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Special Aspects of Forearm Compartment Syndrome in Children

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1. Introduction

Trauma to the wrist and forearm in children resulting in either distal or midshaft fractures are quite common in pediatric surgery practice. The treatment of such fractures is well established and healing in the majority of patients reported as uneventful. However, if there is an extensive muscle hematoma, or extensive swelling due to mangled soft tissue injury, or a heavy bleeding, the situation can suddenly change and run into a disastereous Compartment Syndrome putting muscles, nerves and vessels at risk for destruction. In the following paragraphs our patients collective is presented as an illustrative exemplary and discussed in front of a literature and textbook review.

2. Exemplary patients collective

After suffering an accident with a distal or mid-shaft forearm (Salter Harris I/II, comminute, shaft) fracture, a persistent hemorrhage from the fracture side or a lacerated vessel, eight children (6 male, 2 female) with a Mean age of 12 (range 8 to 15 ½) years developed a forearm Compartment Syndrome (CS) within 1 hour to 1 week requiring fasciotomy. In 3|8 patients an additional mangled upper limb trauma has been notified, in one patient as part of a polytrauma.

All patients displayed clinical signs of elevated compartment pressure in due course. Namely, by elevated compartment pressure measurements in 2|8, immediate impaired neurological function in 1|8, while another 3|8 presented late with signs of Volkmann`s Contracture (VC), Reflex Sympathetic Dystrophy Syndrome (RSDS), or expression of Vanishing Bone (VB) disease.

Primary non-surgical treatment with closed reduction and plaster cast (POP) splinting for fracture treatment respectively splinting alone for sole soft tissue trauma took place in half (4|8) of our patients.

At the time of intervention, open fasciotomy has been performed in each patient according to common incision lines and structures involved. During surgery, the following types of Compartment Syndromes have to be encountered in our exemplary sample:

- 2|8 isolated pronator quadratus hematomas
    (deep, distal anterior-flexor compartment)
- 2|8 distal/middle anterior-flexor compartments (Fig.1)
- 3|8 anterior-flexor and dorsal-extensor compartments
    (exclusively in very late presenters)
- 1|8 isolated proximal dorsal-extensor compartment

Fig. 1. Classical case; clinical picture: massive swelling, sensivity loss of fingers, in much pain. a) At the time of accident. b) During fasciotomy. c) Temporary closure of the fasciotomy by a foam dressing and a dynamic suture. d) Nicely healing scar after skin closure.

Half of our patients showed a late onset of their CS symptoms after an initial so-called uneventful closed reduction and POP splinting. During fasciotomy, in all fracture patients ORIFs (3 x K-wires, 1 x external fixateur (Fig.2), 1 x ESIN, 2 x combinations) have to be performed to stabilize the bone. The non-fracture patient could be managed by sole splinting entirely.
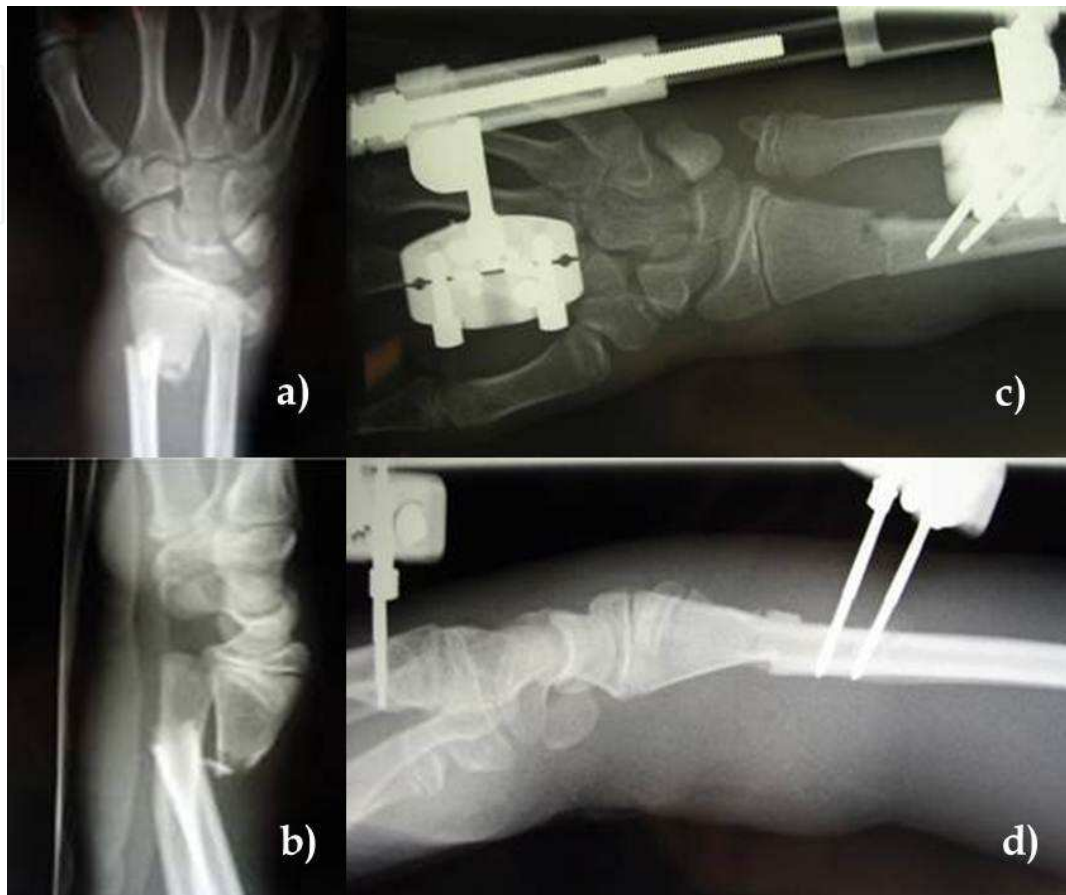
Fig. 2. Classical case; x-rays: a) dislocated distal forearm fracture, plane view. b) dislocated distal forearm fracture, lateral view. c) post reduction and ORIF, plane view. d) post reduction and ORIF, lateral view.

For fasciotomy closure, either a customized foam dressing (polytetrafluorethylene, Epigard®, The Clinipad Corporation) with dynamic sutures (in 5|8) or a topical negative pressure (TNP)/vacuum-assisted closure device (V.A.C.®, KCI Medical, 75-125 mmHg, intermittent mode) (Fig.3) have been used (in 2|8) until final tensionless closure by secondary direct suture.

