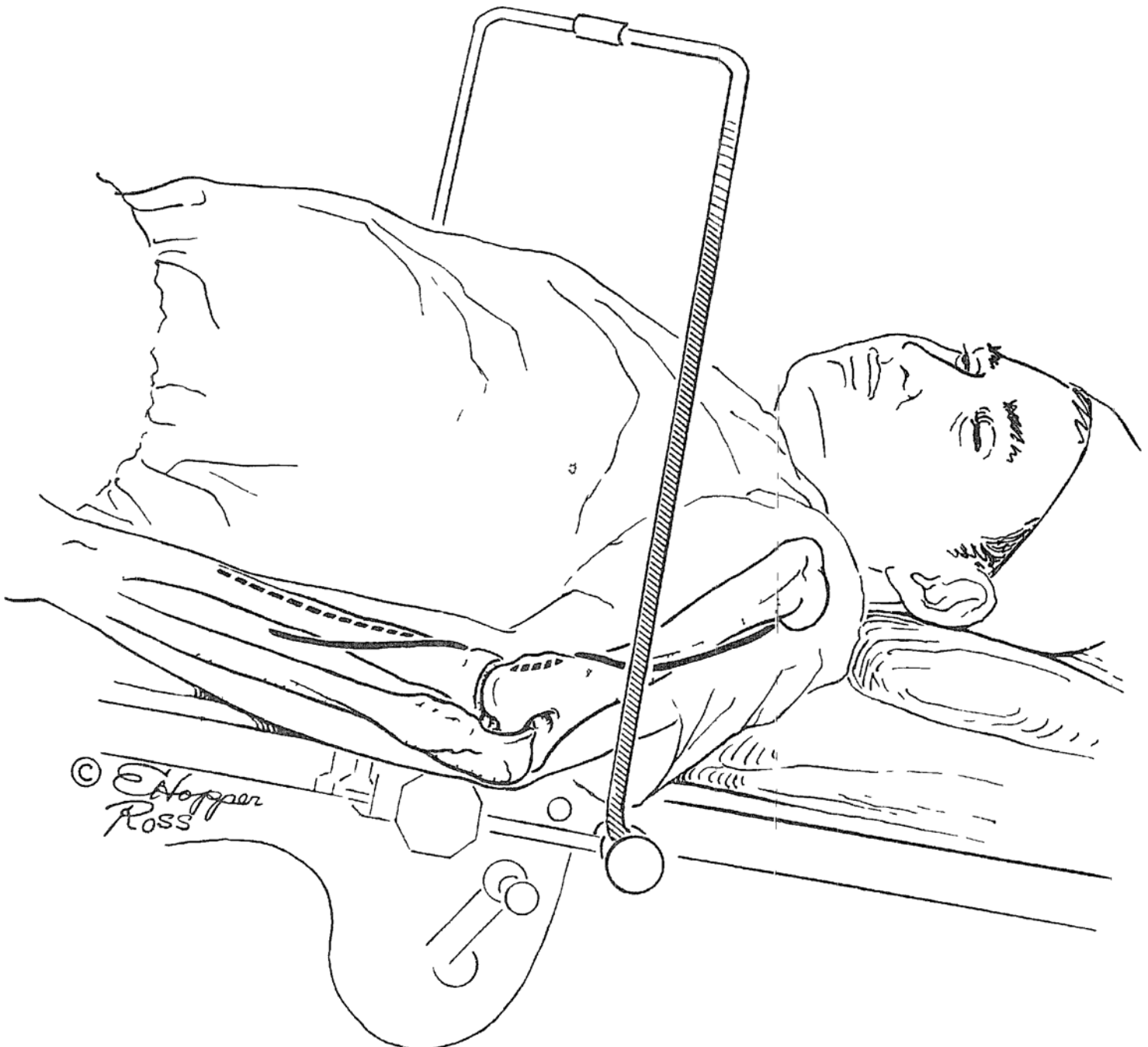


FIGURE 6 Compression of the radial nerve between the humerus and an ether screen

C The Ulnar Nerve

The ulnar nerve may also be compressed against the posterior aspect of the medial epicondyle of the Humerus⁴¹ The compression may be caused by the sharp edge of the table itself if the elbow is allowed to sag just slightly over its side Injuries to the ulnar nerve have also been reported following operations in which the patient's arm was folded across the abdomen or chest, the stretching of the nerve around the medial epicondyle of the humerus by acute flexion of the elbow and the pressure exerted by the weight of the arm itself apparently being sufficient to produce ischaemia This is perhaps because in over 20 per cent of cases the ulnar nerve pursues a more medial course than that described in standard texts, passing behind the backward projecting tip of the epicondyle rather than in the more protected groove (Fig 7)

