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REVIEW ARTICLE*

The arterial tourniquet

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The understanding of tourniquets for many users extends little beyond an appreciation of their mode of action and an awareness that they should not be left inflated for too long. A knowledge of the correct usage of tourniquets, of the complications which may occasionally follow their use, and of the safe duration of their application, is however desirable.

The time of application of venous tourniquets is usually brief and at a low pressure, whereas arterial tourniquets are applied for much longer periods and at greater pressures with consequently greater risks *(1)*. This review is concerned only with the use of arterial tourniquets.

History

The tourniquet, like many simple instruments and skills in medicine, has a long history, extending back at least to ancient Roman times *(2)*, although at that time it consisted of no more than a simple constricting band. It remained as such, apart from the introduction during the 17th century of various devices used to tighten the band in order to provide more effective haemostasis, until Petit, who coined the word 'tourniquet' *(2)*, invented the screw tourniquet early in the 18th century.

The next major advance in design did not appear for nearly 200 years when, in 1904, Cushing described the pneumatic tourniquet *(3)*. Cushing is generally quoted as advocating its use to minimize bleeding during craniotomy, but he also quite clearly describes employing a pneumatic tourniquet to provide a bloodless field for 'cocaine operations' on the hand, and discusses its advantages over the older type of elastic tourniquet. It is, of course, a refined version of Cushing's pneumatic tourniquet that is in general use today.

THE TECHNIQUE OF EXSANGUINATION

The concept of exsanguination of a limb prior to application of the tourniquet is closely linked with the development of the tourniquet itself. This manoeuvre reduces loss of blood still further but, equally important, it greatly facilitates surgery by providing a clear 'bloodless field'; indeed, it was an essential step in the development of surgery of the limbs and, above all, of the hand *(4)*. Exsanguination by simple elevation of a limb for a few moments before inflation of the tourniquet was regularly practised by Lister in the 1860s *(2)*. This remains an effective technique, but credit for the more generally used method of winding a strip of tensile material tightly around the limb is usually given to Johann Friederich August von Esmarch, Professor of Surgery at Kiel. He was, however, not the first person to use such a device. Esmarch himself conceded that his elastic bandage was merely a modification of a woven one used earlier by Grandesso-Sylvestri *(2, 5)*, although according to Tubiana *(4)*, quoting Oberlin, it was Nicoise who originally devised the woven rubber bandage and listed the indications for its use. Oberlin also considered Nicoise's bandage to be superior to the type used by Esmarch insofar as it was stronger and slipped less readily *(4)*. However, none of these names (Esmarch, Nicoise or Grandesso-Sylvestri) should be associated with the broad flat red rubber bandage used today for vascular compression of limbs. The original 'Esmarch bandage' was a rubber tube the thickness of a finger which was wound tightly around the limb to serve as a tourniquet after the blood had been expressed from it by bandaging. The 'Esmarch bandage' used today was designed by von Langenbeck, based on equipment used by Esmarch, and so is correctly termed a Langenbeck bandage *(5, 6)*; however, in order to avoid confusion, the conventional term, 'Esmarch bandage', is used in the course of this review. The Martin bandage, which is a similar device, is made of cream-coloured latex *(5)*.

The use of a pneumatic tourniquet

If a tourniquet is to be used, it should be both effective and safe. The purpose of a tourniquet is to apply sufficient pressure to the blood vessels of a limb to occlude the arterial inflow, but not enough to harm any of the structures compressed *(1)*. A comprehensive guide to its correct application is provided by Tubiana *(4)*, many of whose recommendations are incorporated below.

CHECKING THE EQUIPMENT

It is most important that the pressure gauge should be accurate. A gauge that reads low is particularly dangerous as its use may lead to over-inflation of the cuff and to an excessively high presure being applied to the patient's limb. The value of regular confirmation of gauge accuracy has been emphasised by several authors *(7–12)*. Bruner *(9)* and Flatt *(11)* both recommend daily calibration of the gauge against a mercury manometer before the start of the operating list.

It is also important to ensure that the gauge does not read high, that the tourniquet does not leak and that its connections are secure. Loss or inadequate control of haemostasis at a critical point during operation could impair surgical accuracy, and possibly do a grave disservice to the patient.

SITE AND APPLICATION OF THE TOURNIQUET

Tourniquets should be applied only to the proximal part of a limb where muscle provides protective padding for nerves and vessels *(4, 13, 14)*. It is particularly important to avoid bony prominences and epiphyses, against which tissues would be subjected to excessive compression.

Orthopaedic wool should be wrapped around the limb before the tourniquet is applied in order to avoid minor trauma to the skin *(4, 15)*; stockinette *(16)* or towelling *(6)* is a satisfactory alternative. Padding is of particular importance in the elderly or cachectic patient *(4)*. An exception to this rule is made in the case of the Conn tourniquet *(17)*, and

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some authorities suggest that any pneumatic tourniquet may be safely applied directly to the limb *(18)*.

The tourniquet should be applied closely to achieve effective tissue compression, and to minimize the risk of wrinkling with its potential for skin damage *(1)*.

TOURNIQUET PRESSURE

The patient's systolic arterial blood pressure should be checked before applying a tourniquet with a view to selecting an appropriate inflation pressure. It appears to be generally accepted that a 'suitable' pressure for an upper limb tourniquet is 250–300 mmHg and for a lower limb 500–550 mmHg, irrespective of the actual blood pressure of the patient *(19, 20)*; yet the reasons for selecting these particular values are seldom explained. Eckhoff in 1931 *(14)* suggested that 'a pressure of some 20 mmHg above arterial pressure ought to be sufficient' although he himself generally used a pressure ranging from 160 mmHg in young patients to 200 mmHg in the elderly. Sanders *(13)*, noting that 'the correct pressure is the minimal pressure required to produce a bloodless field', thought that the cuff pressure should not exceed preoperative systolic pressure by more than about 70 mmHg. His authority for this was Hinman *(21)*, who had reported this value as the maximum increase in blood pressure during surgery in his experience (and then only in patients who had suffered 'respiratory difficulty'). Adams *(18)* takes a broadly similar view, noting that cuff pressure should ideally be 30–50 mmHg above systolic pressure. He also comments that recommended pressures on most proprietary tourniquet sets are much higher than are usually necessary. Rorabeck *(22)* states that, in his experience, pressures of 100–150 mmHg above systolic are adequate to provide satisfactory haemostasis in most cases.

Brigden *(17)* suggests that a fixed pressure (maximum) of 275 mmHg in the arm and 550 mmHg in the leg is necessary to compensate for the padding effect of muscle and fat. He claims that these values are 'within safe limits' but, elsewhere in the same chapter, he advocates a pressure of 50 mmHg above systolic for the arm and 100 mmHg above systolic for the leg. The reasons for this alternative recommendation are not explained. Other advocates of a fixed tourniquet pressure unrelated to the systolic pressure of the patient include Spiegel and Lewin *(23)* who, in an article analysing three cases of peripheral nerve damage following the use of a rubber tube tourniquet, recommended the use of the Campbell–Boyd pneumatic tourniquet as being the least damaging to vessesl and nerves, and agreed with the designers *(24)* that, for the average adult, suitable maximum pressures were 10 psi (500 mmHg) for the arm and 15 psi (750 mmHg) for the leg. McElvenny *(1)* went even further claiming that he regularly employed pressures of 350 mmHg on the arm and 22 psi (approximately 1150 mmHg) on the thigh, with no ill-effects being reported over a 5 year period or longer.