Three-quarter of our patients showed an uneventful fracture healing with on time removal of implants (ROI) or POP splints; except the polytrauma and Vanishing Bone patients. Direct and tensionless closure of the fasciotomy/skin incision site could be achieved finally in all cases with only minimal scarring at the final follow-up visit. In general, application of the V.A.C.® device resulted in a markedly faster decrease of the soft tissue swelling and edema, which resulted in a much faster closure of the incision site, too (Fig.3).
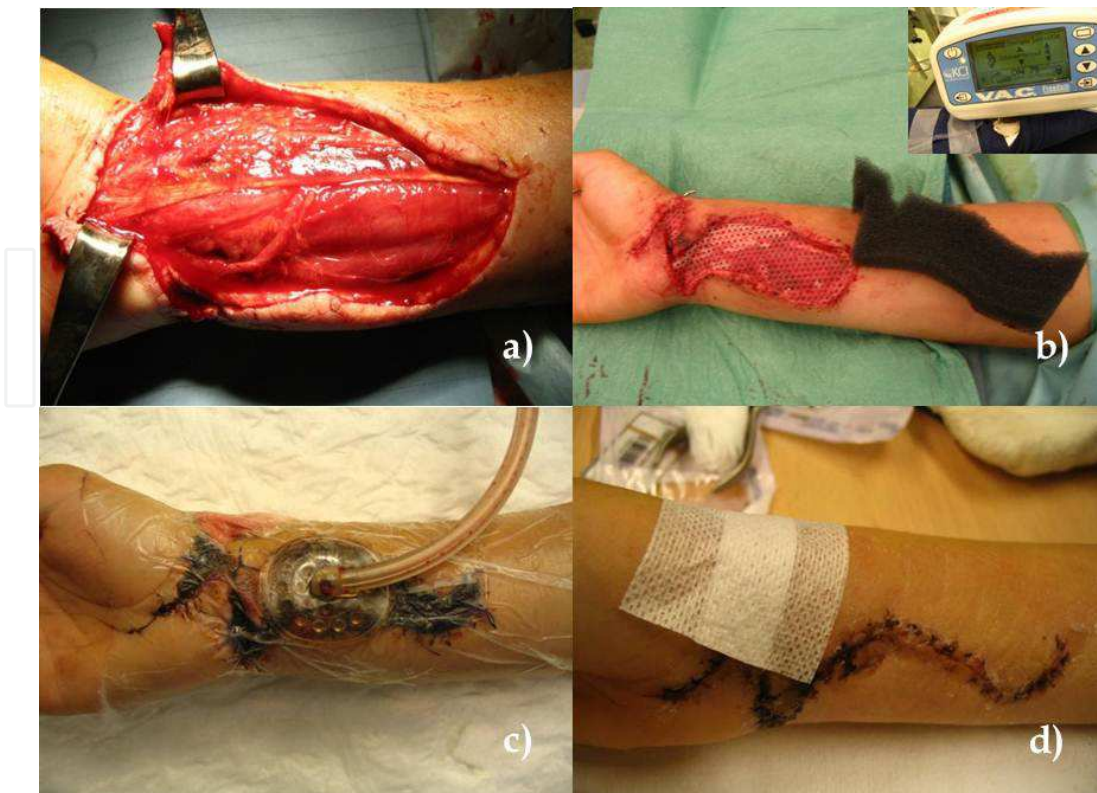
Fig. 3. Special case; clinical picture: massive swelling, increased compartment pressure. a) Immediately after fasciotomy. b) V.A.C.® foam prepared for dynamic suture application. Inlet: V.A.C.® suction unit. c) V.A.C.® dressing in place and in action. d) Nicely healing scar after fast and tensionless skin closure.

In the early presenters (5|8) full ROM, grip and strength could be observed to be back in the majority of patients after 6 to 8 weeks. While in the late presenters (3|8, range 1 to 6 wks after incident) symptoms consistent with RSDS in 1|8, respectively with VC (ie muscle necrosis, contracture) in 2|8 have been expressed. The latter reporting only moderate sequelae after 6 to 9 months of intensive physiotherapy. The latest expressed an additional Vanishing Bone disease and underwent multi-stage secondary reconstruction surgeries to improve his overall forearm function. Our average follow-up comprised 1.5 years.

3. Discussion

The representative findings out of our patient collective, established and progressive management, options and treatment strategies are now discussed in front of a selected literature review.

3.1 Definition of terms: Compartment Syndrome, Volkmann Contracture, Reflex Sympathetic Dystrophy Syndrome and Vanishing Bone disease

A Compartment Syndrome (CS) is defined as the condition in which a raised tissue pressure within the closed skin and soft tissue coat, respectively the tight connective tissue separating muscle groups, leads to a lack of perfusion resulting in neuro-muscular dysfunction and tissue- or organ damage. A Compartment Syndrome has either extrinsic or intrinsic causes, and will be described as acute, subacute, chronic, or recurrent (Bae et al., 2001; Grottkau et

al., 2005; Joseph et al., 2006; Krahn, 2005; Mars & Hadley, 1998; Mubarak & Carroll, 1979; Ouellette, 1998; Paletta & Dehghan, 1994; Prasarn & Ouellette, 2009, 2011; Ragland et al., 2005; Ramos et al., 2006; Sawyer, 2010; Wright, 2009).

Volkmann`s contracture (VC) is defined as the end-result of an ischemic injury to the muscles and nerves of the limb. Volkmann`s ischemia is the acute episode of pain aggravated by passive stretching and neurological deficit resulting from ischemia of muscle and nerve. Volkmann`s ischemia, if untreated, leads to Volkmann`s contracture. It is a disorder of the small vessels, then if the pressure in the tissue rises, the capillaries, venules and arterioles get occluded, while the major arteries nearly always remain patent. During the 1970s, numerous investigators established the pathophysiology of Compartment Syndrome as follows: An injury, that would lead to increased intracompartmental pressure. That, if not relieved, would result in Volkmann`s contracture. Although Hamilton first described ischemic contracture in 1850, Richard von Volkmann is credited with describing it in 1881 in his famous article entitled "Ischemic Muscle Paralysis and Contractures". Hildebrand, in 1906, was the first to use the term "Volkmann`s ischemic contracture". The most frequent historical cause of Compartment Syndrome with subsequent Volkmann`s contracture of the upper extremity in children is the supracondylar fracture of the humerus. Recently reported cases on newborns showing contractures and nerve changes at the time of acute presentation describe it as "Neonatal Volkmann`s syndrome" (Bae et al., 2001; Blakemoore et al., 2000; Grottkau et al., 2005; Krahn, 2005; Krenzien et al., 1998; Mubarak & Carroll, 1979; Ouellette, 1998; Paletta & Dehghan, 1994; Preis, 2000; Ragland et al., 2005; Ramos et al., 2006; Wright, 2009).