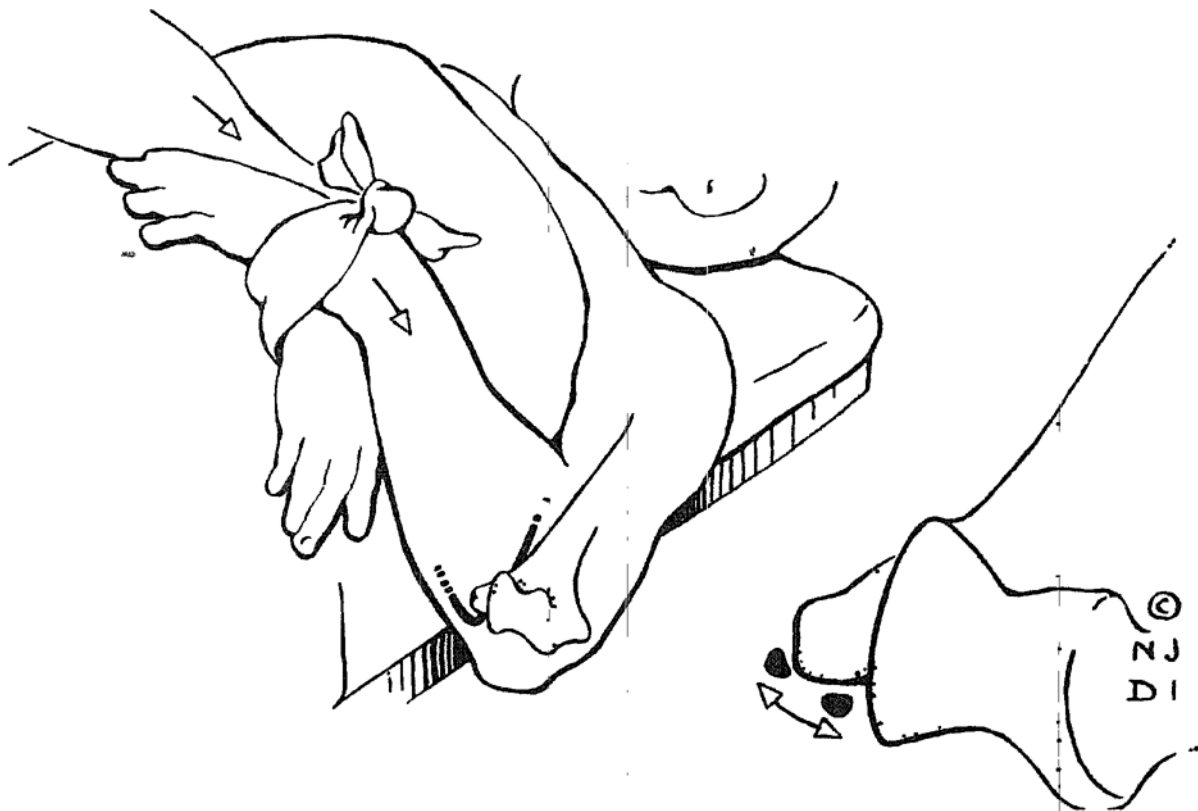


FIGURE 7 Ulnar nerve injury by compression between the medial epicondyle of the humerus and the edge of the operating table *Inset* Anomalous position of ulnar nerve

The grip on the ulnar side of the fist is weak owing to paralysis of flexor carpi ulnaris and the medial half of flexor digitorum profundus The digitorum profundus palsy also weakens flexion of the interphalangeal joints of the medial two digits Flexion of the metacarpal-phalangeal and proximal interphalangeal joints, extension of the distal interphalangeal joints, and abduction and adduction of the medial four digits when extended, are weak because of the loss of innervation of the interossei and the third and fourth lumbricals There is also inability to abduct or oppose the little finger because of paralysis of the hypothenar muscles Sensory and autonomic loss occurs over both surfaces of the medial one and a half fingers and adjacent hand Eventually the intrinsic hand muscles except for the thenar eminence become wasted and contractures develop resulting in a characteristic "claw-like" hand

D. The Median Nerve

The median nerve lies adjacent to the median cubital and basilic veins in the antecubital fossa and it may be damaged during intravenous injection of thiopentone,⁴² either by the direct trauma of the needle or by the extravasation of the thiopentone. Such damage may occur even after the needle has been successfully introduced into the vein and taped in place if the patient should flail his arm about because of too light anaesthesia (Fig 8)

On examination of the patient there is inability to oppose thumb and little finger (paralysis of *opponens pollicis*), weakness of abduction of the thumb (paralysis of *abductor pollicis brevis*), and loss of flexion of the distal phalanx of the index finger (paralysis of the lateral half of *flexor digitorum profundus*)

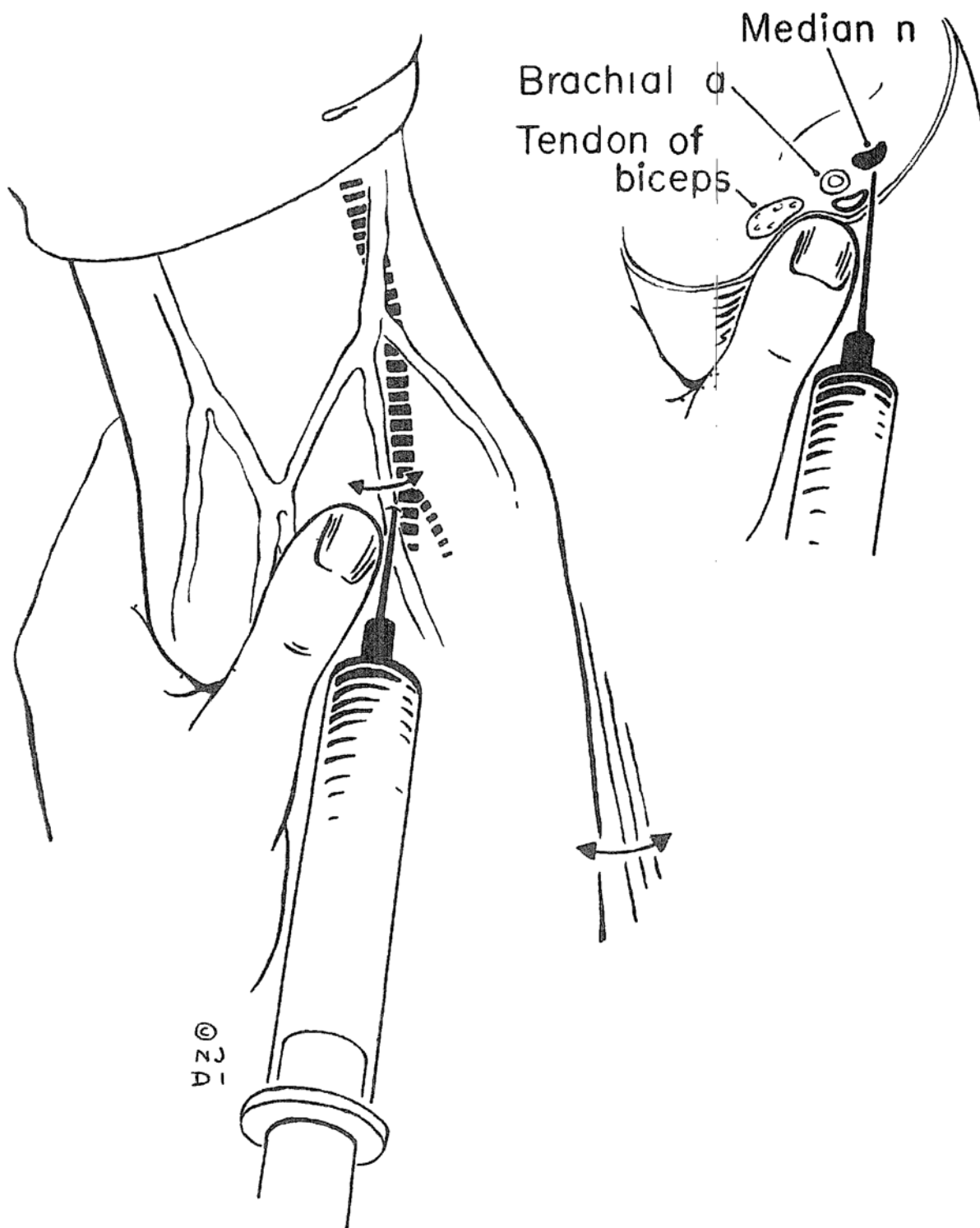


FIGURE 8 The median nerve may be traumatized by a needle in the antecubital fossa

Eventually the thenar eminence becomes flattened. Sensation and sweating are diminished on the palmar surface of the lateral three and a half digits and adjacent palm.

2 The Lower Limb

A The Common Peroneal Nerve

The common peroneal nerve is the most frequently damaged nerve in the lower limb, although injuries to it are not nearly so common as those to the brachial plexus. It may be compressed against the head of the fibula in the lithotomy position.⁴³ In such a position the flexion of the hips and knees stretches the nerve.

