

**Controversies in
the assessment
of bodily injury.
Institute of
Legal Medicine
of Catalonia**



Generalitat de Catalunya
Departament de Justícia

Centre d'Estudis Jurídics i Formació Especialitzada
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My sincere thanks go to all of them.

Dr. Lluïsa Puig
Coordinator

**CONTROVERSIES
IN THE ASSESSMENT
OF BODILY INJURY.
INSTITUTE OF LEGAL
MEDICINE OF CATALONIA**

COORDINATION:
DRA. LLUÏSA PUIG BAUSILI



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Preface

These papers have been prepared by professionals at IMLC.

The topics covered are amongst those that generate the greatest amount of controversy nowadays within the field of bodily injury assessment. In order to obtain consensual solutions to the controversies presented, we decided to create an exhibition of various topics in the format of a clinical session prior to publication. These sessions were conducted weekly at the IMLC offices, and the presentations - which are the product of scientific reasoning, supported by the case law study carried out - were meant to be assessed and questioned by fellow attendees. Therefore, this study was not only made by the authors of the papers, but also through the active and critical participation of those who attended the sessions.

Given the fact that, in general, the number of participants in each project is significant, we decided to appoint specific group coordinators who have been marked with (*) next to their name, and finally a coordinator for the entire project.

It is our hope that the results of the controversies presented prove useful and practical for our daily work. We also hope that all issues raised are resolved, but not in a dogmatic way because as always happens in the field of forensic medicine, each case must be studied individually and exceptions can always occur.

Dr. Lluïsa Puig
Coordinator

Chapter I

Medico-Legal Criteria for Basic Medical Attention and Medical or Surgical Treatment

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1. Reason for the controversy

The distinction between basic medical attention and medical or surgical treatment goes back twenty years but remains a live and everyday controversy in forensic medicine practise. The crucial point is whether or not mandatory medical treatment or minor surgical procedures should be included in the concept of *basic medical attention*. We have reviewed the case law on this issue and discuss its implications for bodily injury assessment.

2. Introduction

The distinction drawn between *basic medical attention* and *medical or surgical treatment* in Act 3/1989 (see Table 1) came under immediate scrutiny by a number of authors due to its medico-legal [1,2] and juridical [3] implications. Moya [4] grouped the interpretations into two opposing blocks: an extensive criterion for basic medical attention with a restrictive one for medical and surgical treatment (for instance in State Attorney General Circular 2/1990; see also Bernal [5] and Orós *et al.* [6]), and a restrictive criterion for *basic medical attention* with an extensive one for medical and surgical treatment (for instance the Supreme Court Ruling of 1 July 1992; see also Hernández [7] and Zugaldía and Hernández [8]). As an illustrative application of these criteria, according to the former stitches would be *basic attention* in the case of minor injuries while the latter says they are always “surgi-

* Group Leader.

cal treatment”. Thus as Moya puts it [4]: “the prevalence of one or the other concept will have major consequences.” The first aim of this study was therefore to find out whether the Supreme Court’s case law has consolidated one or the other of these two approaches.

There is an added difficulty with the medico-legal criterion in establishing undisputed use of the terms as they make up a metalanguage. In other words, they are based on medical concepts but use terminology that belongs to the field of law. Forensic medicine is aware that the legal criterion prevails over the medical criterion and so it explores case law to try to find a rule applicable to all cases, but normally only emerges with a minimal generalisation and some *numerus clausus*. The problem is further exacerbated when new pathological entities, diagnostic tests and treatments emerge that are not in the minimum generalisation and *numerus clausus*, or when as in recent times the concept of healing changes and both restoring health and also improving quality of life have to be taken into account. As a result there is a lack of scientific rigour in the use of the terms, a clear legal and medical conflict in this use, and a lack of unified criteria in forensic medicine. Hence the second purpose of this paper has been to try to establish at least the foundations for a unification of the medico-legal criterion.

Diagram 1. Assault and battery in Section 420 of the previous Criminal Code (the wording from Act 3/1989) and Section 147 of the current Criminal Code.

Section 420.

Whoever by any means or procedure causes injury to another person that impairs their physical integrity or their physical or mental health shall be punished with imprisonment for from six months and a day to six years, provided that the injuries require medical or surgical treatment for their cure in addition to basic medical attention.

However, the fact described in the preceding paragraph shall be punished with the penalties of imprisonment for from one month and a day to six months or a fine from 100,000 to 500,000 pesetas based on the nature of the injury and other circumstances of fact.

Section 147.

1. Whoever by any means or procedure causes injury to another person that impairs their physical integrity or their physical or mental health shall be punished for the crime of assault and battery with imprisonment for from six months to three years, provided that the injury objectively requires medical or surgical treatment for its cure in addition to basic medical attention. Mere medical surveillance or monitoring of the progress of the injury is not considered medical treatment [...].
2. Nevertheless, the fact described in the preceding paragraph shall be punished with the penalty of imprisonment for from three to six months or a fine per day equivalent to from six to 12 months when less serious depending on the means employed or the results produced.

3. Material and methods

We have used La Ley database of Supreme Court rulings from 1989 to 2008, both inclusive. We searched all rulings containing Section 420 of the former Criminal Code and Section 147.1 of the current Criminal Code in their keywords and obtained 549 rulings. We then read all of them to see whether they were relevant to the purpose of our study, in other words if they addressed the distinction between “basic medical attention” and “medical or surgical treatment”. We selected the relevant rulings which we then studied to identify general definitions and the reasoning for applying these definitions to specific injuries or treatments. We also decided which rulings were “original” in the sense that they set out new general definitions. We then held several rounds of discussion between the authors.

4. Findings

Out of the 549 rulings obtained in our search, 122 (22%) were considered relevant; 49 were for the previous Criminal Code and 73 for the current Criminal Code. Table 1 shows the distribution and the percentage of the total number of rulings we obtained in our search by years. Only 3 of the rulings selected, from 1992, 1993 and 1997, were considered completely “original”.

Table 1. Number of rulings considered relevant for the former Criminal Code (FPC) and the current Criminal Code (CCC) and the rulings considered relevant as a percentage of all rulings found in the search by year.

YEAR	FCC	CCC	%
1989	0	0	-
1990	0	0	-
1991	0	0	-
1992	2	0	12%
1993	6	0	21%
1994	11	0	26%
1995	4	0	11%
1996	8	1	19%
1997	9	0	25%
1998	5	7	41%
1999	1	9	44%
2000	2	7	39%
2001	1	2	14%
2002	0	6	27%
2003	0	11	32%
2004	0	8	57%
2005	0	3	27%
2006	0	7	41%
2007	0	7	35%
2008	0	5	36%
Total	49	73	22%

5. Discussion

One limitation of our search is that it cannot be considered exhaustive, as shown by the fact that sometimes the selected rulings refer to previous rulings that the search did not identify. For example, we did not find the early rulings of 26 and 28 February 1992. However, we did have access to their cognisance and that of other rulings through the literature we consulted.