Reflex Sympathetic Dystrophy Syndrome (RSDS) is a condition that features a group of typical symptoms, including pain (often of "burning" type), tenderness, and swelling of an extremity associated with varying degrees of sweating, warmth and/or coolness, flushing, discoloration, and shiny skin. Other terms it is referred to are "shoulder-hand syndrome", "causalgia" or "Morbus Sudeck". The exact mechanism of development is not well understood. Theories include irritation and abnormal excitation of nerve tissue, leading to abnormal impulses along nerves affecting blood vessels and skin. Triggers, in no particular order and besides others, can be trauma, surgery, nerve irritation by entrapment or shingles, shoulder problems or even any non-associated event in one-third of patients. According to our best knowledge especially in the pediatric age group reports are very sparse. The onset of symptoms can be rapid, gradual incomplete and mono- or bilateral, passing through the stages: acute, dystrophic and atrophic. With the first stage showing early x-ray changes like patchy bone thinning, and the last stage showing significant osteoporosis already (Bae et al., 2001; Cooney et al., 1980; Shield, 2011).

Characterized by spontaneous or posttraumatic progressive resorption of bone, Vanishing Bone (VB) disease is a rare entity. Numerous names have been used in the literature to describe this condition such as phantom bone, massive osteolysis, disappearing or vanishing bone disease, acute spontaneous absorption of bone, hemangiomatosis and lymphangiomatosis, or Gorham and Stout disease. Since it has been first described in 1838, the etiology still remains speculative, the prognosis is unpredictable, and effective therapy still has not been determined. Incidence may be linked to a history of minor trauma, although as many as half of the patients do not have any history of trauma at all. Most cases occur in children or adults < 40 years. Approximately 60 % of all cases with Vanishing Bone disease are reported to occur in men. It has been reported that > 15 % of patients die as a

result of their disease and that neurological complications increase the mortality up to 33.3 %. The bones of the upper extremity and the maxillofacial region are the predominant osseous locations of the disease. Multicentric involvement is unusual. Vanishing Bone disease is a rare idiopathic disease leading to extensive loss of bone matrix, which is replaced by proliferating thin-walled vascular channels and fibrous tissue. The process often extends to the soft tissues and adjacent bones, especially at the shoulder girdle. The radiographic appearance becomes diagnostic when unilateral partial or total disappearance of contiguous bones, tapering of bony remnants, and absence of a sclerosing or osteoblastic reaction are present. Radiation and chemotherapy, and antiresorptive medications have all been used with different degrees of success. On the one hand bone grafting techniques yield poor results, indicated by a high incidence of bone graft resorption, however, on the other hand successful reconstructions with bone grafts are reported as well. Recently, the diagnosis was found in the late presenting cases by the residual findings of compartment ischemia and skeletal growth changes in newborns with Compartment Syndrome (Ragland et al., 2005; Rubel et al., 2008; Schnall et al., 1993; Papadakis et al., 2011).

3.2 History and incidence of Compartment Syndrome

First descriptions of "Compartment Syndrome" are found at the beginning of the 19th century by Larrey (1812) and Hamilton (1850), before Richard von Volkmann, a surgeon from Halle in Germany first mentioned "traumatic Compartment Syndrome" in 1881. Later in 1911, Bernhard Bardenheuer first considered fasciotomy as a potential therapy, shortly before JB Murphy in 1914 suggested, that fasciotomy should be used to relieve the pressure in edematous extremities. Finally, PN Jepson established fasciotomy in 1926. Finochietto started research on the Compartment Syndrome of the upper limb in 1920, but the current term "Compartment Syndrome" was not established before 1963 by Reszel et al. from the Mayo Clinic. During the 1940s, numerous reports of "acute anterior compartment syndrome of the leg" due to prolonged marching or other rigorous activities to the extremity appeared. Theories regarding venous obstruction (Brooks 1922) were strongly challenged by Griffiths (1940), who considered that arterial injury with reflex spasm to the collateral vessels has to be considered as the primary cause of Volkmann`s ischemia. Recently, with better definition of the compartment anatomy and the development of pressure measurement devices, it has been realized that such a Volkmann`s ischemic contracture has been caused by an untreated Compartment Syndrome, which could even have been existed before without any major arterial injury. During the 1970s, numerous investigators established the pathophysiology of "Compartment Syndrome" as an injury where increased intracompartmental pressure, if not relieved, might result in a Volkmann`s contracture. Seddon, in 1966, described the four separate compartments of the leg, emphasizing the need for fasciotomy of each compartment being under increased intracompartmental pressure again (Choi et al., 2010; Krahn, 2005; Mubarak & Carroll, 1979; Paletta & Dehghan, 1994).

But still in 1998, less than 50 % of hospitals in ie the United Kingdom had dedicated devices for measuring intracompartmental pressure and it is likely that in less affluent countries even fewer centers would have this opportunity available (Joseph et al., 2006). And this even despite the fact that "Compartment Syndrome" is recognized as a real surgical emergency. With an incidence of 1 to 3 per 1 000 fractures in 2002, if following a supracondylar humerus fracture, which is reported as the most classical incident (Battaglia et al, 2002). Respectively, with a Median annual incidence of 7.3/100.000 in men and 0.3/100.000 in women in Germany in

2005, or an incidence after fractures ranging from 3 % to 17 % (Krahn, 2005). Data of a computerized search of the US National Pediatric Trauma Registry published in the same year reported 85 % of cases as a sequelae of fractures, while 13 % resulted from soft tissue injuries alone. Forearm fractures have been the most common cause in the upper extremity, besides tibia and/or fibula fractures if regarding the lower extremity. Open fractures significantly increased the risk of developing a Compartment Syndrome, either in forearm or leg fractures. Sixty percent of patients went directly from the emergency room to the operating room, suggesting that the other 40 % developed their Compartment Syndrome after admission, or suffered a delayed diagnosis. Boys outnumbered girls by a 1.5 : 1 ratio in the 1 to 4 year old group, with the predominance in adolescent boys increasing to 15 : 1 in the 15 to 19 year old age group. Nearly 94 % of the injuries has been reported as unintentional, nearly 4 % as the result of an assault, and nearly 1 % as self inflicted (Grottkau et al, 2005).