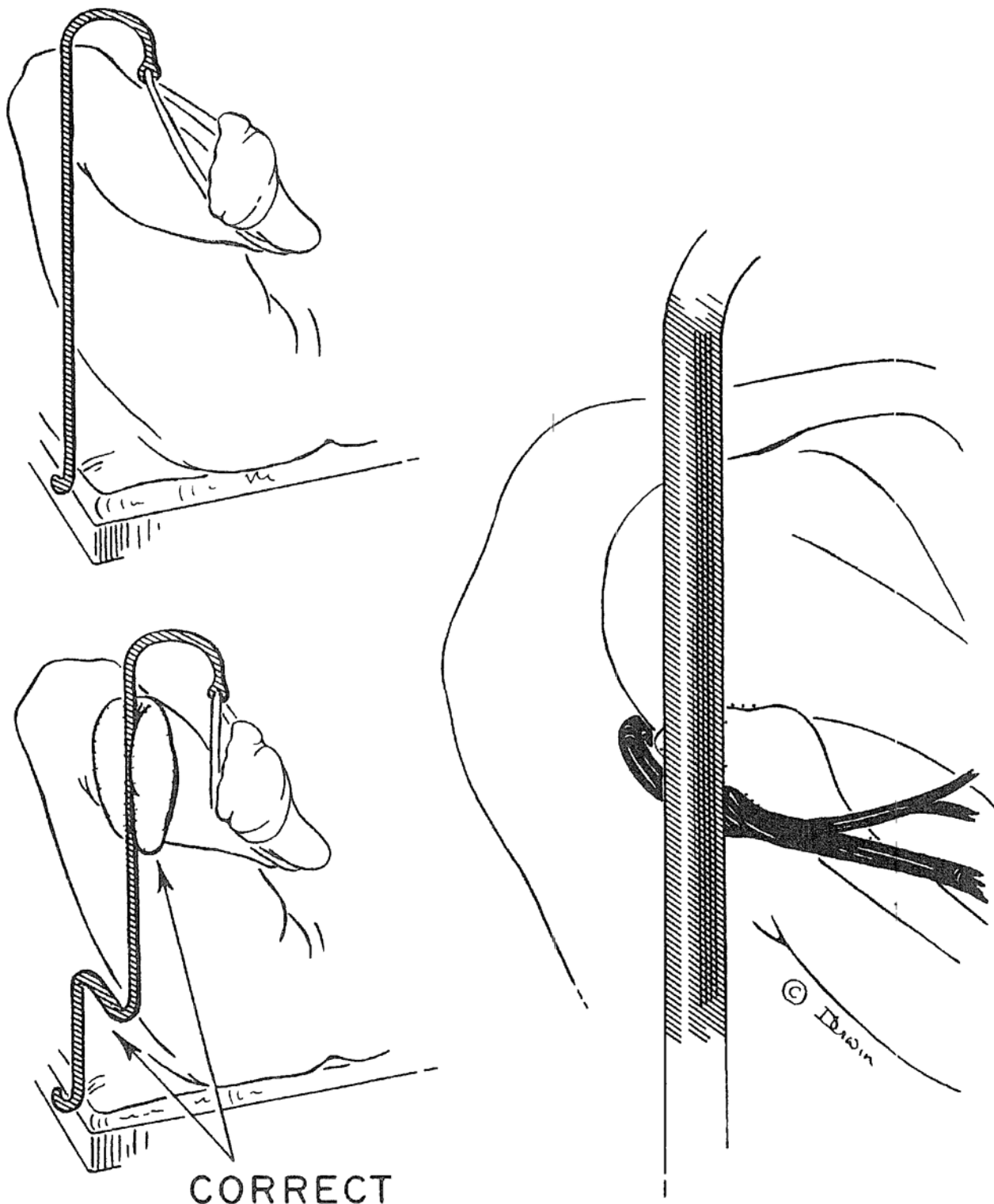


FIGURE 9 Common peroneal nerve compressed between lithotomy stirrup and neck of fibula. This may be avoided by padding.

The neck of the fibula rubs against the vertical metal brace from which the supporting foot strap is slung or against a curved metal support under the knee¹ and the already overstretched nerve is pinched (Fig 9) A similar injury may be produced by hard knee rolls or a tourniquet¹

The physical findings are foot drop (paralysis of tibialis anterior, extensor digitorum longus and extensor hallucis longus), loss of dorsal extension of the toes (paralysis of extensor digitorum longus and brevis and extensor hallucis longus and brevis), inability to evert the foot (paralysis of peroneus longus and brevis), and numbness of the lateral and anterolateral aspects of the calf and medial half of the dorsum of the foot

B *The Saphenous Nerve*

The saphenous nerve may be similarly pinched against the medial tibial condyle if the foot is suspended lateral to the vertical brace¹ (Fig 10) Paraesthesiae develop along the medial and anteromedial side of the calf