There is a clear trend towards uniformity in the general definitions throughout the period studied, albeit with different verbatim formulations, which include the following chosen for their brevity¹: medical treatment is a “system used to heal an illness”² and surgical treatment consists of “acting directly on the body to heal damaged tissue and return it to its previous state”.³ In both cases the concept of “healing” is broad. Indeed, with regard to medical treatment the above definition goes on to say “or to try to minimise the consequences if it is not curable”, whereas for surgical treatment the ruling adds that it includes “speeding up healing or preventing or mitigating unsightly consequences as both purposes can be deemed to be within the broad concept of healing”. In most decisions “basic medical attention” is implicitly defined by exclusion; at most it is “something like the initial diagnosis of the existence of an injury”⁴ and barely extends to include care for injuries that are “trifling, [...] very slight”.⁵

In spite of this trend, there are inconsistencies in the application of the definitions to specific cases. This is the case of assessments that seem to be based on other criteria (“breaking at least one rib is, due to its importance, an impairment of health that requires medical treatment”⁶) or matters of form (basic attention for a nasal fracture causing a deviated septum because treatment is not stated⁷), apparently contradictory rulings (breaking a tooth is considered to require treatment as it has to be repaired⁸ or only basic attention because it did not require any further action⁹), deviations due to excess (considering treatment in relation to a small non-sutured wound¹⁰ or a wound to which

1 All the rulings quoted are from the Second Criminal Chamber of the Supreme Court.

2 Ruling of 13/02/04.

3 Ruling of 14/05/02.

4 Ruling of 06/02/93.

5 Ruling of 01/07/92.

6 Ruling of 12/12/96.

7 Ruling of 27/10/03.

8 Ruling of 25/03/03.

9 Ruling of 02/01/03.

10 Ruling of 04/06/04.

steri-strips were applied,¹¹ or “blows to the head” in this case due to the area involved even though only observation and monitoring were required¹²) or by default (“orthopaedic treatment consisting of splinting an arm and putting on a neck brace” is assessed as basic attention¹³), or medically surprising reasoning (“everyone knows, however limited their medical knowledge may be, that in the case of a cervical sprain it is essential, in general terms and excluding cases of particular seriousness, to immobilise the cervical vertebrae in a certain position which is done by putting on the prescribed neck brace”).¹⁴

The fact is, however, that the Supreme Court commonly refers to its case law as being settled (“determining the concept of medical or surgical treatment is still the subject of dispute in legal scholarship, but the position of the Supreme Court is already unanimous and settled”¹⁵), and there is even a list of therapeutic procedures and their established legal assessment in the literature we reviewed. [9] In this regard our review found that the additions to the new Criminal Code (“objectively” and “mere medical surveillance or monitoring of the progress of the injury is not considered medical treatment”) did not involve any modification of case law. For example, “the word ‘objectively’ has been added to the new text [...] although this is nonetheless completely irrelevant because such objectivity had already been required by the case law of this court.”¹⁶ However, appeals concerning this dispute are still being filed with no clear downward trend at least in the period studied.

In this regard it is worth noting that there are reference texts such as the one by Castellano [10] setting out the “medico-legal criteria” of “basic medical attention” and “medical or surgical treatment”. This author defines “basic medical attention” by a time (“the first time that the doctor comes into contact with the victim to provide health care”) and a behavioural (“the medical procedure by which the effects of the violent mechanism are evaluated”) criterion which includes “identifying the nature of the injury and assessing its severity” and also “prescribing and performing the treatment the patient needs”. Thus “medical treatment” is what follows this “basic medical attention” and is “the subsequent therapeutic approach” in a “continuum of medical procedures”, while “surgical treatment” is determined by the need for major surgery or the intervention of specialists. As can be seen, the “medico-legal criterion” differs from the “case law criterion”: going back to Moya’s distinction [4], the former extends basic attention and restricts medical and surgical

11 Ruling of 17/07/01.

12 Ruling of 13/03/03.

13 Ruling of 28/04/98.

14 Ruling of 15/12/04.

15 Ruling of 09/12/98.

16 Ruling of 09/12/98.

treatment, while in the latter the other version has prevailed. Our review shows that the divergence of these two pathways is due to two different starting points: the medico-legal criterion immediately establishes a definition of basic attention so that treatment has to be anything that goes beyond this, while the case law criterion first defines treatment and thus leaves basic attention practically devoid of therapeutic content.

According to the Supreme Court, “assessment about the nature of treatment, with legal significance, of medical intervention is not for experts in medicine but rather for the Court, as is the case with the determination of the need for medical intervention.”¹⁷ Yet the fact is that commissioning such assessments is a common and everyday occurrence in forensic medicine. Aspects which may be taken into account in this respect include the nature and seriousness of the injury, its evolution and potential complications, the patient’s previous condition and other characteristics, the characterisation of medical procedures (diagnostic, prophylactic, therapeutic, palliative, etc.) and the description of their number, frequency and effectiveness, the length of the healthcare process up to healing or injury stabilisation, the need for compliance with the guidelines prescribed, the qualifications of the person who has provided care, etc. The distinction between a genuine treatment plan and the discretionary advice or recommendations that are almost always found in any care for health problems seems particularly relevant with respect to medical treatment, as is the description of the procedures carried out including information about the severity of the injury for surgical treatment. Three questions should be answered when expressing a view about the treatment. Is it necessary and essential for the healing of the injuries? If healing is not possible, is it necessary and essential to minimise their consequences? And finally, does it comply with *lex artis*? If the answer is yes to any of these questions, then it is an instance of medical or surgical treatment.

6. Final thoughts

Our review and the subsequent discussion among the authors have made the miscommunication between doctors and lawyers especially clear. Thus a medical forensic expert can be faced with conflicts between a medical point of view, rooted in their training, and the outcome of an assessment influenced by case law. In such situations it should always be borne in mind that legal concepts are not identical with what is understood in our profession by medical versus surgical, that severity

¹⁷ Ruling of 12/07/02.

in clinical terms or a prognosis do not necessarily coincide with the criminal description, that the offence of assault and battery itself allows for upward (aggravated injuries) or downward (lower severity) graduation that remains in the hands of legal actors. Here we reiterate our recommendation with respect to treatment: is it necessary and essential for the healing of the injuries? If healing is not possible, is it necessary and essential to minimise their consequences? Does it comply with *lex artis*? If the answer is yes to any of these questions, then it is an instance of medical or surgical treatment.

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Chapter II

Sutures: Surgical treatment v basic medical attention

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1. Reason for the controversy

The Supreme Court understands that when an injured party's medical care involves sutures it is included within the legal concept of medical and/or surgical treatment.

We understand that from medical and legal standpoint (not strictly legal) this is not always the case.

We believe that sutures are not always essential to heal certain wounds (or injuries to the skin) as other treatments (Steri-Strip, adhesives, etc.) that would be included in the legal concept of basic medical attention can be used instead, always with the understanding that stitches do not heal the wound as it is the tissue that grows and closes up. Stiches only join, resist pulling, prevent cavities from forming and shorten healing time.

Therefore, our proposal to resolve this controversy is presented below:

2. Introduction

The intention of our group has been to show the differentiation between medical and legal concepts regarding the surgical treatment, to know what case law states in relation to this concept and to propose

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objective criteria for a more or less agreed way to assess the wounds that have been sutured using either approach (basic medical attention or surgical treatment).

3. Concepts

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Surgical treatment. That which is part of or is related to surgery. Synonym: operative [1]. That which is part of related to surgery or may be corrected using techniques from this branch of medicine [2:1491].