Experimental work offers little help in identifying correct or suitable pressures. Griffiths and Heywood *(25)* carried out a theoretical analysis of the pressures and stresses within a limb subjected to tourniquet compression in 1973, and concluded that there was 'no simple relationship between the readily measurable tourniquet pressure at the surface of the limb and the stresses induced in nerves and vessels within the limb'. This agreed with earlier work by Denny-Brown and Brenner *(26)* who noted discrepancies between the tissue pressure and the external pressure when a tourniquet was applied to the limb of an animal. They attributed this to the variable rigidity of different tissue compartments within the limb. Sinclair had already noted in 1947 *(27)* that a pressure cuff does not necessarily apply a uniform pressure to all points within the limb.

There is thus no clear consensus or guide as to *correct* inflation pressures. It would, however, seem safest to relate tourniquet pressure to the patient's systolic blood pressure, and to inflate the tourniquet to a pressure of between 50 and 150 mmHg above systolic *(4, 13, 22)*, the lowest pressure being used on the arm, and higher pressures on the thigh, to compensate for tissue padding. The manufacturers of the Stille automatic tourniquet advise even lower limits than this—a pressure of only 25 mmHg above systolic for the arm, and a maximum of 75 mmHg above systolic for the broadest thigh. These values have apparently proved satisfactory.

An ideal solution to the problem created by using the minimum occlusive pressure would perhaps be a system which continually measured the systolic pressure, and adjusted tourniquet pressure accordingly to maintain a fixed differential between them throughout operation. This would ensure that the minimum pressure was exerted on the tissues, consonant with preventing loss of haemostasis should there be a rise in the arterial pressure during the operation.

EXSANGUINATION OF THE LIMB

A limb is usually exsanguinated prior to application of the tourniquet. This may be achieved by simple elevation of the limb as practised by Lister *(2)* and favoured by Tubiana *(4)*, or by the use of some form of tissue compression device such as Esmarch's or Martins's bandage, or a pneumatic exsanguinator such as the Northwick Park type *(28)*.

The Northwick Park device compresses the entire limb up to the site of the tourniquet by means of an inflatable sleeve; the tourniquet is then inflated and the sleeve removed. Burchell and Stack *(29)* preferred this method to the Esmarch bandage for several reasons. They considered that it was quicker and easier to apply and that it had less potential for tissue damage and they pointed out that it could be used on a sterile limb during an operation.

The Esmarch bandage is equally effective in exsanguinating a limb, but its use is attended by the real risk of tissue damage, especially as it is often applied by relatively untrained staff. It should be applied over a towel or padding *(17)* from the extremity of the limb to the site of the tourniquet and it is important to avoid twisting of the soft tissues of the limb by 'drag' produced by the elasticity of the bandage as this causes shearing stresses within the limb which may be harmful *(25)*. In the arm the radial nerve is at particular risk as it winds around the shaft of the humerus, while in the leg care must be taken to avoid injury to the common peroneal (lateral popliteal) nerve, which passes around the lateral aspect of the neck of the fibula.

Exsanguination is inadvisable distal to an abscess, for fear of spreading infective material, although exsanguination proximal to this should be safe *(4)*. Partial exsanguination only has been recommended in certain cases to allow ready identification of structures within the surgical field *(30)*. The problem of anatomical definition in a bloodless field was also noted by Thio *(31)* following an inadvertent arterial injury. He recommended periodical release of the tourniquet to improve definition and to ensure that haemostasis was adequate, although Flatt felt that this practice made definition worse, not better *(11)*.

Exsanguination of more than one limb at the same time results in an effective increase in circulating blood volume of up to 800 ml *(32)*. This is not a common procedure but it may be unwise in patients with a poor cardiac reserve *(13)*. Middleton and Varian *(33)* in their survey of the use of tourniquets among members of the Australian Orthopaedic Association reported a death resulting from cardiac arrest following bilateral leg exsanguination.

INFLATION OF THE TOURNIQUET

Inflation should be as rapid as possible in order to avoid venous congestion; slow inflation briefly blocks venous return while allowing the arterial inflow to persist thus producing a degree of congestion.

A suitably fast rate of inflation is probably best provided by an automatic tourniquet apparatus, of which there are

several available commercially. These devices instantly inflate the cuff to a pre-set pressure, which is then maintained throughout the operation.

DURATION OF ISCHAEMIA: 'TOURNIQUET TIME'

There is no simple answer to the perennial problem of how long a tourniquet may safely be left inflated, other than to emphasise that it should be for the shortest time necessary for the planned surgery. The ischaemia tolerance of tissues varies from patient to patient, so that what is acceptable in the fit young person may well not be so in the elderly patient, in a diabetic or in an arteriopathic patient *(9, 11)*.

Functional changes Several authors have commented upon the apparent relationship between the time during which a limb has been ischaemic and the functional changes observed postoperatively. Weingarden *et al. (34)* noted that, in patients undergoing meniscectomy with a tourniquet in place, a high percentage showed postoperative changes in the electromyogram (EMG), and that as tourniquet time increased, so too did the number of recorded EMG abnormalities. Similar findings were reported by Saunders and others *(35)* in a study of patients subjected to knee arthrotomy; changes were particularly likely if the tourniquet had been inflated for more than an hour. Shaw-Wilgis *(36)* concluded from an investigation of 50 patients who required upper limb surgery that the application of a tourniquet for more than 2 hours resulted in 'muscle fatigue' which may contribute to postoperative weakness. Nolan and McQuillan *(27)* claimed that the limit for (human) muscle ischaemia (before irreversible change occurred) was 6 hours, but quoted no evidence to substantiate this statement.

Histological changes A microscopic study in rabbits by Dahlback *(38)* suggested that changes in muscle structure may start to occur after as little as 30 minutes. However, another study, also in rabbits, by Santavirta *et al. (39)* did not demonstrate any morphological change following a 30 minute recovery period after 4 hours of total ischaemia. Twenty-four hours later, no significant changes in the muscle ultrastructure, apart from apparently minor ones in the mitochondria, were visible even under the electron microscope. Studies in other animals have also shown that 3 hours or more of ischaemia are necessary before significant changes start to occur *(40, 41)*. Virtually all the changes observed in the various series were reversible, although the time required was variable but, as with all animal studies, the data obtained are not necessarily applicable to man.

A much-quoted paper by Solonen and Hjelt *(42)* described an analysis of the histological changes in human skeletal muscle occurring in patients aged from 2 to 46 years who underwent hand surgery under tourniquet. They concluded that, although minor histological changes in muscle started to appear after about 35–40 minutes, the 'changes were mild . . . until about 2 hours after the onset of ischaemia'. None of their patients subsequently showed clinical evidence of irreversible muscle damage.

Metabolism The recovery of cellular function and energy metabolism following a period of tourniquet ischaemia lasting for 3 hours was investigated in dogs by Enger, *et al. (43)*. They noted the levels of adenosine triphosphate (ATP), creatine phosphate and lactate in the ischaemic muscle during and after the period of circulatory occlusion, and also recorded the transmembrane potential of the muscle cells over the same time. Changes in this potential are considered to correlate well with changes in membrane function. Their results indicated that after the period of ischaemia followed by restoration of the circulation for 1 hour complete recovery of cellular energy metabolism and membrane function had occurred. Enger and his co-authors concluded that tissue ischaemia could be safely tolerated for at least 2 hours.

Metabolic changes in human muscle following an ischaemic episode were studied by Karpf and others *(44)*, who reached broadly similar conclusions. They thought that 2 hours ischaemia time was well tolerated by skeletal muscle, and noted that adverse metabolic change appeared much more slowly in man than in some animals, a comment worth remembering when considering the results of animal studies.