3.3 Compartment Syndrome anatomy and pathophysiology

Anatomic compartments may be best described as those spaces, or potential spaces, who are limited at their binderies by osteo-fascial borders (Berger & Weiss, 2001; Bibbo et al, 2000; Ogden, 2000). Starting distal on the upper extremity, at the level of the hand, there are 10 compartments discernible, each with its own individual fascial layer. Namely, four dorsal interosseous compartments, three palmar interosseous compartments, a thenar compartment, a hypothenar compartment, and an adductor compartment (Shin et al, 1996). At the forearm level, besides the tight fascial membranes subgrouping the muscles into compartments, the tight interosseous membrane between radius and ulna have to be considered, too. Finally resulting in the three major forearm compartments: anterior, posterior and radial. With the anterior and posterior compartment being subdivided in a superficial and deep component each. With each compartment first containing its adjacent muscle groups, and second being traversed by its own nerve-vessel-cable. For example, the median, ulnar, radial, volar-interosseous or posterior one (for details see Fig. 4). The median route contains the median nerve, the ulna route ulnar artery and nerve, while the radial one contains radial artery and the superficial ramus of the radial nerve. The volar-interosseus route accommodates the anterior interosseus nerve and artery, while the posterior one accommodates the posterior interosseus nerve (for details see Fig. 4) (Battaglia et al., 2002, Zhixin et al., 2010; EORIF, 2011).

Trauma is it in most of the cases, that is the incipient factor, resulting in direct tissue damage and tissue hemorrhage. The resultant increase in local venous pressure lowers the arterio-venous pressure gradient, leading to decreased local blood flow and tissue oxygenation. The relative anerobic conditions that follow promote the loss of cellular oxidative processes, which in turn cause the loss of cell membrane ionic gradients, causing cell membrane destruction and cell death. Increased capillary permeability results, causing an increase in tissue edema, and further loss of tissue oxygen tension, which ultimately causes cell death (Bibbo et al., 2000; Krahn, 2005). In an expansile space, any tendency of elevation of tissue pressure can be accommodated by an increase in volume of the space. In a closed compartment, be this osseo-fascial or fascial alone, increasing pressure cannot be accommodated and the resistance to perfusion increases (Mars & Hadley, 1998). But the raise in intracompartmental pressure alone it is not the only thing that matters, because finally it is multifactorial ! Then, each soft tissue trauma releases bradykinin, histamine and other vasoactive substances causing vessel dilatation and increased capillar permeability.

The result is an interstitial edema caused by the penetrating tissue fluids, and at each vessel the wall gets damaged, too. Collagen fibres and thromboactive factors are released leading to disseminated vessel obstruction causing ischemia. The muscle cell react to the trauma among others by damage to the calcium pump. Influx from ions and water into the cell results, and the cell is swelling up. Potassium, creatinine and other substances leave the muscle cell. In extensive muscle damage rhabdomyolysis and myoglobinuria, kidney failure and respiratory insufficiency (= crush syndrome) can be the results. During ischemia the hypoxia is deemed responsible for the excessive dilatation of the vessels. As soon as the cause of the ischemia is solved and a normal perfusion restored, a massive diapedesis of fluid out of the vessel into the interstitium will be the answer. This amount of fluid can not be absorbed by the compartment space. The result will be a progressing increase in pressure. These pathomechanism can account for an increase in intracompartmental volume on up to 60 %. This pressure adds on the compression forces of the triggering agens increasing hypoxia and its sequelae even more (Krahn, 2005).
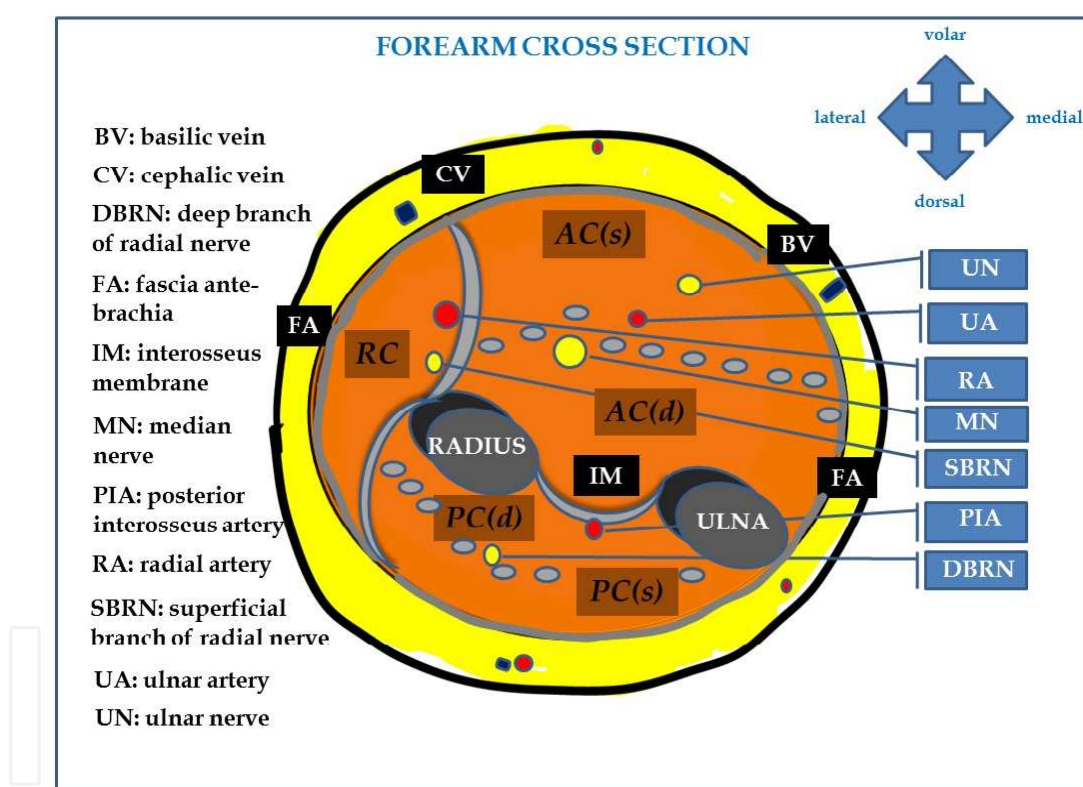

Fig. 4. Cross-section of the forearm. *AC: anterior compartment*. Superfial component accomodating pronator teres muscle, flexor carpi radialis & ulnaris muscle, palmaris longus muscle, flexor digitorum superficalis muscle. Deep component accomodating digitorum profundus muscle, flexor policis longus muscle and pronator quadratus muscle. *PC: posterior compartment.* Superfial component accomodating extensor carpi ulnaris muscle, extensor digitorum muscle, extensor digiti minimi muscle. Deep component accomodating supinator muscle, adductor pollicis longus muscle, extensor pollicis longus & brevis muscle, extensor indicis muscle. *RC: radial compartment* hosting brachioradialis muscle and extensor carpi radialis longus & brevis muscle.