Surgery. (lat. *Khirurgia*, gr. *Kheir, mano, érgon, treball*). Branch of medicine that treats diseases, injuries and deformities through manual or operative means. ...

Operative surgery. Operative or mechanical aspect of surgery, a portion of which concerns manual and manipulative methods or procedures [2:337].

Tissue adhesives. Composition: monomer N-butyl-2-cyanoacrylate. When in contact with tissue fluids it becomes polymerised. Blue and colourless. Indications: closure of skin. Sclerotherapy of esophageal varices and fundus. Colourless, closing up facial wound.

4. Comments on case law with respect to sutures

The concept of *medical and/or surgical treatment* is a technical and legal concept although its interpretation and integration requires knowledge and experience-based guidance offered by medical science.

4.1. There is an insistent and pacific case law doctrine that recommends and requires sutures to be used as part of surgical treatment: “No matter how simple the operation, *it always a remedial measure involving the use of surgical mechanisms*, including those intended for minor surgery and even when the procedure is performed as part of basic medical attention, as *outside the realm of outright prevention or observation*, any injury requiring medical or surgical intervention is considered as *active treatment*”.

4.2. Another ruling refutes certain contradictions in the Court a quo in which after affirming (in the first instance) that “when applying sutures

for wound treatment, it should always be considered that there is more than one medical treatment” followed surprisingly later by “if this is not followed by medical intervention other than basic attention, even if it consists of sutures, it cannot be argued that there has been treatment in addition to basic assistance” Given this inconsistency, the Court of Appeals determined *ad quem* that “there is little doubt that for injuries requiring sutures, in addition to emphasising that after an initial medical diagnosis a surgical intervention was necessary, regardless of how minimal, *the intervention will be defined as remedial to restore or correct through the use of greater or lesser surgical means any functional impairment caused as a result of the injury*”.

4.3. Another first instance ruling stated: “The sutures applied to the patient were NOT essential for wound treatment but only to speed healing and repair cosmetic damage”. It also stated that “the partial fracture of a tooth is not comparable to the loss of the same”. But the Appeals Court ruled on the case and categorically established the absolute inadmissibility of the two previous statements for being in conflict with “the consistent interpretation that JPCIA has made of the subjective and objective elements of the offence of assault and battery from Section 147 of the Criminal Code”, subsequently affirming that “a surgical treatment such as the application of sutures entails acting directly on the body to repair damaged tissue and return it to its previous state [...] *assigning no significance to whether the intervention is major or minor surgery*, and without breaking the logical nexus between the injury and the medical or surgical treatment, as they will be targeted at speeding up healing or mitigating the anti-aesthetic sequelae as both purposes are understood to be included within the broad concept of treatment”.

4.4. More illuminating is the ruling of the Supreme Court which stated categorically that “sutures are considered as minor repair surgery, since the stitching (*sic*) used to join tissues or the outer portions of an open wound *is necessary to restore the damaged tissue and return it to its state previous to the injury even when the sutures are applied as basic medical assistance without the need for further monitoring or special medical care for the Health*”.

4.5. An interesting aspect of the case law is the value placed on the adverb *additionally* in Section 147.1 of the Criminal Code in the sense that medical and surgical care *does not have to necessarily* be provided at a separate time with respect to basic medical attention but that its meaning is to *add or accompany* actions carried out though basic attention. It therefore states that “basic medical attention is *comparable* to a initial diagnosis or medical examination, and it is possible that this assistance

includes curative care administered on an *ad hoc* basis (disinfection, bandages, etc.) that does *not* constitute medical treatment defined as *any treatment prescribed by a qualified medical professional for healing purposes* with no regard to previous or subsequent activities which led to the development *or fulfillment of this system being implemented by a doctor or health professional or by the patient, provided that it has curative purposes* which are not merely preventive in nature”.

4.6. Otherwise, it is affirmed that “the concept of medical treatment entails the existence of a health impairment whose treatment or cure requires medical intervention involving the creation of a healing scheme that will ameliorate its consequences or that leads to non-painful healing process that is objectively necessary and that does not involve simple monitoring or surveillance”.

4.7. The following conclusions are therefore presented:

- Sutures are always a surgical treatment in accordance with criminal code 147.
- Their application is distinguished from the concept of basic medical attention (ie, the adverb ALSO is applicable)
- The party acting in subsequent treatments (for removal of the sutures) is of no significance.
- This scenario gives rise to the need for agreed medical and legal criteria for the application of the concept of *need*.

5. Need criteri

Age criteria. In children under 14 years of age (approximately), provided that wounds are sutured in individuals older than 14 years of age or adults depending on the below described factors.

Depth criteria. Wounds that injure more than one plane need to be sutured in a way that ensures healing in each plane with minimal risk.

Length criterion. Wounds with a length of more than 1.52 cm must be sutured to ensure the union of edges and to reduce the likelihood of aesthetically unwanted results.

Location criteria. In areas of flexion (joints, etc.) sutures are necessary to ensure that the union of edges are maintained.

Facial wounds should be sutured if they do not fall on Langer’s lines. If this is the case, the abovementioned depth and length criteria must be adhered to, although in terms of length we are considering suture wounds of 1 cm (as opposed to 1.5-2 cm).

Criteria for the existence of additional complications. Wounds with tearing (irregular) or to the *scalp* require sutures to ensure the union of edges and to attain appropriate aesthetic results.

In accordance with the foregoing, *in cases that do not meet the stated criteria*, it is considered that wounds involving less than one plane and that are less than 1.5-2 cm deep generally do not require sutures as Steri-Strips could be used in their place (eg) the aim in using them would be to reduce sequelae, infection risks and speed healing.

For facial injuries less than 1 cm, sutures would not be necessary. Their use would result in less severe aesthetic sequelae.

Erosions are never sutured.

Scalp wounds do not need to be sutured (provided they do not meet any of the previously mentioned criteria), but if they are it would be for haemostatic reasons.

6. Special cases

The need for the Friedrich wound-cleaning method: should always be considered as surgical treatment.

Foreign bodies: their removal involves surgical intervention.

Bites (animal/person): they are by definition contaminated wounds which require antibiotics and tetanus prophylaxis (entailing medical treatment). They are NOT sutured for medical reasons, not because it is not necessary, but because they are contaminated wounds which are not sutured through medical choice. In regards to facial bites, plastic surgeons occasionally decide to suture them, entailing exhaustive (daily) monitoring of the healing process, which is rather difficult in habitual practice.