Other studies in man include that of Santavirta *et al. (45)* who, on the basis of clinical and biochemical observations in 1000 patients, deemed a 2 hour limit perfectly acceptable, and that of Tountas and Bergman *(40)*, who concluded that 'no significant changes relative to ischaemia was seen in the muscle of human subjects at the end of two hours'.

Adams *(18)* suggests 1 hour for the upper limb, and 1.5 hours for the lower, and this opinion is endorsed in *Campbell's operative orthopaedics (19)*. Stewart *(20)* notes that 1 hour is the 'generally accepted' maximum period. Bruner suggests that up to 1.5 hours would be reasonable in a fit patient *(9)*. Flatt considered that 2 hours was an acceptable time *(11)*, a view shared by Tubiana *(4)*, although he regarded it as the absolute limit. The same opinion was expressed by the author of an editorial in the *Lancet (6)*.

The above evidence and the quoted opinions of experienced surgeons suggests that a 2 hour limit to a single application of the tourniquet would seem to be a reasonable choice, although it should, of course, be remembered that this applies to fit patients. A lower limit would be preferable in the elderly. Every effort should be made to keep the period of ischaemia to a minimum by, for example, fully preparing the operation site before inflating the tourniquet. The suggestion in a *British Medical Journal* editorial *(15)* that an experienced consultant anaesthetist can reduce tourniquet time by the induction of controlled hypotension shortly before deflation ignores the facts that, firstly, many operations involving the use of a tourniquet are the province of relatively inexperienced anaesthetists, and secondly, that many anaesthetists would question the use of induced hypotension, with its attendant risks, in this context.

If surgery necessarily goes beyond the suggested time limit, then either the limit must be exceeded, with the increased risk that this entails, or the tourniquet should be deflated for a period to allow the tissues to recover, as recommended by Tubiana *(4)*. The question then arises—how long should this recovery period be? Santavirta *(47)* demonstrated in rabbits that the oxygen tension (PO_2) of ischaemic muscle fell steadily during 2 hours of tourniquet ischaemia, yet recovered to approximately 85% of the control value within 10 minutes of release of the tourniquet. Shaw-Wilgis *(36)* investigated 50 patients, aged from 17 to 56, and reported that, in an exsanguinated ischaemic limb, the venous pH and PO_2 fell steadily, while the carbon dioxide tension (PCO_2) rose; after 2 hours the mean pH was 6.9. He suggested that further acidotic change could produce irreversible muscle damage, especially as the pH in muscle was probably even lower than the blood value. He also noted that 15–20 minutes were required with the circulation restored for the pH, PO_2 and PCO_2 to return to normal. Other metabolic and cellular functional changes in the ischaemic limb may well take longer to recover judging by animal experimental data *(48)*. Thus at least 15–20 minutes should be allowed for limb to recover if reapplication of the tourniquet is proposed after a 2 hour period.

Adverse metabolic changes during the period of ischaemia can possibly be reduced by using cold irrigating fluid and avoiding hot theatre lighting directed onto the operative field *(4, 9)*, although Sanders *(13)* implies that the latter has no effect. The value of deliberate hypothermia in reducing damage to the ischaemic tissues has been well demonstrated in animals *(49, 50)*, but so far seems to have found little clinical application in man.

The suggestion has also been made *(51)* that blood flowing into the ischaemic limb via the medullary canal, thus bypassing the tourniquet, may help to relieve the ischaemia, and so prolong the safe period of tourniquet application but experiments carried out in Rhesus monkeys led Klenerman

and Crawley *(51)* to conclude that this was not a valid assumption.

DEFLATION AND REMOVAL OF THE TOURNIQUET

The operative site should be firmly bandaged and the limb elevated before the tourniquet is deflated *(4)*. Deflation, like inflation, should be rapid to avoid the risk of venous congestion and, once deflated, the tourniquet should be removed, as also should the bandaging beneath it, which may also contribute to venous obstruction and congestion *(4, 11)*.

EFFECTS OF TOURNIQUET DEFLATION

Local effects The immediate effect is, of course, a return of blood flow to the ischaemic limb; this flow may be greater than normal due to reactive hyperaemia *(52, 53)*, with a peak flow apparently inversely related to the duration of occlusion *(53)*. More importantly, from the point of view of haemostasis, several studies have shown a diminution in the coagulability of the blood perfusing the limb.

Nakahara and Sakahashi *(54)* noted an increased bleeding tendency, increased fibrinolytic activity and reduced coagulability in blood returning to the hind limb of dogs following 4 hours of tourniquet ischaemia. They also noted that these changes were considerably reduced if the limb was cooled by 10 °C. Rutherford, West and Hardaway *(55)*, investigating the effects of haemorrhagic shock in dogs, suggested that a principal cause of changes in blood coagulation systems was the tissue acidosis induced by ischaemia, and showed that such changes could be ameliorated by the infusion of buffer such as THAM. Miller *et al. (56)* demonstrated intravascular coagulation and increased fibrinolysis in primate limbs after tourniquet application; they suggested that similar factors may contribute to the post-tourniquet bleeding sometimes seen in man. Larsson and Risberg *(57)* had already shown increased fibrinolytic activity in human limbs during tourniquet application, although they were unable to demonstrate any correlation between the duration of occlusion and the degree of change.

All things considered, therefore, the comment by Sanders *(13)* that 'there are no significant microscopic or haemotological changes in the ischaemic limb in . . . man during tourniquet times of less than four hours' is difficult to justify.

Metabolic and other changes in the plasma and ischaemic tissues were investigated by Larsson and Bergstrom *(52)*. Deflation of the tourniquet resulted in a transient increase in potassium concentration in the venous blood leaving the limb, and hence in the systemic arterial blood. The potassium was released from the ischaemic tissues. The levels returned virtually to normal within 30 minutes of deflation, and, although the arterial potassium concentration never reached a dangerous level, a venous concentration as high as 6.0 mml/litre was recorded in three patients.

Other changes observed included temporarily increased blood osmolality and an increased extracellular water content (oedema), which persisted in some cases for at least 48 hours. A study in dogs by Rorabeck *(22)* demonstrated a fall in the levels of blood sugar and of calcium in the ischaemic limb, and confirmed the fall in PO_2 and rise in PCO_2 noted by others.

Systemic effects Many authors have considered the effects of tourniquet application on the tissues of the ischaemic limb but relatively few have analysed the systemic effects of release of the tourniquet.

Modig *et al. (58)* studied 15 patients undergoing knee arthroplasty under lumbar epidural anaesthesia, who had had a tourniquet applied for up to 2.25 hours. Immediately after deflation of the tourniquet, they noted a significant drop in blood pressure (in spite of the rapid infusion of plasma expander), a fall in the arterial PO_2 and pH, and increases in serum potassium, lactate and arterial PCO_2. These changes generally reached their peak within 3 minutes of release of the tourniquet and thereafter rapidly returned to normal. No evidence of myocardial or pulmonary disturbance was seen. Modig and his co-authors therefore concluded that the systemic effects of tourniquet release were moderate and readily reversible. Their results incidentally confirm, in part, the work of Larsson and Bergstrom *(52)* which has already been quoted.