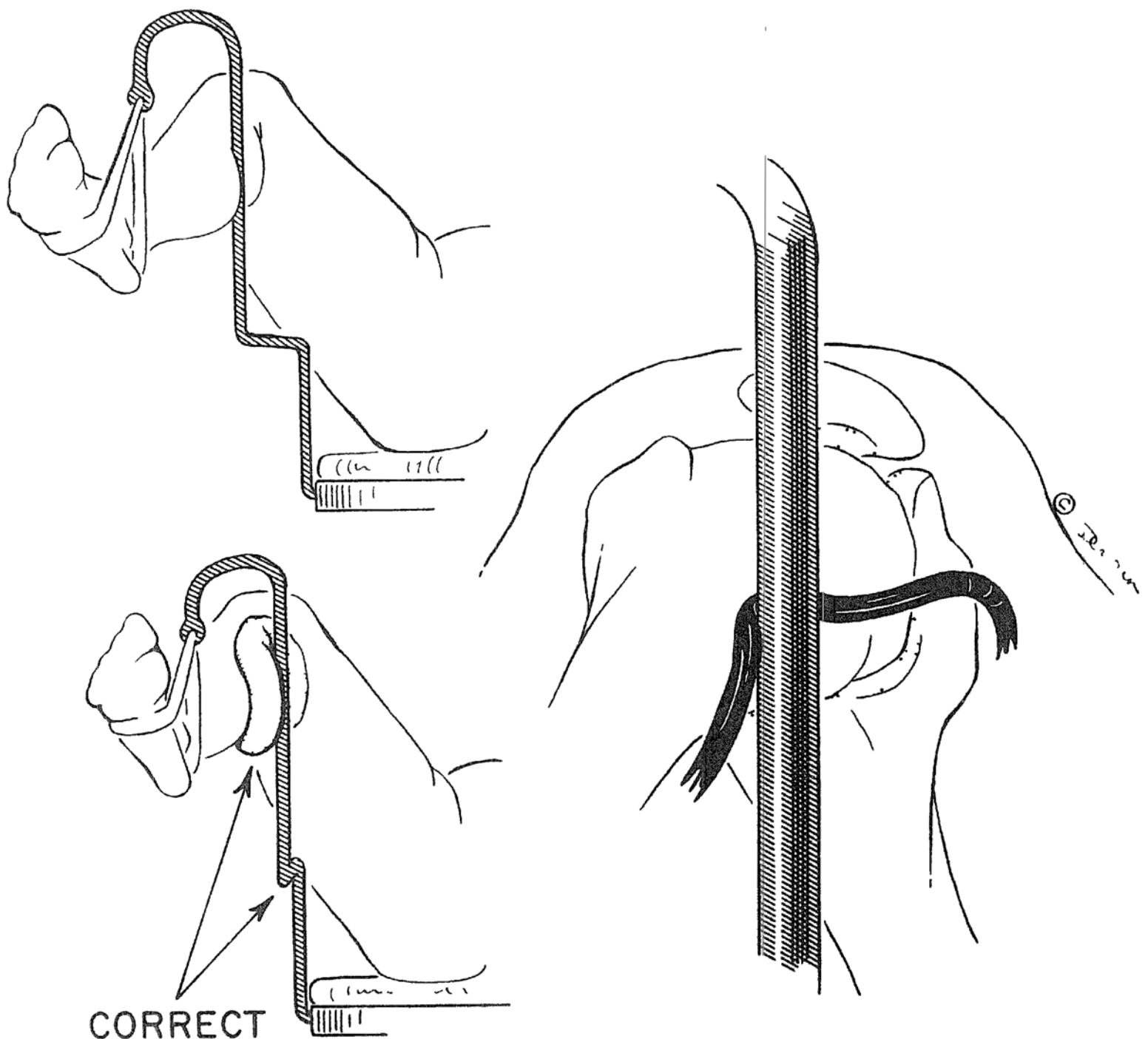


FIGURE 10 Saphenous nerve compressed between lithotomy stirrup and tibia

C *The Sciatic Nerve*

The sciatic nerve may be compressed as it escapes from under cover of the piriformis in a thin emaciated patient, lying on a hard table when the opposite buttock is elevated as in a hip-pinning procedure¹² It may also be traumatized by an intramuscular injection into the buttock.³⁰

There is paralysis of all muscles below the knee and perhaps also of the hams, as well as numbness of the lateral half of the calf and almost all of the foot, with the exception of the inner border of the arch.

D *The Pudendal Nerve*

The pudendal nerve may be pressed against the ischial tuberosity owing to traction of both legs against a poorly padded orthopaedic post, resulting in loss of perineal sensation and incontinence of faeces⁴⁴

E *The Femoral Nerve*

The femoral nerve has been damaged by pressure and lateral deflection by the leg of a self-retaining retractor used during a gynaecological laparotomy (Fig 11)

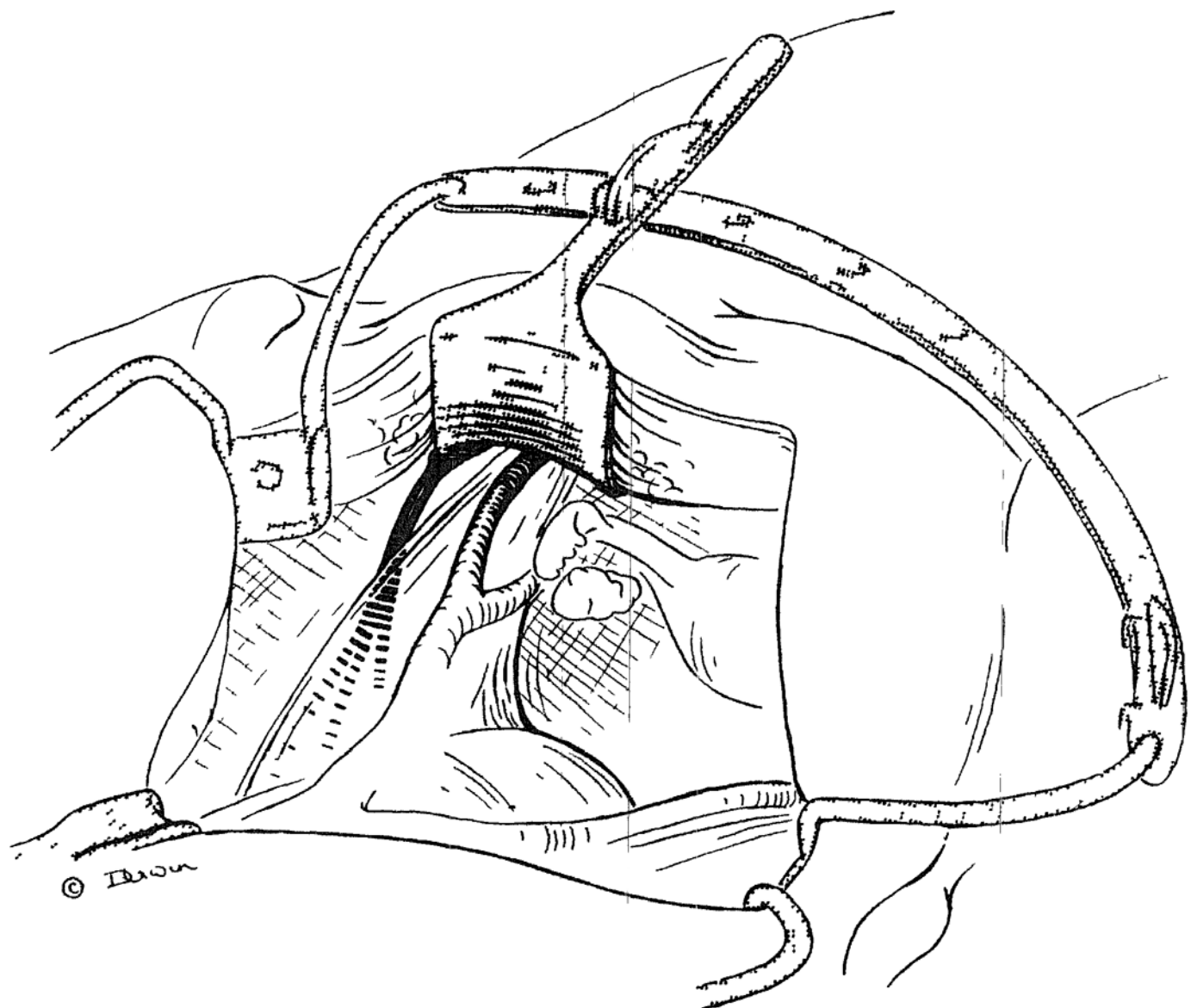


FIGURE 11 Compression of the femoral nerve at the pelvic brim by the blade of a self-retaining retractor

On examination there is loss of flexion of the hip and extension of the knee due to quadriceps femoris palsy. Sensation and automatic function are lost over the anterior aspect of the thigh and medial and anteromedial side of the calf.