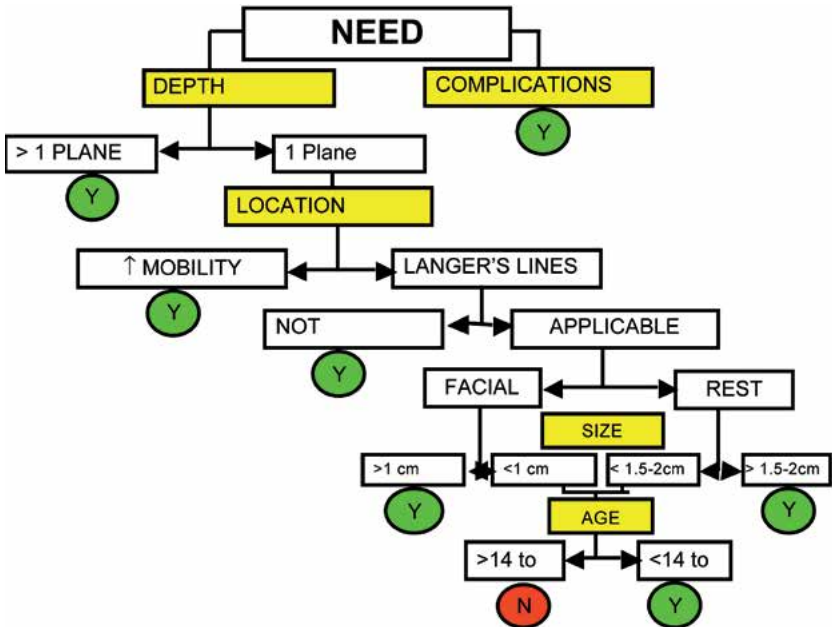
7. Conclusions

From all that has been outlined, we consider the most salient conclusion to be that necessity criteria (age, extension, depth, location and additional complications) will determine whether we consider them to be compatible with the legal precepts for basic medical attention (those which do not meet them) or for medical and/or surgical treatment (those which do not meet them).

We consider it appropriate, for teaching purposes in the legal field, to mention in a systematic way in the medical health report, after stat-

ing the treatment that has been received (for example: pharmacological treatments, complementary tests, sutures, topical treatments, etc.) what is our criteria is in regards to compatibility with the legal concepts, using the phrase “this treatment received is compatible with what is known from a legal standpoint as basic medical attention/surgical treatment”.

This is summarised in the following table:



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Chapter III

General and Specific Assessment of Previous Condition

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1. Introduction

Previous condition is one of the most important and yet most controversial factors in assessing causation in bodily injury. Knowing a person's anatomical and functional condition prior to a traumatic event and above all the influence of this condition on the evolution and outcome of trauma is one of the most problematic aspects in medico-legal expert assessment of bodily injury.

Once the traumatic event has occurred, there are a number of factors that influence the evolution of injuries and especially the final condition of the person concerned. Knowing the person's prior inherent circumstances and how they influenced the final process is essential to establishing causation.

This aspect is one of the most controversial issues in habitual forensic medical assessment of bodily injury. It is for this reason that we will attempt to understand how previous condition influences the diagnosis of pathologies and their evolution and also establish guidelines to determine causation and thus be able to provide objective expert assessment.

Obviously previous condition affects any area of the work of a medical forensic expert. However, since individualised assessment of each process would be impossible, we have chosen the most frequent pathologies or the ones that we think may lead to most conflicts and which affect the shoulder, spine and knee.

Nonetheless we also believe that the aspects set out below affect any medico-legal aetiology that calls for expert assessment, i.e. irrespective of the origin of the traumatic event which may be a traffic or occupational accident or assault.

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2. Rotator Cuff Pathology

Dr. Elena Cano Rodríguez and Dr. Maria Vela Quesada

2.1. Introduction

Painful shoulder pathology is one of the symptoms that often appear in medical forensic expert consultation during bodily injury assessment of people who have suffered some type of violent event. It may be most common in assessment of traffic accident injuries but it is also cited in cases of assault.

Most of the processes that lead to painful shoulder pathology are in the subacromial joint. In this case the main source of pain is rotator cuff pathology. Within this structure it is specifically supraspinatus muscle pathology which generates the largest proportion of painful conditions and complaints.

Shoulder pain is a symptom that a significantly large number of people suffer from naturally. Hence the reason for the controversy is how to decide when the pathology of these structures is attributable to or independent of the violent event reported so as to include it in or discard it from the symptoms and assessment of bodily injury.

To understand the pathology at hand we need to review the anatomy of the area due to its influence on the origin of the problem. In order to assess the relationship of the lesion with the violent pathology it is also essential to review the pathogenesis and types of injury that we may come across. Once the root of the problem has been located we then need to evaluate its forensic medical aspects, beginning with an analysis of the mechanism of injury.

2.2 Anatomy

The shoulder is a complex joint which is actually made up of a number of joints: glenohumeral, acromioclavicular, sternoclavicular, scapulothoracic and subacromial.

Rather than strictly speaking being a joint, the subacromial joint is a corridor for tendon structures. The upper part of this space or corridor consists of the coracoacromial arch, built in turn by the coracoid apophysis, the acromion-coracoid ligament and the acromion. The corridor is bordered at the bottom by the upper side of the humeral head, specifically the greater tuberosity.

The subacromial joint or space acts as an anatomical corridor for a set of tendons which run from various regions of the scapula to join the proximal humerus; these tendons are called the *rotator cuff* and consist of the supraspinatus tendon, the infraspinatus muscle tendon, the subscapularis muscle tendon and the teres minor muscle tendon.

Of these four it is the supraspinatus muscle tendon that has most of its route within the subacromial corridor.

Another element that occupies this subacromial space is the subacromial bursa, which is above the supraspinatus tendon and acts as a buffer structure.

2.3 Pathogenesis of the injury

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The main role of the rotator cuff is abduction of the shoulder, accompanying the action of the deltoids; specifically the supraspinatus is needed to begin abduction.

When the shoulder is in abduction between 60° and 120°, the supraspinatus muscle tendon is compressed against the top of the space (consisting of the coracoid apophysis and acromion); in this position the supraspinatus tendon maintains the coaptation of the glenohumeral joint while it prevents the greater tuberosity impacting the acromion (an effect due to the abductor action of the deltoids) by interposing itself between the two structures. As a result it is also compressed caudally against the greater tuberosity.

Thus with shoulder abduction from 60° onwards the supraspinatus has to work in pretty unfavourable conditions as a result of compression and friction against the bony structures surrounding it, a phenomenon known as impingement. The context in which the injury occurs is the development of stresses when the supraspinatus tendon is chronically compressed against the bony structures and this is the reason why this is the anatomical structure most related with painful shoulder pathology.

Under normal conditions the subacromial bursa acts as a buffer, preventing the direct compression of the tendon against the roof of the joint, but it is usually not enough to avoid the appearance of lesions on the tendon. Thus supraspinatus tendon injury may be accompanied or not by subacromial bursitis. The extension of the damage due to involvement of the other structures in the cuff occurs as the compromise of the subacromial space becomes more chronic over time.

The influence of two other factors has to be considered when understanding the pathogenesis of the injury: the first is the result of a problem in healing of the microlesions that have developed due to friction, a problem in turn derived from the fact that the supraspinatus tendon has an area deficient in irrigation which interferes with tissue repair and healing mechanisms. Furthermore, when a tendon injury occurs in proximity to the insertion, this injury is subjected to tensile forces in opposite directions from the three remaining cuff muscles and the weight of the arm. This traction negatively interferes in the progress of healing, generating very weak scarring areas. This is the reason why damage to the tendon occurs cumulatively.

The second factor that directly affects the pathogenesis of the lesion is the morphology of the acromion, specifically its underside. Acromial morphology can be altered as a result of the development of osteoarthritis phenomena in the form of osteophytes. In addition studies of cadavers [3] have described three morphological variants in the articular facet of the acromion:

Type I: flat acromion

Type II: curved acromion, with the concavity directed towards the subacromial space

Type III: hooked acromion

The interest of these morphologies lies in the compromise of the subacromial space (thus increasing the compression on the tendon) that each of them can generate. Specifically it is the type III morphology which has the greatest relation with supraspinatus muscle pathology.