Skeletal muscle can withstand a minimum 4 hours of ischemia; 4 to 8 hours of ischemia have variable outcomes. More than 8 hours of ischemia to skeletal muscle usually produces irreversible damage. However, even after 4 hours of ischemia, complications of muscle ischemia may occur, like ie significant myoglobinuria, that may result in renal failure due to acute tubular necrosis. Peripheral nerve tissue has been shown to maintain normal conduction with up to 1 hour of ischemia. Reversible neuropraxia occurs after 1 to 4 hours and irreversible axonotmesis after 8 hours of ischemia. Progressively higher compartment pressures may result in earlier irreversible changes. Reperfusion injury is also a factor in Compartment Syndromes that develop after tissue perfusion is re-established after an ischemic period. Reperfusion injury involves the production of oxygen free radicals, lipid peroxidation, and calcium influx, resulting in the disruption of the mitochondrial oxidative phosphorylation (Bibbo et al., 2000).

An injury to a major artery (Type I, Holden (1975)) might produce a Compartment Syndrome by one of these two mechanisms as well. First, the Compartment Syndrome may result if there is an inadequate collateral circulation, or if the vessel is only partially occluded, like ie arterial spasm or intima tear. In this situation the decreased perfusion and ischemia of both capillaries and muscles will cause an increase in the permeability of the capillary walls. The resulting edema will cause even more ischemia and the vicious circle will be set up. Second, with complete arterial occlusion, once the circulation is restored a Compartment Syndrome may develop from post-ischemic swelling. With complete arterial occlusion secondary to massive emboli or prolonged use of a tourniquet, in which the circulation is not restored, gangrene and not a Compartment Syndrome will result (Mubarak & Carroll, 1979).

3.4 Typical and atypical causes of forearm Compartment Syndrome

Fractures and fracture hematoma of the upper extremity, in particular those involving the elbow, forearm and wrist, have been reported as the most common causes of Compartment Syndrome in the pediatric population (Bae et al., 2001; Battaglia et al., 2002; Blakemore et al., 2000; Grottkau et al., 2005; Kadiyala & Waters, 1998; Kowtharapu et al., 2008; Krahn, 2005; Mubarak & Carroll, 1979; Paletta & Dehghan, 1994; Preis, 2000; Ramos et al., 2006; Yuan et al., 2004). Considering open vs closed forearm fractures the relative risk for developing a Compartment Syndrome is markedly increased in the open fracture group (Grottkau et al., 2005). Accompanying or fracture-related arterial injury (Choi et al., 2010; Krenzien, 1998; Mars & Hadley, 1998; Mubarak & Carroll, 1979; Ramos et al., 2006), repeated or multiple unsuccessful attempts for reduction, but also simple or undue external compression from bandages, tourniquets, plaster casts, or any position on the operating table during musculo-skeletal surgery, either elective or emergency, can augment to the Compartment Syndrome development risk, too (Bae et al., 2001; Mars & Hadley, 1998; Ramos et al., 2006; Wright, 2009).

As well as other medical procedures or complications thereout like venous access, intravenous fluid infiltration or extravasation, or a subclavian vein thrombosis can do (Bae et al., 2001; Paletta & Dehghan, 1994; Mars & Hadley, 1998). In children, Compartment Syndrome is also a recognized complication of high-energy and crush injuries, small or extensive blunt trauma, or burns (Grottkau et al., 2005; Mars & Hadley, 1998; Prasarn & Ouellette, 2011; Ramos et al., 2006; Shin et al., 1996; Wright, 2009). Not to forget rhabdomyolysis of any orign (Paletta & Dehghan, 1994; Ramos et al., 2006), ischemia-reperfusion events (Bae et al., 2001; Ramos et al., 2006; Shin et al., 1996), all types of infection

and septicemia (ie influenza, HIV, enteroviruses) (Bae et al., 2001; Mars & Hadley, 1998), and bleeding disorders (Bae et al., 2001; Paletta & Dehghan, 1994; Ramos et al., 2006). According to more and more frequently arising case reports Compartment Syndrome could also evolve after animal or insect bites (Bae et al., 2001; Mars & Hadley, 1998; Paletta & Dehghan, 1994; Ramos et al., 2006; Sawyer et al., 2010) or suction injures (Shin et al., 1996; Tachi et al., 2001).

However, an untreated Compartment Syndrome might not only be the cause of a Volkmann`s Contracture (VC), Reflexe Sympathetic Dystrophy Syndrome (RSDS) or a Vanishing Bone (VB) disease, vice versa, any of these diseases at any level or stage might trigger the development of a Compartment Syndrome as well, which finally can lead to a complete functional loss of the whole extremity (Cooney et al., 1980; Gousheh, 2010; Landi & Abate, 2010; Papadakis et al., 2011; Raimer et al., 2008; Rubel et al., 2008; Shield, 2011; Schnall et al., 1993; Zhixin et al., 2010). Especially in newborns, there are an increasing number of reports of forearm Compartment Syndromes and associated conditions with disastreous sequelae and outcomes found in current literature reviews (Christiansen, 1983; Landi & Abate, 2010; Ragland et al., 2005; Raimer et al., 2008).

Atypical causes of Compartment Syndrome reported are acute hematogenous osteomyelitis (author`s remark: in the reference case in the lower extremity but in our case in the upper extremity) (Stott, 1997) and myositis ossificans (Melamed & Angel, 2008).

3.5 Clinical signs and symptomes for diagnosis of Compartment Syndrome

Clinical signs and symptomes of a Compartment Syndrome are sometimes masked by polytrauma, a more sinister key trauma, dressings and splintages in place, or by other impressing soft tissue injuries or lesions. However, finding the right diagnosis fast is of key importance to avoid life-long sequelae. This is underscored by recent analyses of closed malpractice claims involving Compartment Syndrome, where the failure of diagnosis is one of the most common cause of litigation against medical professionals in North America. With the average indemnity payment for CS cases of 426 000 US Dollar being considerably higher than the average 136 000 US Dollar indemnity payment for any other orthopedic cases (Choi et al., 2010).