F *The Obturator Nerve*

A case of obturator nerve palsy with paralysis of the adductors and numbness over the medial side of the thigh has been seen by the authors following an epidural anaesthetic. The patient had had a difficult forceps delivery and the obstetrician recalled having to "pull harder" on the forceps than he had ever done before. The aetiology in this type of injury is therefore thought to be of obstetrical rather than anaesthetic origin.

3 *The Trunk*

A *The Phrenic Nerve*

The phrenic nerve may be traumatized during the performance of a brachial plexus block by the supraclavicular route with consequent paralysis and elevation of the diaphragm.⁹

B *The Stellate Ganglion*

The stellate ganglion may be similarly injured, resulting in Horner's syndrome—miosis, enophthalmos, lid lag, and anhidrosis.

C *The Lumbar Spinal Nerves*

Damage to these nerves may occur during an attempted lumbar sympathetic block, with subsequent pain along the distribution of the affected nerve.

4 *The Head*

A *The Optic Nerve*

Damage to this nerve has most serious consequences for the patient and unfortunately is not too uncommon. Pressure against the eyeball, especially during controlled or accidental hypotension, causes thrombosis of the central retinal artery with blindness upon awakening from the anaesthetic. This injury is an exception to the rule that the ischaemia is of intraneural origin. In addition there may be disorganization of the entire globe.

Such pressure is very apt to occur in the prone position and it is in this very position that controlled hypotension is often used, for instance for a spinal fusion or a Harrington rod insertion. Walkup reports two cases of unilateral blindness following pressure on the eye where a horseshoe headrest was used for pulmonary resection in the prone position,⁴⁶ and the authors have knowledge of a similar case produced by this type of headrest during a neurosurgical procedure. Severe compression of the eye also occurs if a Bailey head rest slips over the eye during a posterior fossa bone flap in the sitting position.¹ Blood flow in the central retinal artery is likely to be already reduced because of the head-up position (Fig 12). Similar injury to the eye may be produced by the pressure of a face mask which is too large for the patient.

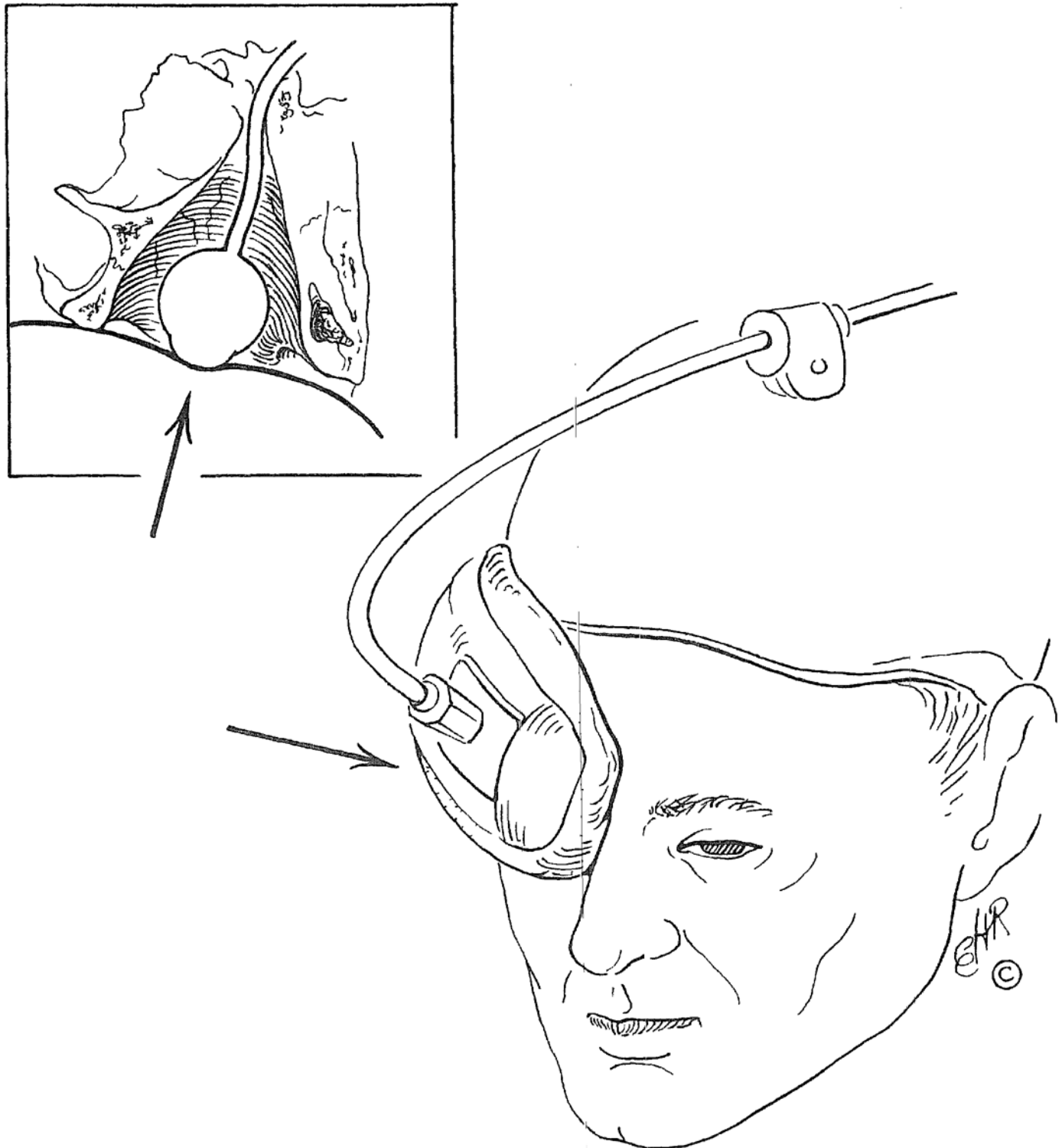


FIGURE 12 Ocular compression by a misplaced Bailey headrest

On examination within the first 24 hours the pupil is dilated and reacts consensually but not directly to light. The cornea is slightly hazy and the lids often oedematous. The retinal arterioles are dilated and the venules engorged. There is oedema of the macula and the retina surrounding the optic disk. If the damage is severe there may be a cherry red spot in the fovea. After several days the optic disk becomes pearly white. The arterioles narrow to white threads. The macula and the peripapillary retina become diffusely pigmented and faint radial scarring appears in the fovea.⁴⁷

B *The Supraorbital Nerve*

This nerve can be compressed by the mental connector of an endotracheal tube if insufficient padding is used (Fig 13). There is photophobia, numbness of the forehead, and pain in the eye.⁴⁸