2.4 Types of injuries

The involvement of the supraspinatus tendon due to impingement of the subacromial space can occur in two ways: by developing tendonitis or as rupture.

Supraspinatus tendinitis, as its name suggests, is an inflammation of the tendon. Tendinitis evolves in three phases; the first with the onset of acute inflammation, primarily in the form of oedema, the second when haemorrhaging appears in the body of the tendon in the context of the production of micro-ruptures and outbreaks of necrosis, and the third of tissue repair in the form of fibrosis and scarring, phenomena which materialise as degenerative changes of the tendon.

Rupture is usually the end result of chronic tendonitis, i.e. a tendon affected by degenerative changes, and albeit less frequently can be acute. Tendon ruptures may be complete or partial. Chronic complete rupture is accompanied by retraction of the tendon ends together with fat atrophy and degeneration of the muscle belly due to the loss of muscle activity resulting from lack of use.

When rupture is acute we would expect to find a variable degree of oedema and fluid in the subacromial space but we would not observe fat atrophy or degeneration of the muscle belly.

2.5 Mechanism of injury

Based on the above, we need to distinguish between two types of patients when we have to analyse the mechanism of injury depending on the presence or absence of signs of degeneration of the tendons. This means we have to look at the epidemiology of the symptoms.

In general, signs of degeneration of these tendons usually appear around age 40; they are severely weakened tendons due to repeated microtrauma over time which furthermore, and due to the reasons described above, has not healed well.

Degenerative disease typically occurs more often in men, usually in the age range between 30 and 50. As a degenerative disease and linked to the use of the arm, it is an injury of the dominant arm and hence is more common in the right arm. How much this joint is used is closely related to the type of job the person does. Thus occupations involving use of shoulder abduction (without the support of the arm) or strength against resistance with slight shoulder abduction and those where weights have to be lifted above shoulder level are particularly prone to this disease.

In such patients with an underlying pathology of the structure, the most likely injury is partial or complete rupture of the tendon. Hence the presence of degenerative disease constitutes a previous condition.

Tendon rupture is not common in young people with intact muscle and tendon structures. Given that the tendon is not damaged in such patients, the most common injury is cortical avulsion in the tendon insertion area (cortical avulsion in the greater tuberosity).

Hence only tendon rupture can be considered as injury from the medico-legal standpoint. Tendinitis is an underlying pathology and thus constitutes a previous condition. When this is the case, thorough analysis of the issues described will be required in order to assess whether this previous condition has had any influence on the development of the injury.

As for analysis of the mechanism of injury, the first thing to bear in mind is that acute, traumatic injury of the rotator cuff (and specifically of the supraspinatus tendon) requires direct impact against the shoulder. This impact has to occur with sufficient and generally high force and hence tends to be associated with other injuries in the same region, for example accompanying scapular-humeral dislocation. In this context tendon rupture can occur in both patients who have a degenerative disease and ones who do not.

Acute, traumatic and isolated rupture (not accompanied by other lesions of the shoulder joint) is a form of injury found in patients who already present degenerative tendon pathology and is therefore affected by the existence of the underlying pathology, by the weakening of the tendon structure. In these cases, direct moderate-to-high impact on the shoulder or a sudden movement when the shoulder is in abduction or antepulsion, i.e., in the range of motion when the supraspinatus tendon is compressed against the acromion, is required.

Another possible rupture mechanism could be falling with support from the hand (with the arm extended) or on the elbow where the

humerus head is projected vertically, hitting the acromion and pinching the supraspinatus tendon. However, rupture under these conditions requires an underlying degenerative process in the tendon.

2.6. Diagnostics and physical examination

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Tendinitis is characterised essentially by the presence of shoulder pain.

It is a pain on the outer side and can radiate to the trapezius; the pain intensifies with abduction of the shoulder from 60° onwards. The pain caused in this position is due to restriction of active shoulder mobility and passive mobility shows there is no obstacle to performing the full range of motion.

In case of tendon rupture there are more clinical signs; it may start with a feeling of tendon tearing, and in cases of complete rupture complete and absolute functional limitation appears immediately which subsequently evolves to a limitation only in abduction. Functional limitation is associated with spontaneous pain, pain on active mobilisation and mobilisation against resistance.

If the rupture is incomplete, pain associated with abduction of the shoulder is the main sign.

The main means for diagnostic confirmation is examination of the patient by imaging.

X-raying the shoulder shows the state of the acromion (morphology, presence of osteophytes) and the presence of calcifications in the structures contained in the joint and makes it possible to measure the degree of collapse of the acromiohumeral space.

Ultrasound will enable us to assess the condition of the tendon and muscle belly. It is a good method in the case of large ruptures and complete rupture of the tendon. In these cases it is not possible to see the tendon due to retraction of the muscle mass.

Magnetic resonance imaging is the solution of choice for the study of rotator cuff pathology. Three factors should be evaluated in the study of this disease: the state of the acromioclavicular corridor, the state of the acromion and the state of the tendon structures. The influence of the acromioclavicular corridor has to do with the shape or state of its articular facets, whether they are smooth or show signs of degenerative joint disease (presence of osteophytes, subchondral oedema, capsular hypertrophy), and also with the degree of compromise of the joint space itself. Two factors are assessed for the influence of the acromion: the morphology of its lower side (smooth, curved or hooked) depending on the degree of compromise of the space it creates and its inclination.

As for the examination of tendon structures, a distinction has to be drawn between signs of degenerative tendinopathy (called *tendi-*

nosis) and signs of rupture. Signs of tendinosis are alterations in the tendon signal (according to the sequence), thickening of the structure and irregularities in the contour of the tendon. The signs of rupture that primarily need to be identified are those forming complete rupture which are changes in the tendon signal, tendon retraction, atrophy and fatty degeneration of the muscle belly and the presence of fluid in the subacromial bursa.

2.7 Treatment and its medico-legal assessment

Treatment for inflammatory processes involves the prescription of anti-inflammatory drugs and establishing a pattern of rehabilitation. Generally it means the administration of non-steroidal anti-inflammatory drugs, although steroidal anti-inflammatory drugs and anaesthetics can be administered locally by infiltration. The importance of posture in the treatment of these injuries should be considered and positions that facilitate compression of the tendon should be eliminated or changed. However, while these measures reduce pain and the intensity of the underlying inflammatory process they do not help to restore the tendon.

Partial ruptures should be approached conservatively using treatment with anti-inflammatory drugs and rehabilitation to treat the pain and injecting the subacromial space to maintain the mobility of the joint.

If the situation does not improve in spite of this conservative treatment, surgery can then be justified. This involves decompressing the space, filing the bone structure and removing the acromioclavicular ligament, and then cleaning the lesion focus and suturing it. Functional rehabilitation will be required later on.

Surgery is used in the case of complete rupture after a period of conservative treatment (analgesic). The procedure involves suturing the tendon ends and cross-bone tendon reinsertion. A programme of functional rehabilitation is also essential.

The aim in the case of surgery is obviously to recover the functional capacity of the tendon.

In the case of claims for inflammatory or degenerative tendon processes (tendinitis), since this entity cannot be considered the outcome of a single traumatic event (traffic accident, assault, etc.) any treatment prescribed should not be considered as a forensic medicine injury assessment opinion.