Besides a positive history, the classically described physical findings for Compartment Syndrome are the "7 Ps": *Pain*: progessive, not relieved by narcotics, out of proportion to examination; *Pain with Passive stretch* of the fingers; *Paresthesia*: diminished sense of light touch, pin prick, two-point discrimination; *Pulse*: absent pulses, absent Doppler signals; *Poikilothermia-Pallor*: a cool, pale extremity; *Paralysis*: motor deficits; *Palpation-Pressure*: a tense compartment (Bae et al., 2001; Bibbo et al., 2000; Paletta & Dehghan, 1994; Sawyer et al., 2010). With severe, persistent pain (Johnson & Chalkiadis, 2009; Paletta & Dehghan, 1994) and the tense swelling of the involved extremity (Bibbo et al., 2000; Joseph et al., 2006) being the most common reported physical findings. The individual signs and symptoms are designated as early, middle, or late according to when they become apparent during the clinical progression of the Compartment Syndrome (Sawyer et al., 2010). An adequate sensory neurological examination was not possible in most of the (pediatric) patients due to the poor patient cooperation in this particular age group (Kadiyala & Waters, 1998; Paletta & Dehghan, 1994; Prasarn & Ouellette, 2011). A high index of suspicion for a Compartment Syndrome should always be maintained in all obtunded patients. Obtunded patients are those with a dulled or altered physical or mental status secondary to injury, illness, or anesthesia; those

with diminished or absent sensation because of nerve injury or anesthesia, and the mentally ill or disabled. These patients represents the most vulnerable group whose inability to demonstrate the hallmark symptoms and signs of the syndrome put them in jeopardy of a late diagnosis of a Compartment Syndrome and its potentially devastating sequelae. This patient, but not exclusively, should be examined most closely and frequently, and any changes over time must be documented most carefully (Ouellette, 1998; Prasarn & Ouellette, 2011).

According to some authors an increasing analgesic requirement in frequency and dosage might be an early indicator of impending Compartment Syndrome even before classical signs, like "7 Ps" become evident (Bae et al., 2001; Choi et al., 2010; Yang & Cooper, 2010). Since the one of these cardinal symptoms is immense pain, a literature review was undertaken in order to assess the association of epidural analgesia and Compartment Syndrome in children, especially whether epidural analgesia delays the diagnosis, and how to identify patients who might be at risk. Evidence was sought to offer recommendations in the use of epidural analgesia in patients at risk of developing a Compartment Syndrome of the lower (!) limb. Increasing analgesic use, breakthrough pain and pain remote to the surgical site were identified as important early warning signs of minding Compartment Syndrome in the lower limb of a child with a working epidural. The presence of any should trigger immediate examination of the painful site, and active management of the situation (Johnson & Chalkiadis, 2009; Yang & Cooper, 2010).

In the special age group of the newborns, recently, all patients with Compartment Syndrome presented with a sentinel forearm skin lesion. Patterns and involvement ranged from mild skin and subcutaneous lesions to dorsal and volar Compartment Syndrome with or without distal tissue loss, compressive neuropathy, muscle loss, late skeletal changes, and distal bone growth abnormalities (Christiansen et al., 1983; Landi & Abate, 2010; Ragland et al., 2005; Raimer et al., 2008).

In summary, since most of the classic signs and symptoms of Compartment Syndrome are unreliable or unobtainable in the pediatric age group, a high clinical suspicion is needed to make an accurate and timely diagnosis (Bae et al., 2001; Bibbo et al., 2000; Choi et al., 2010; Johnson & Chalkiadis, 2009; Joseph et al., 2006; Kadiyala & Waters, 1998; Paletta & Dehghan, 1994; Prasarn & Ouellette, 2011; Sawyer et al., 2010, Wright, 2009; Yang & Cooper, 2010). Additional tests, such as compartment pressure measurements might be used to establish the diagnosis further (see next paragraph) (Mars & Hadley, 1998; Paletta & Dehghan, 1994).

3.6 Pressure measurements in children with Compartment Syndrome

Confirmation of the diagnosis of Compartment Syndrome currently entails direct recording of the tissue pressure by invasive means and different techniques of invasive recording of intracompartmental pressure have been described in literature so far. As Compartment Syndrome is a progressive phenomenon, a single recording of normal compartment pressure at one point of time does not imply that all is well. Tissue pressure can build up gradually and rise to unacceptable limits. Consequently, it may be necessary to monitor intracompartmental pressure sequentially. This can be done by repeated measurements involving skin punctures on each occasion or by leaving an indwelling catheter till the clinical situation warrants it. Both of these options have obvious disadvantages, particularly in children (Battaglia et al., 2002; Bibbo et al, 2000; Choi et al., 2010; Joseph et al., 2006; Kowtharapu et al., 2008; Krahn, 2005; Mars & Hadley,1998; Ouellette, 1998; Paletta &

Dehghan, 1994). Therefore, in allday practice in children continous monitoring is used less and in the majority of cases, compartment pressure is measured in the operating room after induction of general anesthesia to confirm the diagnosis and document the compartment pressure before fasciotomy is performed (Paletta & Dehghan, 1994).

Besides a lot of commercially available compartment pressure measurement devices, there are other handmade devices in use like a handheld saline infusion manometer (Fig.5) or arterial line and pressure transducer devices (Bibbo et al, 2000; Choi et al., 2010; Krahn, 2005; Paletta & Dehghan, 1994). A standard 18-gauge hypodermic needle connected to an arterial line and pressure transducer is also an acceptable method for measuring compartment pressure (Bibbo et al, 2000; Paletta & Dehghan, 1994). This technique has the advantage of being readily available in most emergency departments, and it has been demonstrated to correlate well with compartment pressure readings taken otherwise from commercial compartment pressure measuring devices (Bibbo et al, 2000).

Fig. 5. Compartment pressure measurement device in place.

In order to assess the feasibility of using measurement of tissue hardness as a method of diagnosing Compartment Syndrome non-invasively in children, a simple handheld device to measure tissue hardness was fabricated. The relationship between hardness and compartmental pressure was studied in an experimental model and in three fresh amputated lower limbs. Experimental data from this study suggest that there is a nonlinear relationship between intracompartmental pressure and tissue hardness. The study also showed that tissue hardness can be measured reproducibly in the forearm of children with this device. However, further refinement of the measuring device and well designed clinical trials are needed to establish whether CS can finally be diagnosed reliably by measuring tissue hardness non-invasively alone (Joseph et al., 2006).

At the tissue level an increase in compartment pressure leads to compression of the venous and lymphatic outflow. When this obstruction to outflow is greater than the mean arterial pressure, decreased tissue perfusion and ischemia occur. But adequate cell nutrition, and hence viliability, is dependent upon this adequate tissue perfusion with adequate diffusion of nutritients, including oxygen, within the tissue. While the ultimate driving pressure is the mean arterial blood pressure, it is the pressure in the microcirculation, opposed by tissue and venous pressures, which determines the adequacy of nutrient bloodflow in the capillaries. Cell viability can therefore be threatened by any mechanism, which elevates venous and tissue pressures or which reduces mean arterial pressure, and it is the relationship between these two competing forces which determines the outcome. In an expansile space, any tendency of tissue pressure elevation can be balanced by an increase in volume of the space. In a closed compartment, either osseo-fascial or fascial alone, the increasing pressure cannot be balanced and resistance to perfusion increases (Mars & Hadley, 1998; Sawyer et al., 2010).