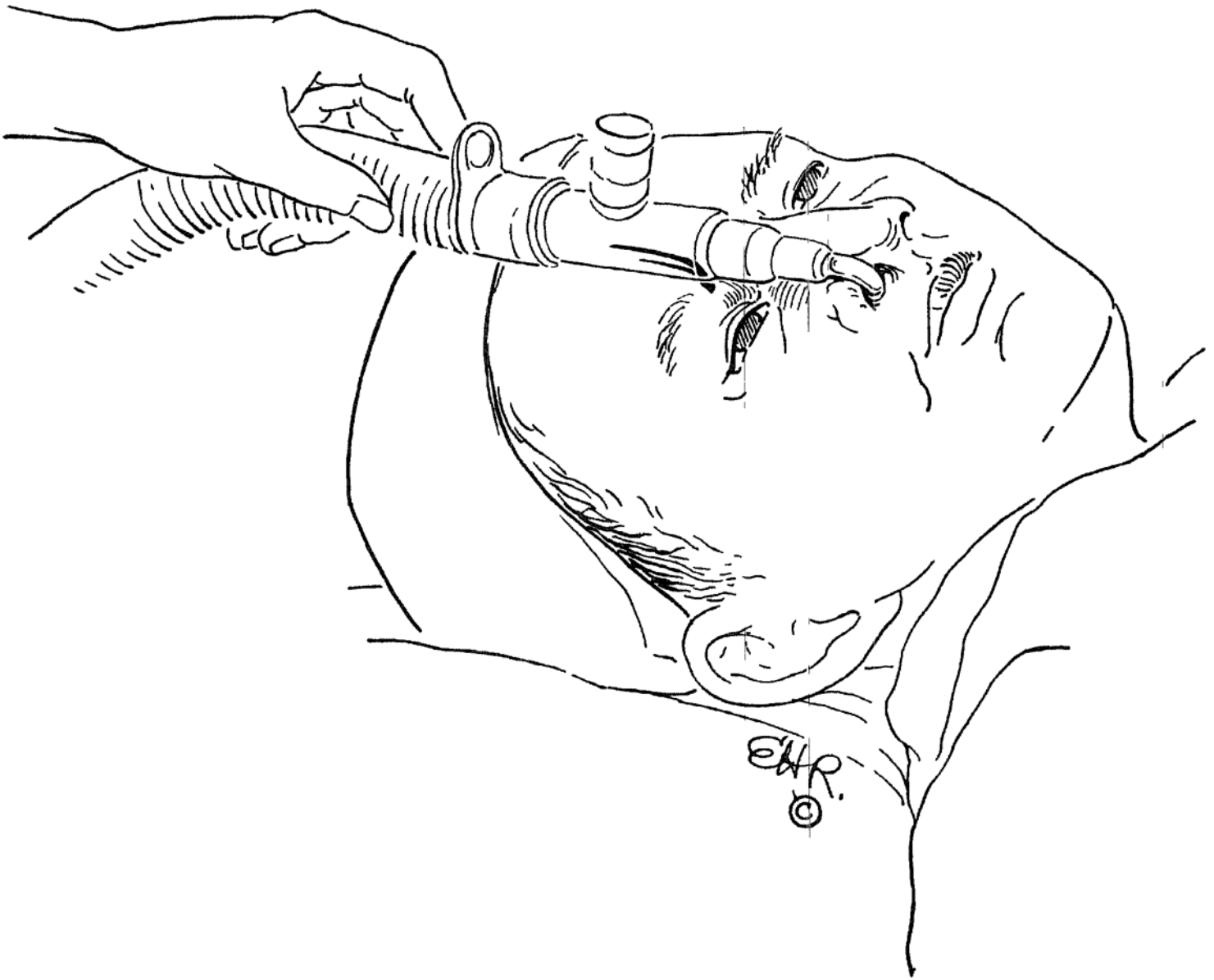


FIGURE 13 Injury to the supra-orbital nerve by pressure of an endotracheal tube connector

C *The Facial Nerve*

The facial nerve may be pinched between the fingers and the ascending ramus of the mandible if unusual forward pressure is required to maintain a clear airway (Fig 14)

The corner of the mouth sags, saliva drools, chewing is difficult, and the affected side of the face is smoother than normal⁴⁹

D *The Buccal Branch of the Facial Nerve*

There have been reports of injury to this branch by pressure from a too-tightly fitted mask or head strap. This is especially apt to occur if the course of the nerve is superficial to the parotid gland. There is loss of function of the orbicularis oris muscle³⁰ (Fig 15)

E *The Abducens Nerve*

Paralysis of every cranial nerve except the 10th has been reported following spinal anaesthesia, but in 90 per cent of cases the sixth cranial nerve is affected. The incidence is probably greater than the reports indicate, as many a slight case of paresis is likely to be overlooked when the patient only complains of a little