When there has been tendon rupture (either whole or partial) as a result of the traumatic event and as long as the criteria for causation between the two (rupture and event) are met, medical intervention is considered essential for healing the injury and therefore the corresponding medico-legal description would be *medical or surgical treatment*.

If the claim is for supraspinatus tendon rupture but the criteria of causality are not met (the alleged mechanism of injury does not match the injury or radiology confirms that it is a chronic rupture), the pathology should not be considered as an injury from the medico-legal standpoint and therefore any treatment indicated should also not be considered from the medico-legal standpoint.

2.8 Forensic medicine issues

Thus when we see a patient claiming injury to these structures, we need to look at a number of factors in order to establish the causal relationship between the alleged injury and the event to which is attributed.

Hence the first thing to be considered is what type of injury they report they have presented: tendinitis, tendinosis or rupture. Attention should be paid to the presence of injuries associated with the same shoulder. Radiological examination, especially magnetic resonance imaging, is highly significant when examining the type of injury claimed as it makes it possible to objectify the presence of signs related to chronic tendon rupture (muscle retraction, muscle atrophy and fatty degeneration).

Secondly, we need to analyse the patient from the epidemiological point of view (age, sex, type of work) in order to identify whether they are in the population group at risk of presenting an underlying pathology in shoulder structure tendonitis.

Once the patient's symptoms have been identified, next comes analysis of the mechanism of injury, i.e. the way in which the patient says these injury symptoms came about:

Has there been a direct blow on the shoulder joint or not?

If there has been a direct blow, what type of blow was it, or in other words how strong was it?

If there has been a direct blow, what position was the shoulder in when it received the blow?

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3. Previous Condition and Spine Pathology

Dr. Antonia Bertomeu Ruíz and Dr. Dolors Giménez Pérez

3.1 Introduction

Degenerative disease of the spine is one of the most common in the field of medico-legal expertise and also one of the most problematic. Assessing the previous condition of the spinal column and its influence on the final situation of the individual is one of the most controversial issues in establishing causation between trauma and the injuries diagnosed.

The spine is made up of different bones and ligaments and contains and protects the central nervous system and the nerve roots of the peripheral nervous system form. Despite the importance of all these structures, it seems fair to say that in the field of legal medicine the intervertebral disc of the spine is the most contentious in terms of assessment. For this reason we will focus primarily on this structure. Thus we will describe the most common biomechanics of injury focusing on intervertebral disc injuries and endeavour to discover how far trauma can trigger herniated disc pathology. However, we will not ignore other conflictive pathologies such as discal protrusions, spondylolysis, spondylolisthesis and Schmörl nodes or hernias.

3.2 Concept of disc herniation and mechanism of injury

We define a herniated disc as the emergence of the nucleus pulposus through the annulus fibrosus of the intervertebral disc. Disc herniation occurs in most cases in spines with signs of disc degeneration.

The aetiology of disc herniation involves metabolic, biochemical, genetic, psychosocial, age (articular degeneration or ageing) and anatomical factors. Mechanical factors also play an important role and include the following:

- Microtrauma
- Repetitive flexion-extension of the trunk mechanisms when carrying a lot of weight (jobs that require major physical exertion). Continued pressure on the disc causes it to deteriorate.
- Ongoing rotational motion over time (shear effect).
- Excess weight and body size.
- Atrophy of paraspinal muscles.

The currently most accepted aetiology is multi-factorial and its emergence is due to a combination of the above factors.

The typical mechanism for producing a herniated disc consists of three factors:

1. Trunk flexion: the disc space opens backwards.
2. Increased load: the nucleus pulposus which has moved backwards might be trapped on the posterior edges of the vertebral bodies
3. Raising the trunk while maintaining the load: the disc material is narrowed and forced out backwards breaking the remaining intact fibres of the annulus fibrosus.

3.2.1 Epidemiology

Some studies have shown that at the age of 50, 85% to 95% of adults present evidence of disc degeneration at autopsy. Others find that between 20% and 28% of asymptomatic patients have hernias and most present degenerative disc pathology. As for posttraumatic origin, the prior traumatic event as a trigger has only been definitively demonstrated in large herniated disc surgical series in figures ranging from 0.2% to 2%.

Cervical discopathy. The cervical spine is designed to give greater mobility to the head and support little weight. Hence the most common cause of disc pathology in the cervical spine is degenerative.

Lateral disc protrusion is more common in young people, with or without traumatic history, with the C7 root being most involved. Most herniated discs occur between C5 and T1 and are typically unilateral.

Whiplash as a cause of hernia is very rare and a recent Aso Ascario study found that out of a total of 1574 instances of whiplash, only 5 (0.31%) could be described as posttraumatic hernias. Posttraumatic hernia in the lower back is even less common after an accident.

Studies of asymptomatic subjects found 20% of people aged 45 to 54 had cervical protrusions or herniations and this figure rises to 57% in people aged over 64.

Thoracic discopathy. thoracic hernias are rare compared to cervical or lumbar ones. The incidence is estimated at from 0.25 to 1% of all herniated discs. Population incidence is about 1 per million patients per year. Most thoracic hernias are central or centre-side. Many are calcified.

Most thoracic herniated discs are presented during the 3rd to 5th decades and are largely below the T7 level. Several studies suggest that approximately 25% of these hernias are associated with traumatic injury. However, there is controversy about the importance of trauma as a cause of thoracic disc herniation. There are studies where a traumatic antecedent appears in 14% to 63% of patients. The average prevalence in 10 random series is 34%. In some patients the causal relationship is undeniable while in others the trauma may be an aggravating factor and simply coincidental. The degree of trauma described responsible for herniation varies from slight deformations in torsion and chiropractic manipulation to serious falls or traffic accidents.

Lumbar discopathy. Lumbar disc herniation is more common between L5-S1. Other places in order of frequency are L4-L5, L3-L4, L2-L3. The most frequent trauma cause is bending.

Hernias occur most frequently due to degeneration of the posterior longitudinal ligament and the annulus fibrosus. This degeneration is common in adults over the age of 35 and can lead to chronic low back pain.

There are some studies which conclude that a herniated disc is not the result of a single trauma and it is currently thought that pure traumatic herniated discs (with no disc degeneration) are highly unusual. In fact, a study by Matsumoto in 1998 of 497 volunteers came to the following conclusions:

- He found disc degeneration in 17% of men and 12% of women aged 20 and over.
- Aged 60 and over there is degeneration in more than 85% of cases with protrusions in more than 70% of them.
- 50% of people have disc protrusions.
- 30% of people have asymptomatic herniated discs.

3.2.2. Clinical signs and additional diagnostic examination

There are some clinical data to guide the diagnosis of an acute herniated disc compared to one caused by degeneration.

Thus *acute* hernias are usually more common in young male subjects and present radiculalgia immediately or a very short time after the trauma. Symptoms are usually monoradicular and the Dejerine sign is usually positive (pain increases with the Valsalva manoeuvre). It usually affects a single level (examination objectifies signs of monosegmental alteration). The axial disc compression manoeuvre (Spurling manoeuvre) usually causes radiating pain.

By contrast, **degenerative** herniated discs usually occur in older people (over 30), there is frequently a previous history of lower back (or neck) pain, symptoms are usually more insidious and predominantly motor and the degeneration usually affects several levels (pluriradicular or bilateral involvement clinical picture).