Hassan, Gjessing and Tomlin *(59)* observed changes in the blood gases following tourniquet release in healthy adult subjects under spinal anaesthesia. After 75 minutes of ischaemia (mean time) deflation of the tourniquet was followed by a fall in base excess, and a fall in the arterial PCO_2 lasting 20 minutes or more. The authors reported no ill effects in their patients as a consequence, but suggested that, as elderly patients tolerate acidosis poorly, such patients should perhaps be given a small dose of sodium bicarbonate just berfore the tourniquet is released.

The information available is limited, but there seems to be relatively little evidence that release of the tourniquet produces significant adverse systemic effects for which action needs to be taken in advance.

Complications associated with the use of tourniquets

NERVE INJURY

There is extensive literature on this complication, dating back to 1880 (Mantes, quoted by Spiegel and Lewin *(23)*). Reports encompass injuries of varying severity, but many are concerned with paralysis following use of a tourniquet—many of these cases of paralysis followed the use of a simple rubber band tourniquet, however, and not of a pneumatic type. (Although the latter was first described in 1904 *(3)*, rubber band tourniquets were still being used—and apparently causing damage—during the period 1945 to 1950 *(23, 26)*.

A widely held view regarding the cause of such paralyses was based on the work of Denny-Brown and Brenner *(26)* who, in a long and detailed paper, argued that ischaemia due to compression of blood vessels supplying the nerve bundles, was the primary cause of neural damage. They also demonstrated that, even under considerable pressure, these vessels were not always completely occluded, and thus paralysis did not invariably follow the application of a tourniquet. Their opinion has not been universally accepted, however. A number of authors, including Eckhoff *(14)*, McElvenny *(1)*, Moldaver *(60)*, Rudge *(61)* and Bolton and McFarlane *(62)*, after analysing the histories of patients who had suffered nerve injury following the use of a tourniquet, concluded that the neural damage was due to direct pressure of the tourniquet upon the nerve, rather than to the ischaemia it produces.

Experimental evidence strongly supporting this view has been presented by Ochoa *et al. (63)* who applied tourniquets to the hind limbs of baboons for periods of up to 3 hours. They demonstrated that nerve damage was maximal below the edges of the tourniquet where the nerve microstructure was seriously distorted by the 'squeezing' of the section of nerve under the edge into the adjoining section, the nerve just under the cuff being slowly invaginated to a greater or lesser degree into the adjacent distal (or proximal) part. The same authors, in a subsequent paper describing further experimental work *(64)*, reported that, in general, injured nerves could conduct impulses normally in sections peripheral to the site of injury—that is, those parts subjected to ischaemia while the tourniquet was inflated. Ochoa *et al. (63)* concluded that a pressure gradient existed between the tissues below the edge of the cuff and those beyond it, and that this gradient was responsible for the neural disruption and hence for the clinical signs of nerve damage. It is true that these authors produced their observed effects by using a tourniquet pressure of 1000 mmHg—considerably greater

than the pressures used in clinical practice—but their work nonetheless provided a convincing demonstration of the pathophysiology of nerve injury by tourniquets. It seems reasonable to assume that the lower pressures used clinically are less likely to cause such injuries, but that, when they do, the mechanism is the same. Ochoa *et al.* also suggested that the duration of conduction blockade—that is, the severity of neural injury—was related to the duration of tourniquet compression, and that the risk of damage occurring at all was related to the amount of pressure applied to the limb by the tourniquet. Support for these ideas is provided by the findings of Rorabeck *(22)*, who demonstrated impaired conduction in canine sciatic nerve following tourniquet application for periods of up to three hours. The degree of impairment and times required for recovery of function appeared to vary directly with the amount and duration of applied pressure. Neural damage was noted to be maximum for the section of nerve directly below the tourniquet and least for nerve distal to it.

Yet further support for the concept of tissue damage as a consequence of pressure comes from a theoretical analysis of the effect of a tourniquet upon tissues by Griffiths and Heywood *(25)*. They showed that the application of any form of tourniquet around a limb tends to produce potentially harmful shear stresses within the soft tissues of that limb.

Current opinion thus seems to favour the view that nerve injury following the use of a tourniquet is primarily the result of stresses induced in the tissues by the pressure of the tourniquet. This view does not, of course, preclude the possibility of ischaemia and the metabolic changes it induces, being contributory factors to the pathogenesis of tourniquet-induced nerve injury *(22, 65)*, although Lundborg *(66)* demonstrated in rabbits that nerves were generally resistant to ischaemia, as a result of their efficient microcirculation.

Nerve injuries have an excellent prognosis, and normally recover over a period varying from a few days *(14)* to several months *(12, 14, 60, 62)*, although considerable disability may be present during the recovery period.

It is interesting that even a low-pressure venous tourniquet may produce nerve damage, if left on for too long. Casscells, Resnick and others describe a case of meralgia paresthetica following the application of a tourniquet to the patient's thigh, as treatment for acute pulmonary oedema *(67)*.

MUSCLE INJURY

The harmful effect of ischaemia upon muscle distal to the tourniquet has been discussed earlier by reference to the adverse changes observed histologically, metabolically and on EMG examination. The extent of damage was in all cases related to the duration of ischaemia, emphasising the necessity to keep this to a minimum.

Injury to muscle lying directly beneath the tourniquet, as distinct from that peripheral to it, has also been investigated. A study on rhesus monkeys, by Patterson and Klenerman *(41)* described microscopic injury to muscle lying directly beneath the tourniquet which was more severe than that seen in muscle subjected to ischaemia only, distal to the tourniquet. The degree of injury again appeared to be related to the duration of application of the tourniquet—a 5 hour period of ischaemia invariably produced severe muscle damage, whereas a 3 hour period caused similar changes in only a quarter of the monkeys examined. The effect of different tourniquet pressures upon the degree and speed of onset of the muscle changes was not investigated. These results are derived from animal experimental work, but there is no reason to suppose that qualitatively similar effects would not obtain in man.

POSTOPERATIVE OEDEMA

Sanders *(13)* discussed the peripheral oedema that may sometimes develop following the use of a tourniquet. He thought that it was probably the result of ischaemic damage to the tissues, and related to the duration of application of the tourniquet. Bruner *(9)* took essentially the same view and noted that in his experience female patients 'of menopausal age' were especially prone to this complication. Ward *(68)* described a series of patients requiring surgery for Dupuytren's disease(sic), some of whom underwent operation under tourniquet and others with their arm supported on an elevated hand table only. He showed that those patients to whom a tourniquet had been applied had more marked and more persistent swelling of their hand postoperatively than those in whom simple elevation only had been employed.

Nakahara and Sakahashi *(54)* demonstrated in the laboratory that increased capillary permeability in dog limbs followed the application of a tourniquet to the limb for 4 hours. They attributed this to release of a factor (probably a kinin) which altered capillary permeability. Support for this concept comes from the work of Bukov *et al. (69)* who claimed that disturbances in the acetylcholine-cholinesterase system, serotonins, kinins, and proteolytic enzymes all played an important role in the development of oedema. Miller *et al. (70)* demonstrated increased interstitial pressure in primate limbs for up to 24 hours following a period of tourniquet ischaemia. They considered that this was due to oedema, secondary to tissue damage caused by the tourniquet itself and/or ischaemia.

The tissue swelling and oedema which may follow use of a tourniquet is thus probably multifactorial in origin, although the precise mechanisms have not yet been elucidated. Whatever the causes, however, postoperative oedema is an undesirable complication, particularly in the hand, where it may seriously impair mobility and compromise function. Sanders *(13)* recommends the use of firm dressings, and elevation of the hand for several days to minimise this problem; in addition, the ischaemic period should be kept to a minimum—as always—and early mobilisation instituted.