Some investigators believe, that this occurs at a compartment pressure of 40 mmHg or more, while others believe it occurs when the pressure gradient between arterial inflow and venous outflow approaches 30 to 40 mmHg of mean arterial pressure (Krahn, 2005; Mars & Hadley, 1998; Sawyer et al., 2010). Other trauma protocols consider compartment pressure to be significantly elevated when the pressure reading falls within 30 points of the diastolic blood (mmHg) pressure; with the utility of comparing this compartment pressure reading to the patient`s diastolic blood pressure to be proven in experimental Compartment Syndrome models to be predicitive of the threshold for neuromuscular ischemic damage (Bae et al., 2001; Bibbo et al, 2000; Krahn, 2005). The normal pressure in a muscle compartment is reported to be less than 10 to 12 mmHg and the blood flow in the capillary circulation is reported to cease when compartment pressure exceed 35 mmHg (Ramos et al., 2006). But intracompartmental pressure are reported to vary within the lowest pressure measured at 25 mmHg and the highest at 86 mmHg (Krahn, 2005; Paletta & Dehghan, 1994, Sawyer, 2010). And authors are cited, that peak compartment pressures do not influence the patient`s outcome at all (Battaglia et al., 2002; Krahn, 2005), respectively that they even vary considerably periprocedural (ie pre- and post reduction) and according to the site of location, as well as amongst individuals themselves (Battaglia et al., 2002). While some authors recommend decompression at an absolute compartment pressure, others believe that there is no absolute pressure defining Compartment Syndrome at all. However, the overall experience suggest that in children, directly measured compartment pressures are useful data, but should not deter fasciotomy in patients with obvious deteriorating physical findings or displaying the classical signs and symptoms already mentioned (Bibbo et al., 2000; Krahn, 2005; Kowtharapu et al., 2008; Ouellette, 1998; Ramos et al., 2006).

Briefly summarized: Besides the typical clinical findings, initial and repeated compartment pressure measurements are a recommended diagnostic tool, but it is an invasive and scarring procedure, time consuming and the value of the readings is often limited or at least questionable. And peak intracompartmental pressure do not necessarily match with the severity of the condition of the patient in due course.

3.7 Management and treatment of Compartment Syndrome

Surgery remains the mainstay in treatment for acute Compartment Syndrome and definitive treatment must consist of emergent fasciotomy of ALL involved compartments. With early

diagnosis and expeditious treatment being of key importance (Bae et al., 2001; Battaglia et al., 2002; Berger & Weiss, 2001; Bibbo et al., 2000; Blakemore et al., 2000; Choi et al., 2010; Christiansen et al., 1983; Cooney et al., 1980; Gousheh, 2010; Grottkau et al., 2005; Kadiyala & Waters, 1998; Kowtharapu et al., 2008; Krahn, 2005; Krenzien et al., 1998; Landi & Abate, 2010; Mars & Hadley,1998; Mubarak & Carroll, 1979; Ogden, 2000; Ouellette, 1998; Paletta & Dehghan, 1994; Prasarn et al., 2009; Prasarn & Ouellette, 2011; Preis 2000; Ragland et al., 2005; Ramos et al., 2006; Sawyer et al., 2010; Shin et al., 1996; Stott et al., 1997; Tachi et al., 2001; Yuan et al., 2004; Zhixin et al., 2010).

Fasciotomy should always be performed according to the individual compartments and structures most likely involved, and according to the common incision lines regularly used in hand surgery practice. Ensuring a full and complete fasciotomy is also essential, since an incomplete fasciotomy poses a considerable risk for damaging the forearm structures even further. Getting proper access to the region of interest is essential, but any additional (surgical) trauma to the tissue must be reduced to a minimum. Any bleeder must be stopped meticiously. Incision lines should never cross skin folds perpendicular and never leave badly perfused flaps or skin margins behind (Berger & Weiss, 2001; Krenzien et al., 1998; Ogden, 2000). Splinting, elevation, and delayed wound closure usually follows the simple compartment fasciotomy (Bae et al., 2010; Battaglia et al., 2002; Blakemore et al., 2000; Choi et al., 2010; Cooney et al., 1980; Christiansen et al., 1983; Kowtharapu et al., 2008; Krahn, 2005; Landi & Abate, 2010; Mars & Hadley, 1998; Mubarak & Carroll, 1979; Ogden, 2000; Ouellette, 1998; Prasarn et al., 2009; Prasarn & Ouellette, 2011; Ragland et al., 2005; Ramos et al., 2006; Sawyer wt al, 2010; Shin et al., 1996; Stott et al., 1997; Zhixin et al., 2010).

Concomitant fractures will be fixed according to actual guidelines and textbooks, using either cast splintage or ORIF techniques. The latter becoming more and more prefered (Dietz et al., 1997; Fette et al., 2009; Grottkau et al., 2005; Kadiyala & Waters, 1998; Ogden, 2000; Preis, 2000; Yuan et al., 2004), since in those limbs being at risk for Compartment Syndrome, bony fixation like ie percutaneous pinning or intramedullary fixation prevents problems usually noticed with the standard cast treatment (Fette, 2010, Grottkau et al., 2005; Kadiyala & Waters, 1998; Ogden, 2000; Preis, 2000). However, there is also an increased incidence of Compartment Syndrome reported for patients treated with intramedullary fixation (ESIN), especially for the ones with long surgery times and long use of intraoperative fluoroscopy, when compared to patients treated with closed reduction and casting alone (Yuan et al., 2004). Any bone reconstruction must be stable enough right from the beginning for an early (self-) mobilization by the child or a guided pediatric physio- or occupational therapy. Removal of implants usually can be scheduled according to common standards and protocols (Dietz et al., 1997; Fette et al., 2009; Fette, 2010; Grottkau et al., 2005; Kadiyala & Waters, 1998; Ogden, 2000; Preis, 2000; Yuan et al., 2004).

Concomitant nerve-, vessel- or tendon injuries upgrade the simple Compartment Syndrome into a complex one. In principle, surgical therapy comprises freeing of the delicate structures and their meticulous (micro-) surgical repair to restore function as much as possible, quite often as a multi-staged and interdisciplinary procedure (Berger & Weiss, 2001; Cooney et al., 1980; Fette, 2010; Gousheh, 2010; Krenzien et al., 1998; Landi & Abate, 2010; Ogden, 2000; Ragland et al., 2005; Raimer et al., 2008, Zhixin et al., 2010).

The same applies for potential trigger or potential in due course morbidities like Volkmann`s Contracture, Reflex Sympathetic Dystrophy Syndrome or Vanishing Bone disease. Here usually the whole armentarium of non-surgical and reconstructive-surgical treatment modalities are required. These include invigorating activities and extensive physio- and occupational therapy, if indicated as well (Fette et al., 2002; Papadakis et al., 2011; Schnall et al., 1993; Shield, 2011).