It is important to bear in mind that:

A posterior disc protrusion does not present clinical signs but a lateral foraminal one does in the shape of root clinical signs.

The following *additional tests* can be used:

1. Plain X-rays: to observe the degenerative condition and whether it applies to the entire column.
2. Electromyogram: root symptoms take at least 3-4 weeks to appear (the time required for root irritation). The age of the radiculopathy can be determined (if chronic radiculopathy is reported there is less likelihood of posttraumatic origin)

3. CT: the disc is grey, penetrating into the medullary cavity
4. MRI: radiological markers of degeneration:
 - height (lower height of the intervertebral disc)
 - composition (grey at T2 is indicative of chronic condition = dehydration)
 - other spinal condition: osteophytosis, closely degenerative of the spinal canal, lesions at different levels
 - the presence of Modic changes: they are sign changes in the bone marrow region adjacent to the vertebral endplate which seem to be indicative of the existence of chronic biomechanical disorders in the mobile intervertebral segment. They are classified into three grades:
 - I: vascularised fibrous tissue in the adjacent trabecular bone
 - II: disruption of the vertebral endplate with lipid replacement of the trabecular bone adjacent to the endplate
 - II: bone sclerosis

The early presence in MRI of II or III Modic signs after trauma would be indicative of a chronic and therefore degenerative process.

3.2.3 Diagnosis of traumatic and degenerative lesions



Schmorl node in red circle. Red arrow: D8 disc wedging. Blue arrow: D8-D9 posttraumatic hernia
Source: [7: 108-120].

Individualised assessment is required to diagnose whether a discopathy is traumatic or degenerative based on the following factors to establish causality:

- Subject's age.
- Medical history.
- Job.
- Type of trauma and biomechanics.
- Posttraumatic symptoms and evolution.
- Evaluation of results of additional tests conducted during the care process.

Thus with respect to a posttraumatic herniated spine:

1. Traumatic origin is more likely in people aged under 30.
2. The trauma must be severe: falling from a great height and frequently associated with spinal bone pathology (vertebral fractures) or more commonly hyperflexion mechanisms.
3. The clinical signs should be acute, i.e. immediately or just a few hours after the traumatic event.
4. There should be no degenerative disease in the rest of the spine found in the additional tests conducted.
5. There should be no medical history (clinical signs) for the spine.

Degenerative hernias have the following characteristics:

1. They are the most common.
2. They occur in people over 30 who suffer repetitive microtrauma in the spine.
3. The clinical signs are usually not so acute but appear some time after trauma: signs and symptoms of spinal cord compression (predominantly motor symptoms) which appear slowly and gradually.
4. Degenerative pathology of the spine is objectively found in the results of the additional tests.
5. There is usually a history of spine discomfort.

Posttraumatic herniated disc	Degenerative herniated disc
Acute beginning	Gradual beginning
Immediate neurologic involvement	Late neurologic involvement
Radiculopathy is more frequent	Myelopathy is frequent
X-rays: normal	X-rays: intervertebral disc space narrowing, osteophytes, etc.

3.2.4 Influence of the previous condition, the trauma and the herniated disc

The conflictive factors to consider are as follows:

- Did the hernia diagnosed exist before the trauma?
- Do the additional tests indicate a degenerative pathology of the spine?

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Based on the above, from an expert point of view and in the case of an individual with prior degenerative spine disease and severe spine trauma (e.g. falling from a great height) combined with subsequent diagnosis of a herniated disc and acute clinical signs of lower back pain (the most common), the prior pathological condition has facilitated/determined the appearance of this hernia. If there is no medical history indicating previous clinical signs, the medico-legal causal relationship between the trauma as the triggering event for the hernia in a prior “facilitating” context for the appearance of this hernia (previous pathological condition) has to be established and therefore the clinical signs derived from it would be the sequela of the accident.

Hence the herniated disc (the clinical sign) should be included in the healthcare period and sequela assessment in the medico-legal assessment of the bodily injury of these lesions.

One aspect to be assessed in order to attribute the herniated disc to the trauma is the severity of this trauma. For example, in whiplash in car accidents at low speed and with no other additional injuries, the existence of disc protrusions diagnosed later on are pre-existing and therefore from the medical expert standpoint there would be no causal relationship.

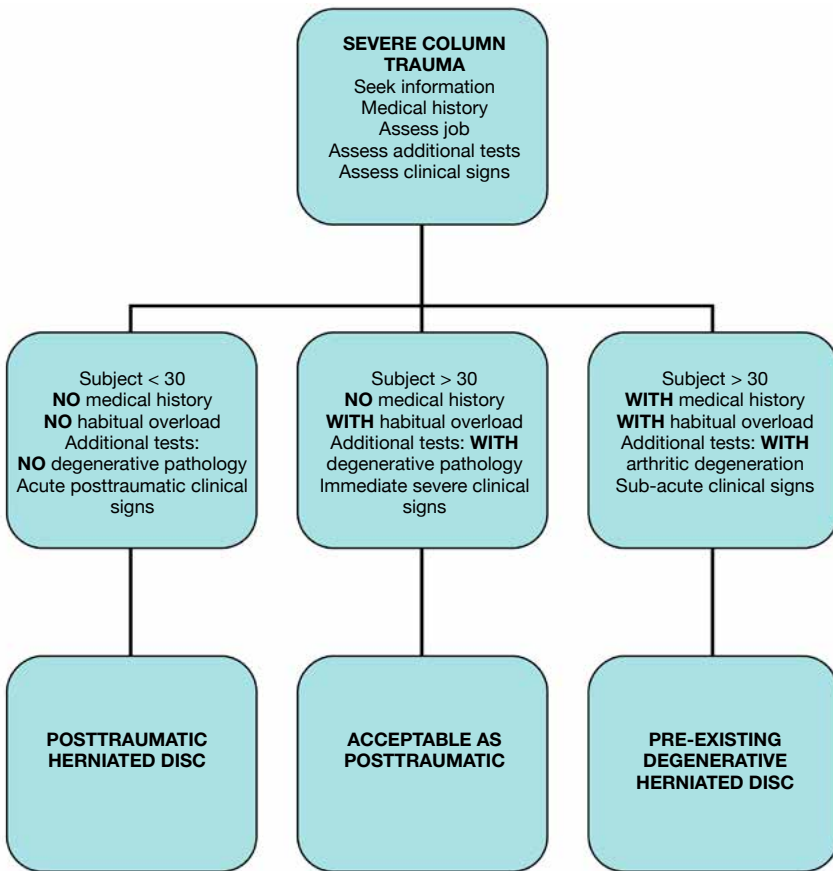
3.2.5 Medico-legal assessment

If based on the issues raised above we think that it is a posttraumatic hernia, the medico-legal assessment of injuries would be as follows:

- Medical and/or surgical treatment
- The healthcare period would include the treatment of the hernia
- Assessment of sequelae: those derived from the herniated disc
- A section featuring medico-legal considerations about the previous condition as a facilitator of the appearance of the herniated disc should be added to the report

If we consider that the hernia was pre-existing and degenerative and that the trauma has only increased or caused the clinical signs to emerge, i.e. an increase in or appearance of pain, medical-legal assessment of lesions would be:

- Basic medical attention or medical treatment depending on the evolution of the symptoms.
- Healthcare of injuries would only include the treatment of acute pain.
- Sequelae: aggravation of an earlier condition.
- If this hernia is surgical, healthcare corresponding to surgical treatment should not be assessed as the hernia would be deemed to be pre-existing.