VENOUS THROMBOSIS

Love *(5)* stated that 'many reports . . . over the years' have claimed an increased risk of venous thrombosis to be a consequence of using a tourniquet, although he cited no authority to support this comment; however, the impairment of the clotting mechanisms discussed earlier might suggest that the use of a tourniquet is more likely to diminish, rather than enhance, the possibility of thrombus formation. This concept derives some support from a paper by Klenerman *et al. (71)* They demonstrated that, following deflation of a completely occlusive tourniquet used during orthopaedic operations, the fibrinolytic system in the systemic circulation was briefly stimulated. They suggested, therefore, that application of a tourniquet might be of use as a simple form of prophylaxis against deep venous thrombosis in patients undergoing operations such as total hip replacement, in which post-operative thrombosis is a serious complication but they did not specify precisely how the technique might be applied in clinical practice. The increased fibrinolysis was attributed to the effect of anoxia on the vasa vasorum, which stimulated them to release plasminogen activator.

Klenerman's suggestions were subsequently criticised by Risberg *(72)* on the basis of the latter's experimental work, in both animals and man. Risberg demonstrated that prolonged ischaemia (more than 2 hours) of the cheek pouches of hamsters produced a reduction in fibrinolytic activity, which persisted for some time after the ischaemic area had been reperfused. His studies in man were reported fully in a paper published, with Larsson, a few months later *(57)*, in which they described a study of fibrinolytic activity in biopsies of skin and superficial veins taken from a series of patients before, during and after operations on the knee performed under tourniquet. Fibrinolysis in the skin biopsies was found to be significantly increased during the operation but had returned to normal within 15 minutes of deflation of the tourniquet, irrespective of the duration of its application. Forty-eight hours later, fibrinolytic activity in the same

samples showed a marked reduction, but had again returned to normal by the fourth postoperative day. The activity in the vein biopsies showed a significant increase only at the start of the operations—thereafter, activity was little different from control values. Risberg's and Larsson's findings, although interesting, do not refute Klenerman's work, however, as the studies are not comparable; furthermore, their work throws little light on the relationship between the use of tourniquets, and thrombus formation. This is, firstly, because fibrinolytic activity in hamster cheek pouches does not necessarily reflect activity in the human venous system, and secondly, because the changes in activity were observed in skin and superficial vein biopsies; these may not parallel occurrences in the deep veins of the leg, in which thrombosis generally occurs.

Simple clinical investigation of the problem was carried out by Krose and Stiris *(73, 74)*, who described two separate studies in patients undergoing surgery of the lower limb under tourniquet. They performed venography within 48 hours of operation on all patients, of which 10% in the first series and 17% in the second revealed signs of acute thrombosis, one patient in each series suffering a pulmonary embolus. The authors commented that these figures were surprisingly low compared with other reports of post-operative thrombosis *(75, 76)*, and concluded that use of a tourniquet did not seem to increase the incidence of thrombosis. This conclusion is perhaps supported by Klenerman's study, but tends to be contradicted by that of Risberg and Larsson. Nonetheless, Kroese and Stiris *(73, 74)* advised against using a tourniquet in patients who were at particular risk of deep venous thrombosis, such as those who had suffered trauma to the lower limb (and especially if they had been immobilized preoperatively), and advised caution if the patient had a history of venous thrombosis. Their advice is in some degree supported by Austin *(77)*, who described fatal pulmonary embolism occurring in two patients during exsanguination of the leg prior to tourniquet application—the Esmarch bandage apparently dislodged clot from the deep veins of the leg; both patients had suffered traumatic fractures of the leg, and both had been immobilised in bed for several days before operation. Although the tourniquet itself was not the cause of thrombosis or embolism in these patients, the preparations prior to its use were responsible, and this serves to emphasise that care in assessing patients before employing a tourniquet is important.

It is therefore difficult to come to any definitive conclusions regarding the risk of venous thrombosis engendered by using a tourniquet. That the latter is a necessary adjunct to certain operations is inescapable, and the possibility of a deep venous thrombosis may become a relatively minor consideration when set against the need to provide the optimum conditions for essential surgery. Nonetheless, it is not possible categorically to deny that exsanguination and use of a tourniquet increases the dange of thrombus formation and it would seem prudent to follow the advice of Kroese and Stiris *(73–74)* to avoid using a tourniquet in patients at particular risk.

BLEEDING DESPITE PROPER APPLICATION OF THE TOURNIQUET

An occasional problem reported while a tourniquet is in use is that of blood oozing into the operative field, in spite of a properly applied and inflated tourniquet *(6, 78)*. This oozing is apparently due to blood bypassing the tourniquet through the medulla of the humerus or femur, and typically starts about 30 minutes into the operation. The blood enters the medulla above the tourniquet, and leaves below it, via nutrient vessels. Since there can not be any such 'medullary bypass' across the knee or elbow joints, blood can only pass through vessels in the soft tissues of these sites. Furlow *(78)* reported that such oozing may be effectively prevented by the use of a firm bandage around the knee or elbow.

MISCELLANEOUS COMPLICATIONS

A number of miscellaneous complications ascribed to the use of a tourniquet have been reported in the literature. These include burns from a hot tourniquet *(33)*, burns due to a quantity of alcohol-based skin preparation fluid trickling beneath the tourniquet *(11)*, various forms of injury to blood vessels, both direct *(33)*, and indirect *(31)*, cardiac arrest following the application of bilateral tourniquets (mentioned earlier) *(32)* rupture of biceps tendon *(79)*, and minor skin damage due to careless application of the tourniquet *(1)* including temporary hypopigmentation of a small area of skin below the tourniquet *(80)*.

The list of complications associated with the use of a tourniquet may appear formidable, yet it should be appreciated that in reality, their incidence is remarkably low relative to the frequency with which a tourniquet is used. Furthermore, the majority are easily avoidable by care in the application of the tourniquet, together with an awareness of its potential dangers and their prevention.

CONTRAINDICATIONS TO THE USE OF A TOURNIQUET

Chronically hypoxic tissues tolerate a period of virtually total anoxia very poorly and evidence of ischaemia due to peripheral arterial disease *(6)* is a contraindication.

The presence of calcified vessels (in the lower limb) is another contraindication because such vessels may prove incompressible. The use of a tourniquet can only produce venous occlusion, with increased bleeding as a result. Furthermore, a high pressure tourniquet could, in theory, fracture the vessel wall, and thereby produce an acute occlusion of a previously patent vessel *(81, 82)*.

If the limb has suffered a severe crush injury the circulation is often precarious, and exsanguination or application of a tourniquet may result in necrosis of potentially viable tissue.

In certain other circumstances, exsanguination and the application of a tourniquet may not be strictly contraindicated, but its use should be carefully considered. The application of an arterial tourniquet in the presence of sickle-cell disease produces circulatory stasis, gross hypoxaemia and acidosis distal to the site of application. These are ideal conditions to induce sickling of the red cells, with the risks of thrombosis, infarction and haemolysis, and would seem to suggest that the tourniquet is strictly contraindicated in patients carrying the sickle-cell gene. It has been reported, however, that, provided the limb is carefully exsanguinated prior to inflation of the tourniquet *(83, 84)*, the patient is at no particular risk. The general management appropriate for a patient suffering from sickle-cell disease should of course be followed.

Exsanguination with an Esmarch bandage should be avoided altogether or performed only from above the site of infection *(4)*, in the presence of severe (local) infection to avoid dissemination of infective material.

If proven or suspected deep venous thrombis is present.