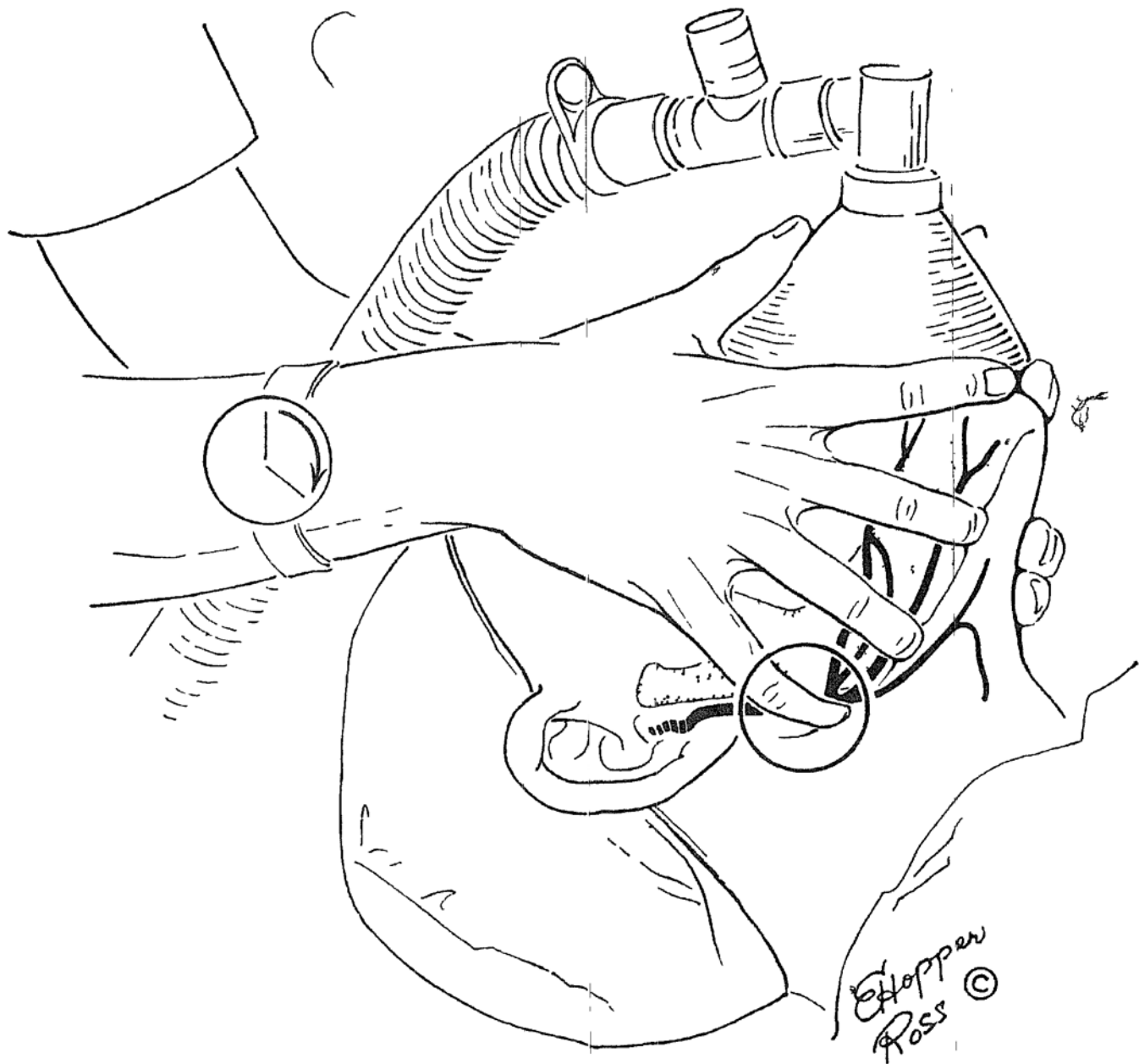


FIGURE 14 Motor root of facial nerve injured by traction on the angle of the mandible

blurring of vision which clears up in a few days. In the majority of reported cases there is diplopia, which is preceded by severe headache, stiff neck, nausea, dizziness, and photophobia. The onset of the palsy varies up to 21 days post-operatively,³⁰ and recovery may not be complete for up to two years.⁵⁰

F *The Trigeminal Nerve*

Trigeminal analgesia is the most common toxic manifestation of the degradation products of trichloroethylene. However, lesions involving all the cranial nerves except the eighth have been reported.

Numbness and coldness around the lips begins one to two days after the anaesthetic. During the next few days the area of the sensory loss spreads to involve the whole field supplied by the trigeminal nerve. Difficulty with chewing and jaw drop may be present. In addition, physical findings indicative of other cranial nerve palsies may be present. The mortality rate is high, being due to a toxic type of encephalitis. However, if the patient survives, complete recovery usually occurs in about eight weeks.^{28,29}

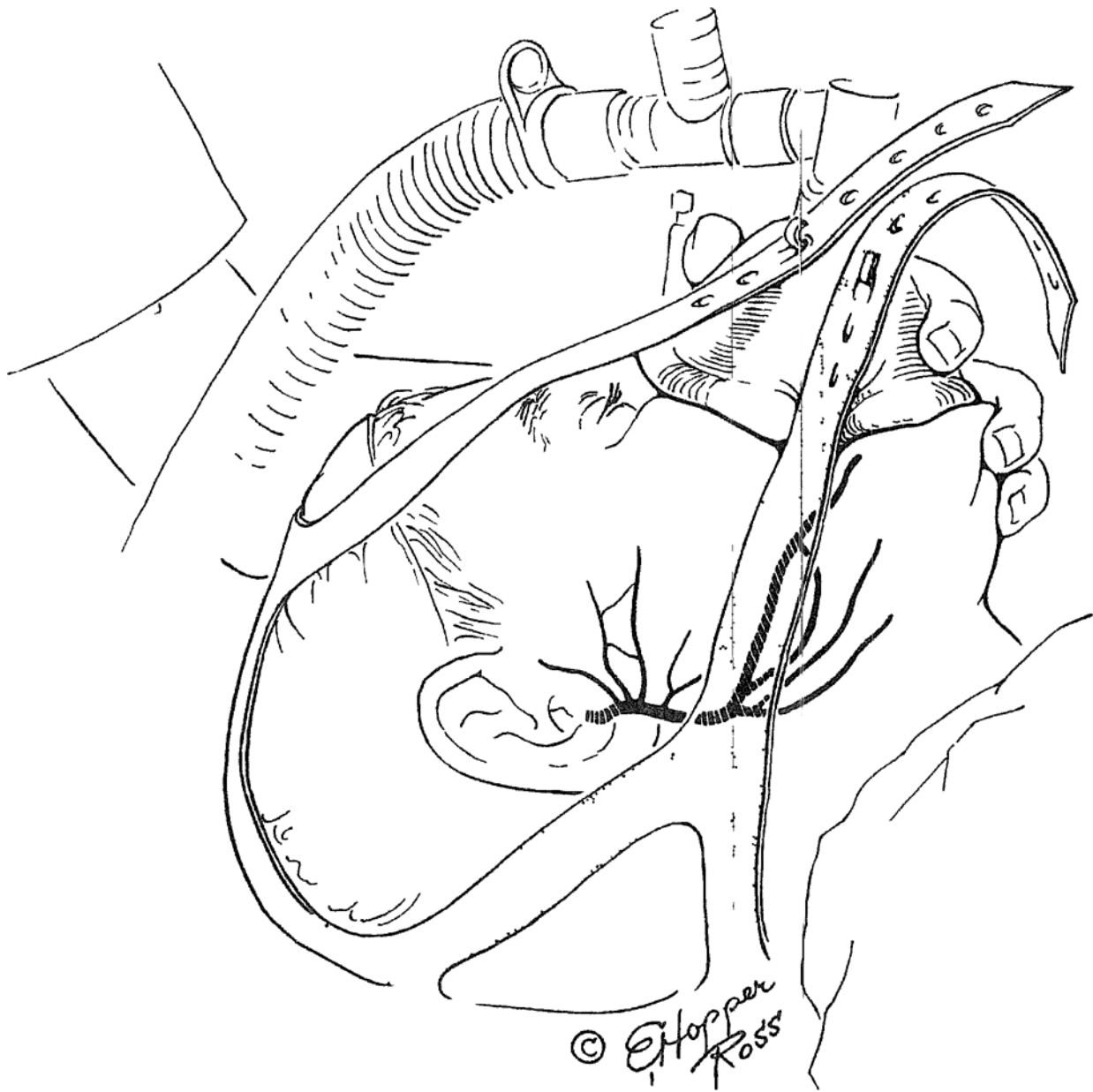


FIGURE 15 Buccal branch of the facial nerve injured by pressure of a tight head harness

TREATMENT

Prevention is of course the treatment of choice^{6,51} Arm boards are to be avoided especially in the Trendelenburg position Rather the arms should be kept tucked closely to the patient's sides with a draw sheet or a metal arm guard (Fig 16). If the latter is used, its inner side must in turn be well padded. If a draw sheet is used, overly obese surgeons must be discouraged from using the patient's arm as a resting place for their abdomens If abduction of the arm is absolutely necessary, the arm board must not be at an angle of more than 60° to the operating table, and its upper surface must be at the same level as the mattress The arm should be kept pronated.