Classical management of the fasciotomy is delayed primary (= secondary) closure. The splitted fascia and skin are usually covered by impregnated gauze and cotton wool or by a foam dressing. The margins in most cases are approximated by a dynamic suture, until the swelling has settled down completely, and the skin can be finally closed by direct suture. Respectively, by a skin graft or even more rarely by a flap (Fette et al., 2002; Paletta & Dehghan, 1994; Tachi et al., 2001). Modern treatment takes advantage of the topical negative pressure therapy (ie V. A. C.®, KCI Medical, Wiesbaden/Germany). The wound surface is protected by a silicone sheet, the wound margins by a hydrocolloid, just before the foam is applied, and fixed to the wound margins by stitches or staples. This altogether is sealed by foil, the TRAC™ pad is installed and the negative pressure in continuous mode (authors remark: 75 mmHg) set up by the V. A. C.® machine. As soon as the swelling went down (24 to 72 hours), the machine is switched to intermittend mode and the negative pressure can be increased in the majority of cases. Immediately putting some "massage-like" and stretching effect on the tissue and skin margins and therefore acting like a "tissue expander"-device. Making fasciotomy closure finally fast, easy and complete tension-free (Fette & Epp 2007; Fette, 2008; Fette & Doede, 2009; Willy, 2005).

3.8 Outcome after fasciotomy for forearm Compartment Syndrome

Early recognition and expeditious treatment are essential to obtain a good clinical outcome and prevent permanent disability (Bae et al., 2001; Prasarn & Ouellette, 2011; Shin et al., 1996; Ramos et al., 2006; Wright, 2009). Thus, the large majority of the patients will achieve full restoration of function (Bae et al., 2001; Gousheh, 2010; Prasarn & Ouellette, 2011; Shin et al., 1996; Ramos et al., 2006; Tachi et al., 2001). However, a well and uneventful healed fasciotomy after a Compartment Syndrome regularly gets a 10 % occupational disability credit in the German medical appraisal system (Krahn, 2005). Improved approaches to assessment and better management, together with early recognition, helped to reduce the bad outcomes (Krahn, 2005; Shin et al., 1996; Wright, 2009). But there is still a considerable risk for developing Volkmann`s Contracture with significant morbidity (Cooney et al., Gousheh, 2010; Krahn, 2005; Kretzien et al., 1998; Landi & Abate, 2010; Mubarak & Carroll, 1979; Ragland et al., 2005; Raimer et al. 2008; Paletta & Dehghan, 1994), Reflex Sympathetic Dystrophy Syndrome (Shield, 2011; Cooney et al., 1980), or Vanishing Bone disease (Papadakis et al., 2011; Rubel et al., 2008; Schnall et al., 1993) anyway. Recent reports on this entire entity focusing especially on newborns showed even more complex and disastreous outcomes requiring even more complex and demanding reconstructive procedures than ever seen previously in the past (Christiansen et al., 1993; Landi & Abate 2010; Ragland et al., 2005; Raimer et al., 2008).

According to a historical report, the volar and occasionally the volar and dorsal compartment together are mainly involved in older patients with supracondylar humerus and forearm fractures (Mubarak & Carroll, 1979).

Average time to surgical intervention from the time of increasing signs and symptoms are reported to be around 18-25 (range 1-96) hours (Bae et al., 2001), but there are others reporting >> 24 hours delay in the initial diagnostic process as well (Mubarak & Carroll, 1979). Full functional recovery is anticipated within 6 months in most patients (Bae et al., 2001), and average follow-up reported is 22 months (Prasarn et al., 2009).

Since there is no specific predictor or indicator, nor a clinical sign or symptom or any technical or lab investigation to diagnose a Compartment Syndrome fast and safely, the most powerful indicator is the suspicion of the attending (pediatric) surgeon. Compartment pressure measurements might be helpful and indicative, but especially in the pediatric age group it has to be considered, that they are painful, invasive and need specified technical equipment, that is not commonly available. Surely, this high tech equipment can be replaced by components easily available in any pediatric surgery department or ER, but their set-up is time consuming and measurements taken still less reliable. Overall experience in diagnosing (pediatric) Compartment Syndrome among ER or pediatric surgery staff is usually limited, as well as it is with putting a pressure probe device in action. And so far there are still no table of normal values available and compartment pressure can vary considerably in due course and even within the individual. Conditions like polytrauma, splintage, Volkmann Contracture, Reflex Sympathetic Dystrophy Syndrome or Vanishing Bone disease might mask or even trigger the underlying Compartment Syndrome. Waiting for the so-called historical signs, impalpable pulse and neurological deficit, is too dangerous, then when realising them it is much too late.

4. Conclusion

The potential risk of an even late onset Compartment Syndrome should always be considered in all children presenting with a remarkable forearm injury. Clinical signs and symptoms are various, inconstant and unreliable. The only consistent finding is the suspicion of the attending surgeon. Immediate surgical release of the Compartment Syndrome, stable fixation of the concomitant fracture and meticulous repair of all important anatomic structures involved is mandatory to prevent these children from disastreous sequelae and outcomes. By reducing the swelling and in parallel stretching the skin, the V.A.C.® device, throughout the intermittend mode, facilitates and fastens the tensionless direct closure of the fasciotomy site.

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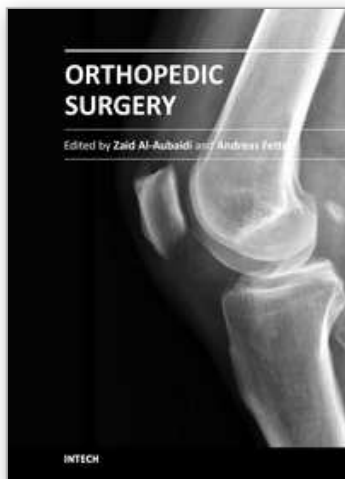
Orthopedic Surgery

Edited by Dr Zaid Al-Aubaidi

Orthopaedic surgery is the widest and the strongest growing surgical specialty. It is clear, that the process of improving treatments and patients care, requires knowledge, and this requires access to studies, expert opinion and books. Unfortunately, the access to this knowledge is being materialized. As we believe that access to the medical knowledge should be reachable to everyone free of charge, this book was generated to cover the orthopaedic aspect. It will provide the reader with a mix of basic, but as well highly specialized knowledge. In the process of editing this book, my wife Jurgita has been, as usual, the most supportive person. I would like to thank her for being in my life. I would like to thank Mr. Greblo, the Publishing Process Manager, for all his help and last but not least thanks to our readers, as without them this book would have no meaning.

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