OTHER USES OF A PNEUMATIC TOURNIQUET IN MEDICINE

This review has so far considered aspects only of the principal use of the arterial tourniquet; that of rendering the upper or lower limb ischaemic to facilitate surgery. There are, however, many other uses for pneumatic arterial tourniquets in medicine, including; as a cranial tourniquet to minimise bleeding during operations on the scalp *(85)*; in intravenous regional anaesthesia (Bier's block) *(86)*; to allow regional neuromuscular block for limb operations permitting the use of light general anaesthesia, and minimising the dose of relaxant *(87)*; in the submaximal effort test, used in the evaluation of analgesics *(88)*; to minimise or prevent the spread of cytotoxic agents given intravenously beyond the area requiring treatment *(89, 92)*; in the production of

intravenous regional sympathetic block for causalgia *(93)*; in producing hypoxia to enhance the effect of radiotherapy in the treatment of limb sarcoma *(94, 95)*; as atraumatic 'clamps' for vessels during vascular surgery *(96, 97)*; to release a fibrous capsular contracture around a synthetic breast implant (i.e. to produce a closed capsulotomy) *(98)*; in the isolated forearm technique, which permits the detection of awareness during general anaesthesia in which minimal anaesthetic agent has been given (e.g. for Caesarean section) *(99)*; and in the measurement of blood pressure by the Riva-Rocci method.

OTHER ASPECTS OF TOURNIQUET USE

THE ESMARCH BANDAGE AS A TOURNIQUET

The use of the Esmarch bandage is associated with greater risk of nerve injury *(33)* but Griffiths and Hamilton *(100)* pointed out that it has some advantages over a pneumatic tourniquet in that it is simple to apply, it never fails, it takes up little space and stays in position even on flabby or conical limbs. Excessive presure is the main cause of nerve damage and it is obviously relevant to examine the pressure exerted by an Esmarch bandage used in this way; this has been done both by Griffiths and Hamilton *(100)* (on the arm) and by Middleton and Varian *(33)* (on the thigh). The latter noted that the pressure exerted by such a tourniquet was variable but 'certainly greater than is generally appreciated'. Griffiths and Hamilton calculated that, as a guide, four turns of an Esmarch bandage applied under moderate tension to an 'average' arm ((38 cm—15 inches) in circumference) gave an approximate pressure of 250–300 mmHg, which is comparable to that generally used with a pneumatic tourniquet.

Neither of these two groups specifically recommended using the Esmarch bandage instead of an inflatable tourniquet but neither condemned it; Griffiths and Hamilton *(100)* commented that 'on occasion, its use may be unavoidable', although they were not specific. A possible advantage is that an elastic tourniquet can be applied higher on the thigh than the pneumatic tourniquet, which may be of value in certain circumstances *(19)*; however, with the number of efficient pneumatic tourniquets now available, it seems inadvisable to expose the patient to the risks of an elastic tourniquet without compelling reason *(18)*. If the use of an Esmarch in this way be deemed necessary, a description of the mode of application may be found both in *Campbell's operative orthopaedics (19)* and in *Standard orthopaedic operations: a guide (18)*.

IMPROVEMENTS TO THE BASIC PNEUMATIC TOURNIQUET

Numerous improvements to the basic pattern of pneumatic tourniquet have been described or advocated. Love *(5)* suggested a pneumatic tourniquet made of a series of parallel tubes with the inflation pressure diminishing towards the edge of the tourniquet in order to reduce the shear stress imposed on the tissues which is believed to be the main cause of nerve injury. Devices to maintain tourniquet pressure at a fixed value, so avoiding loss of haemostasis during operation, have been described by Houghton *(101)*, Jones and Gregory *(121)* and Klenerman *(28)*; tourniquet apparatuses such as those by Kidde, Schuco, Zimmer and Stille also provide this facility. Wheeler and Lipscomb *(103)* inserted a simple pressure relief valve between the gauge and arm-band of the tourniquet to avoid excessive inflation pressures; Faulconer, Lawrence and Seldon described a similar device *(104)*.

THE TOURNIQUET AS A FIRST AID DEVICE

It is probably fair to say that tourniquets are associated in the popular mind with prevention either of life-threatening haemorrhage, or of the systemic spread of venom following snake bite.

Tourniquets are, however, of only limited value in the latter circumstance *(105)* and their use in providing emergency haemostasis is condemned as generally producing more harm than good *(2, 4)*. The scope for damage during application of a tourniquet by an untrained person under the stress of an emergency should be apparent from this review. The current British Red Cross manual recommends direct pressure over a dressing to a severely bleeding wound with elevation of the injured part if possible. It is only if bleeding cannot be controlled by such means that direct pressure on the major artery supplying the area (femoral or brachial) is advocated, and then for no more that 15 minutes *(106)*; nowhere in the manual is the use of a tourniquet recommended.

LEGAL CONSIDERATIONS IN THE USE OF THE TOURNIQUET

It is worth remembering that the risks to the patient inherent in using a tourniquet should not be dismissed too lightly. Even in 1931, Eckhoff *(14)* noted that 'recovery from a nerve injury may be delayed by the problem of litigation', and Wachsman *(107)* describes the upholding, on appeal, of damages awarded against a New York Hospital in respect of a patient whose left arm was paralysed due to excessive tourniquet pressure.

Enquiries to the Medical Defence Union revealed that 10 cases were notified involving a tourniquet (type not specified) during the years 1979 and 1980. Injuries associated with its use ranged from ulceration and muscle-wasting to total paralysis of a limb.

The Medical Protection Society was unable to supply details of tourniquet injuries reported to it, although recent Annual Reports describe a sciatic nerve palsy following the use of an Esmarch bandage, apparently employed both to exsanguinate the limb and to serve as a tourniquet *(108)*.

DIGITAL TOURNIQUETS

Minor operations on fingers or toes are facilitated by placing a tourniquet around the base of the digit, so producing a bloodless field. The traditional method of applying such a tourniquet is to wind a rubber tube around the base of the finger or toe, and to secure its free ends between the jaws of a pair of artery forceps *(109, 110)*. An alternative method, using the finger of a disposable rubber glove, has also been described *(109, 111)*. Small plastic or rubber rings to serve the same purpose were available commercially *(112)* but they have been criticised as they may easily be overlooked at the end of the operation *(110)*, and both Defence Societies have been involved in litigation arising from such oversights. For this reason, the traditional style of digital tourniquet may be preferable.

Conclusion

The tourniquet is an invaluable aid to modern limb surgery, but its use is attended by risks of which the intending user should be aware. These risks are least with the pneumatic tourniquet, and it seems difficult to justify the continued use of a simple rubber tourniquet. However, inappropriate or careless use of any type of tourniquet may expose the patient to serious injury.

References

1 McElvenny RT. The tourniquet—its clinical application. Am J Surg 1945;69:94–106.
2 Klenerman L. The tourniquet in surgery. J Bone Joint Surg 1962;44B:937–43.
3 Cushing H. Pneumatic tourniquets: with special reference to their use in craniotomies. The Medical News 1904;84:577–80.
4 Tubiana R. L'usage du garrot au niveau des membres-ses dangers et leur prevention. Rev Chir Orthop 1973;59:239–45.
5 Love BRT. The tourniquet. Aust N Z J Surg 1978;48:66–70.
6 'The tourniquet'. (Editorial.) Lancet 1958;1:1161–2.
7 Brantley P. Pneumatic tourniquets in the OR. The O.N.A. Journal 1977;4:172–3.
8 Dorsch D. The pneumatic tourniquet in the OR. The O.N.A. Journal 1976;3:127–8.
9 Bruner JM. Time, pressure and temperature factors in the safe use of the tourniquet. Hand 1970;2:39–42.