Shoulder braces should be well padded and placed over the acromia and not over the clavicles Alternatively, special boots may be affixed to the bottom of the table to prevent the patient from slipping downward¹¹ A special non-slip mattress may also be used to achieve the same end^{52,53} The patient must never be suspended by the wrists The use of too steep a Trendelenburg position⁵⁴ and large gall bladder rests is to be discouraged

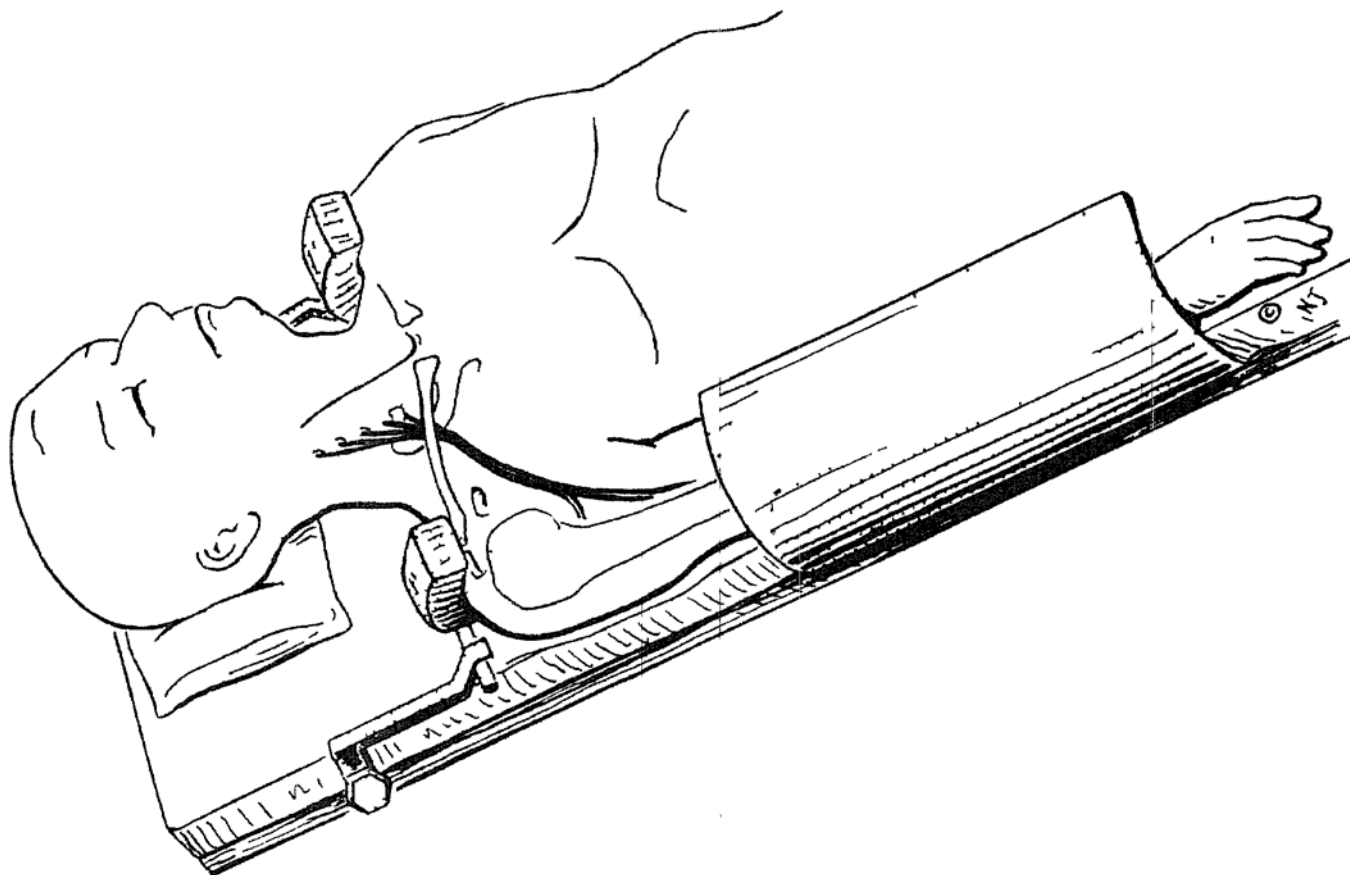


FIGURE 16 Correct position for patient in the Trendelenburg position

In the lithotomy position there must be ample padding between the legs and vertical metal braces or the supports under the knees. Orthopaedic posts should also be well padded.

In the region of the head, ample padding should be placed between an endotracheal tube connector and the patient's face. Mask straps must not be too tight. Excessive forward pressure against the angle of the jaw and unnatural head positions must be avoided. The eyes must always be free of pressure and it is wise to note this fact on the chart.

When performing regional or conduction anaesthesia, use a fine-gauge needle with rounded bevel edges,⁵⁵ which will press the fibres of the dura apart without cutting them.⁵⁶ Avoid excessive probing and ensure rigid sterility of technique.

A closed-circuit carbon dioxide absorption system is absolutely contraindicated with trichloroethylene or chloroform anaesthesia.

Avoid the use of an already neurologically damaged limb for either conduction anaesthesia or intravenous injection.

If a nerve palsy does occur, analgesics may be necessary for the first few days to control pain. The anaesthetic skin must be protected from injury and ulceration. Joint mobility should be maintained by massage and manipulation. Contractures may be prevented by splinting. Daily intermittent galvanic stimulation should be applied to the affected muscles. Exercise, at first passive and then active, should be commenced as soon as feasible.

If recovery does not occur after a thorough trial of conservative treatment and there is no response to galvanic stimulation applied proximal to the site of the injury, surgery must be considered. This may consist of excision of scar tissue.

to assist in regrowth of the nerve or of muscle and tendon transplants to compensate for lost function

With a sixth nerve palsy the affected eye should be covered to eliminate diplopia and prevent nausea. Eye muscle exercises and fusion training may also help. Surgical intervention may be necessary if recovery has not occurred after two years.

SUMMARY

The various types of nerve injuries incurred during anaesthesia have been discussed. The mechanisms of their cause, the physical findings which they produce, and their prevention and treatment have been outlined.

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