10 Fry D. Inaccurate tourniquet gauges. Br Med J 1972;1:511.
11 Flatt AE. Tourniquet time in hand surgery. Arch Surg 1972;104:190–2.
12 Calderwood JW, Dickie WR. Tourniquet paresis complicating tendon grafting. Hand 1972;4:53–5.
13 Sanders R. The tourniquet. Instrument or weapon? Hand 1973;5(2):119–23.
14 Eckhoff NL. Tourniquet paralysis. Lancet 1931;2:343–5.
15 'Tourniquet techniques'. (Editorial.) Br Med J 1972;2:247–8.
16 Thomassen EH. An improved method for application of the pneumatic tourniquet on extremities. Clin Orthop 1974;103:99–100.
17 Brigden RJ. Orthopaedic operations. In: Brigden RJ, ed. Operating theatre technique. Edinburgh: Churchill-Livingstone, 1980;511–13.
18 Adams JC. Introduction. In: Adams JC, ed. Standard orthopaedic operations. Edinburgh: Churchill-Livingstone, 1976;4–6.
19 Edmonson AS. Surgical Techniques. In: Edmonson AS, Crenshaw AH, eds, Campbell's Operative Orthopaedics. St Louis. The CV Mosby Company, 1980; 1–31.
20 Stewart JDM. Tourniquets. In: Stewart JDM, ed. Traction and orthopaedic appliances. Edinburgh: Churchill-Livingstone, 1975:181–9.
21 Hinman F. The rational use of tourniquets. International Abstracts of Surgery 1945;81:357–66.
22 Rorabeck CH. Tourniquet-induced nerve ischaemia: an experimental investigation. Trauma 1980;20:280–6.
23 Spiegel IJ, Lewin P. Tourniquet paralysis. J A M A 1945;129:432–5.
24 Campbell WC, Boyd HB. A pneumatic tourniquet. J Bone Joint Surg 1937;19:832–3.
25 Griffiths JC, Heywood OB. Biomechanical aspects of the tourniquet. Hand 1973;5:113–18.
26 Denny-Brown D, Brenner C. Paralysis of nerve, induced by direct pressure and by tourniquet. Arch Neurol 1944;51:1–26.
27 Sinclair DC. Observations on sensory paralysis produced by compression of a human limb. J Neurophysiol 1948;11:75–92.
28 Klenerman L. A modified tourniquet: preliminary communication. J R Soc Med 1978;71:121–2.
29 Burchell G, Stack G. Exsanguination of the arm and hand. Hand 1973;5:124–6.
30 Fisher RH. Bloodless field for tourniquet? Clin Orthops 1974;103:96.
31 Thio RT. False aneurysm of the ulnar artery after surgery employing a tourniquet. Am J Surg 1972;123:604–5.
32 Bradford EMW. Haemodynamic changes associated with the application of lower limb tourniquets. Anaesthesia 1969;24:190–8.
33 Middleton RWD, Varian JP. Tourniquet paralysis. Aust. N Z J Surg 1974;44:124–8.
34 Weingarden SI, Louis DL, Waylonis GW. Electromyographic changes in postmeniscectomy patients. J A M A 1979;241:1248–50.
35 Saunders KC, Louis DL, Weingarden SI, Waylonis GW. Effect of tourniquet time on postoperative quadriceps function. Clin Orthop 1979;143:194–9.
36 Shaw Wilgis EF. Observations on the effects of tourniquet ischaemia. J Bone Joint Surg 1971;53:1343–6.
37 Nolan B, McQuillan WM. Acute traumatic limb ischaemia. Br J Surg 1965;52:559–65.
38 Dahlback L. Effects of temporary tourniquet ischaemia on striated muscle fibers and motor end-plates. Scand J Plast Reconstr Surg (Suppl) 1970;7:1–91.
39 Santavirta S, Luoma A. Arstila AU. Morphological and biochemical changes in striated muscle after experimental tourniquet ischaemia. Res Exp Med (Berl) 1979;174:245–51.
40 Tountas CP, Bergman RA. Tourniquet ischaemia: ultrastructural and histochemical observations of ischemic human muscle and of monkey muscle and nerve. J Hand Surg 1977;2:31–7.
41 Patterson S, Klenerman L. The effect of pneumatic tourniquets on the ultrastructure of skeletal muscle. J Bone Joint Surg 1979;61B:178–83.
42 Solonen KA, Hjelt L. Morphological changes in striated muscle during ischaemia. Acta Orthop Scand 1968;39:13–19.
43 Enger EA, Jennische E, Medegard A, Haljamae H. Cellular restitution after 3 h of complete tourniquet ischemia. Eur Surg Res 1978;10:230–9.
44 Karpf M, Stock W, Gebert E, Kruse-Jarres JD, Zimmerman W. Stoffwechselveranderungen und Restitution nach temporarer Tourniquet-Ischemie beim Menschen. Langenbecks Arch Chir Suppl 1974; Chir Forum Exp Klin Forsch 307–11.
45 Santavirta S, Kauste A, Rindell K. Tourniquet ischaemia. Clinical and biochemical observations. Ann Chir Gynaecol 1978;67:210–13.
46 Haljamae H, Enger E. Human skeletal muscle energy metabolism during and after complete tourniquet ischemia. Ann Surg 1975;182:9–14.
47 Santavirta S, Hockerstedt K, Niinikoski J. Effect of pneumatic tourniquet on muscle oxygen tension. Acta Orthop Scand 1978;49:415–19.
48 Enger ER. Cellular metabolic response to regional hypotension and complete ischemia in surgery. Acta Chir Scand [Suppl 481] 1978;144:1–20.
49 Shehadi S, Paletta FX, Cooper T. Effect of hypothermia on circulatory responses in the canine hind limb after tourniquet ischaemia. Surg Forum 1961;12:466–8.
50 Paletta FX, Willman V, Ship AG. Prolonged tourniquet ischemia of extremities. J Bone Joint Surg 1960;42A:945–50.
51 Klenerman L, Crawley J. Limb blood flow in the presence of a tourniquet. Acta Orthop Scand 1977;48:291–5.
52 Larsson J, Bergstrom J. Electrolyte changes in muscle tissue and plasma in tourniquet-ischemia. Acta Chir Scand 1978;144:67–73.
53 Larsson J, Lewis DH. The local haemodynamic effects of operation in a bloodless field. Eur Surg Res 1978; 10:24–32.
54 Nakahara M, Sakahashi H. Effect of application of a tourniquet on bleeding factors in dogs. J Bone Joint Surg 1967;49:1345–51.
55 Rutherford RB, West RL, Hardaway RM. Coagulation changes during experimental hemorrhagic shock. Ann Surg 1966;164:203–14.
56 Miller SH, Eyster ME, Saleem A, Gottlieb L, Buck D, Graham WP III. Intravascular coagulation and fibrinolysis within primate extremities during tourniquet ischemia. Ann Surg 1979;190:227–30.
57 Larsson J, Risberg B. Fibrinolytic activity in human legs in tourniquet ischemia. Thromb Res 1977;11:817–25.
58 Modig J, Kolstad K. Wigren A. Systemic reactions to tourniquet ischaemia. Acta Anaesthesiol Scand 1978;22:609–14.
59 Hassan H, Gjessing J, Tomlin PJ. Blood gas changes following tourniquet release during spinal anaesthesia. Br J Anaesth 1978;50:76.
60 Moldaver J. Tourniquet paralysis syndrome. AMA Arch Surg 1954;68:136–44.
61 Rudge P. Tourniquet paralysis with prolonged conduction block. An electrophysiological study. J Bone Joint Surg 1974;56B:716–20.
62 Bolton CF, McFarlane RM. Pneumatic tourniquet paralysis. Neurology 1978;28:787–93.
63 Ochoa J, Danta G, Fowler TJ, Gilliatt RW. Nature of the nerve lesion caused by a pneumatic tourniquet. J Neurol Neurosurg Psychiatry 1972;35:638–47.
64 Fowler TJ, Danta G, Gilliatt RW. Recovery of nerve conduction after a penumatic tourniquet: observations on the hind-limb of the baboon. J Neurol Neurosurg Psychiatry 1972;35:638–47.
65 Dery R, Pelletier J, Jacques A. Clavet M, Houde JJ. Metabolic changes induced in the limb during tourniquet ischaemia. Can Anaesth Soc J 1965;12:367–78.
66 Lundborg G. Ischemic nerve injury. Scand J Plast Reconstr Surg (Suppl 1) 1970;4:1–113.
67 Casscells W, Resnick N, Thalinger K, Herzog A. Meralgia parasthetica due to unusual compression. N Engl J Med 1978;298:285.
68 Ward CM. Oedema of the hand after fasciectomy with or without tourniquet. Hand 1976;8:179–85.
69 Bukov VA, Myslovaty BS, Korolev VV, Sekamova SM, Levandovsky IV. On the mechanisms of origination of oedema in the durably ischemized extremity, its prophylaxis and treatment. (Translated title.) Vestn Akad Med Nauk SSSR. 1975;7:35–9.
70 Miller SH, Price G, Buck D *et al.* Effects of tourniquet ischemia and postischemic edema on muscle metabolism. J Hand Surg 1979;4:547–55.
71 Klenerman L, Chakrabarti R, Mackie I, Brozovic M, Stirling Y. Changes in haemostatic system after application of a tourniquet. Lancet 1977;1:970–2.
72 Risberg B. Fibrinolysis and tourniquet. (Letter.) Lancet 1977;2:360–1.

73 Kroese AJ, Stiris G. Does peroperative pneumatic tourniquet cause thrombosis? Journal of Oslo City Hospital 1974;24:69–73.

74 Kroese AJ, Stiris G. The risk of deep-vein thrombosis after operations on a bloodless lower limb. A venographic study. Injury 1975;7:271–3.

75 Becker J, Schampi B. The incidence of postoperative venous thrombosis of the legs. Acta Chir Scand 1973;139:357–67.

76 Kakkar VV, Flanc C, Howe CT, Clarke MB. Natural history of postoperative deep-vein thrombosis. Lancet 1969;2:230–2.

77 Austin M. The Esmarch bandage and pulmonary embolism. J Bone Joint Surg 1963;45B:384–5.

78 Furlow LT. Cause and prevention of tourniquet ooze. Surg Gynaecol Obstet 1971;132:1069–72.

79 Logel RJ. Rupture of the long tendon of the bicepts brachii muscle. An unusual case related to use of the pneumatic tourniquet. Clin Orthop 1976;121:217–21.

80 Testa NN, Waingankar S, Auerbach R. Hypopigmentation at the site of application of a tourniquet. J Dermatol Surg Oncol 1978;4:473–4.

81 Klenerman L, Lewis JD. Incompressible vessels. Lancet 1976;1:811–12.

82 Jeyaseelan S, Stevenson TM, Pfitzner J. Tourniquet failure and arterial calcification. Anaesthesia 1981;36:48–50.

83 Stein RE, Urbaniak JR. Sickle cell disease and the tourniquet. J Bone Joint Surg 1975;57A:1027.

84 Gilbertson AA. The management of anaesthesia in sickle cell states. Proc R Soc Med 1967;60:631–5.

85 Orticochea M. A pneumatic cranial tourniquet to control haemorrhage during operations on the scalp. Br J Plast Surg 1977;30:223–6.

86 Holmes CMcK. Intravenous regional analgesia. Lancet 1963;1:245–7.

87 Torda TA, Klonymus DH. Regional neuromuscular block. Acta Anaesthesiol Scand 1966;24:177–82.

88 Smith GM, Egbert LD, Markowitz RA, Mosteller F, Beecher HK. An experimental pain method sensitive to morphine in man: the submaximum effort tourniquet technique. Pharmacol Exp Ther 1966; 154:324–32.

89 Lathrop JC, Frates RE. Arterial infusion of nitrogen mustard in the treatment of intractable pelvic pain of malignant origin. Cancer 1980;45:432–8.

90 Karakousis CP, Rao U, Holtermann OA, Kanter PM, Holyoke ED. Tourniquet infusion chemotherapy in extremities with malignant lesions. Surg Gynaecol Obstet 1979;149:481–90.

91 Simister JM. Alopecia and cytotoxic drugs. Br Med J 1966; 2:1138.

92 Hennessy JD. Alopecia and cytotoxic drugs. Br Med J 1966; 2:1138.

93 Hannington-Kiff JG. Intravenous regional sympathetic block with guanethidine. Lancet 1974;1:1019–20.

94 Tubiana M. New methods of treatment in radiation therapy. Clin Radiol 1964;15:142–51.

95 Van den Brenk HAS. The tourniquet technique in radiotherapy of limb tumours. Nuntius Radiologicus 1968;34:207–14.

96 Bernhard VM, Boren CH, Towne JB. Pneumatic tourniquet as a substitute for vascular clamps in distal bypass surgery. Surgery 1980;87:709–13.

97 Dunn DC. The use of the arterial sling tourniquet in surgical practice. Br J Surg 1973;60:594–6.

98 Frank DH, Robson MC. A pneumatic tourniquet as an aid to release of capsular contracture around a breast implant. Plast Reconstr Surg 1978;61:612–13.

99 Tunstall ME. Detecting wakefulness during general anaesthesia for caesarian section. Br Med J 1977;1:1321.

100 Griffiths JC, Hamilton PH. The Esmarch bandage as a tourniquet. J R Coll Surg Edinb 1970;15:114–17.

101 Houghton IT. A self-compensating tourniquet. Lancet 1972;2:800–2.

102 Jones CS, Gregory AF. A self-compensating tourniquet. Lancet 1958;2:400–1.

103 Wheeler DK, Lipscomb PR. A safety device for a pneumatic tourniquet. J Bone Joint Surg 1964;46A:870.

104 Faulconer A, Lawrence VS, Seldon TH. A safety valve for the pneumatic tourniquet. Anesth Analg (Cleve) 1970;49:575–6.

105 In 'Poisoning by animals' by George CF. Medicine magazine, 2nd series, 1975 et seq, 228–9.

106 In 'First aid', the manual of the British Red Cross Society, 1972, third edition, p. 90.

107 Wachsman HF. Jury decision on injury upheld. Legal aspects of Medical Practice 1978;9:64–5.

108 Medical Protection Society Annual Report for 1972, pp 30–1 and p 43.

109 Salem MZA. Simple finger tourniquet. Br Med J 1973;2:779.

110 Hoare EM. Simple finger tourniquet. Br Med J 1973;3:293.

111 Bouchier-Hayes DJ. Simple finger tourniquet. Br Med J 1973;3:293.

112 Smellie GD. Exsanguinating finger tourniquet. Lancet 1962;2:78.

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