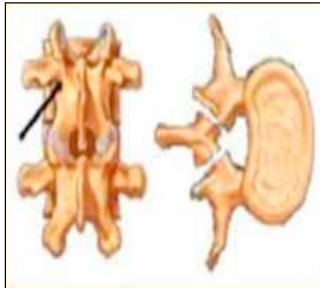
3.3 Disc protrusions

The pathophysiology of disc protrusion is the same as for hernias, as is the epidemiology, so it is a stage prior to a herniated disc. Consequently we believe that the medico-legal assessment of disc protrusions will involve the same criteria as mentioned above for herniated discs.

3.4 Spondylolysis, spondylolisthesis, Schmörl nodes

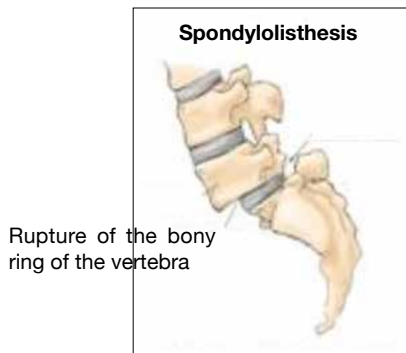
3.4.1 Spondylolysis

It literally means the dissolution or disappearance of a vertebra. In practice it is usually defined as the lack of radiographic union between two parts of a vertebra, usually at the height of the isthmus. This condition must be viewed as pre-existing and should not be confused with fractures after accidents unless these findings are in somebody who is seriously injured with fractures at other levels, in which case we would have a different pathology.



3.4.2 Spondylolisthesis

It is the movement of one vertebra relative to the vertebra immediately below. This displacement is most commonly anterior (anterolisthesis) but may also be posterior (retrolisthesis).



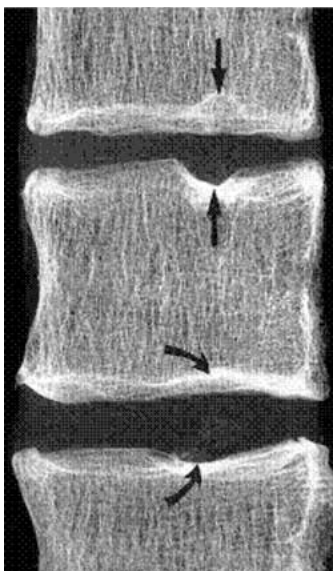
Side view of the lower spinal column

Spondylolisthesis can be classified by its aetiology (Bennett classification):

- *Type I: congenital.* It is frequently observed in adolescents and is usually associated with congenital deformities of the sacrum.
- *Type II: isthmic.* The most accepted cause of this type of spondylolysis is fracture of the isthmus bone due to microtrauma and mechanical fatigue.
- *Type III: degenerative.* It is most common at L4-L5 and L3-L4, especially in females. It is usually associated with stenosis of the canal with a syndrome of intermittent claudication.
- *Type IV: traumatic.* It is quite rare in medical practise. Some authors limit the diagnosis to cases where there has been severe trauma leading to a fracture of the pedicle but not the pars. In many cases there is spontaneous regression.
- *Type V: pathological.* Usually associated with infections or tumours.
- *Type VI: postsurgical.*

3.4.3 Schmörl nodes

They are small intervertebral disc displacements towards the interior of the vertebral body and are associated with moderate degenerative changes. They appear to be associated with factors that are anatomical in addition to being degenerative (straighter or fractured vertebral plates are more frequently associated with these Schmörl nodes or hernias). They can be found in the elderly but also in young people. There is a decrease in disc space.



3.5 References

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4. Previous Condition and Knee Cruciate Ligament Pathology

Dr. Daniel Fernández Doblás

4.1 Anatomical reminder and function.

The cruciate ligaments are dense, very solid, short and not very elastic ligaments which means when lengthened they break rapidly (they do not tear). They are the strongest attachment and bonding structures in the knee.

4.1.1 Anterior cruciate ligament (ACL)

It is the weaker of the two ligaments and begins in the anterior intercondylar area of the tibia, immediately behind the insertion of the medial meniscus. From here it follows a posterior and lateral cranial path to the posterior part of the medial side of the femoral lateral condyle. It is relaxed by bending the knee and tightens with the full extension of the joint. It prevents the anterior displacement of the tibia on the femur (or the posterior displacement of the femur on the tibia) and knee hyperextension.

4.1.2 Posterior cruciate ligament (PCL)

It begins in the back of the intercondylar area of the tibia. From there it follows an anterior and medial cranial path to the lateral side of the femoral medial condyle. It tightens when the knee is bent. It prevents the posterior displacement of the tibia on the femur (or the anterior displacement of the femur on the tibia).

4.2 Mechanism of injury

They can be damaged (partial or total rupture) both by direct trauma on the knee or by a range of movements. They may be associated with osteoligamentous and meniscus injuries depending on the mechanism and energy of the trauma or movement.

4.2.1 Anterior cruciate ligament

- Hyperextension. The ligament is broken against the intercondylar notch. Examples: missed kicked (footballers), motorcycle accidents where the rider supports the bike's weight on an extended leg.

- Valgus-flexion-external rotation. It is accompanied by lesions of the meniscus and internal collateral ligament. Examples: footballers or skiers changing direction with one foot fixed to the ground.
- Varus-flexion-internal rotation. Less frequent. Associated with external injuries. Examples: the same as in the previous paragraph.
- Posteroanterior trauma to the proximal end of the tibia.
- Posteroanterior trauma to the proximal end of the tibia (associated with PCL injury)
- Abrupt quadriceps contraction. Examples: falling after a jump when there may be an anterior subluxation of the tibia. Skier rising quickly from a position of hyperflexion.

4.2.2 Posterior cruciate ligament

- Tibial retropulsion. Generally due to trauma on the proximal end of the tibia. Examples: falling with knee impact on the floor with the ankle in plantar flexion. Head-on collision of a car in which the front passenger's knee collides with the glove compartment.
- Forced hyperflexion. Example: skier going downhill; in this case there may be forced and sustained bending exceeding the physiological limits of the ligament.
- Hyperextension. Usually accompanied by ACL injury.
- Others. The PCL can be damaged in rotations and forced valgus, always associated with lesions of other structures of the knee.

4.3. Diagnostics and examination

4.3.1. Anterior cruciate ligament

- Pain. The injured may have a subjective feeling of stiffness or something being broken. Usually the pain is very intense and relatively short-lived since as the ligament is completely broken the damaged fibres stop working.
- Haemarthrosis/haematic joint effusion. It is the main sign even though its absence does not rule out the injury.
- Joint instability.
- Functional deficit. It is secondary to the pain and joint effusion and is usually recovered on its own when they disappear.

4.3.2 Posterior cruciate ligament:

In isolated PCL ruptures clinical signs are less severe than in the case of ACL and may go unnoticed during first examination. The joint

condition may be minimal. Symptoms that may occur include pain, joint compromise and haematic effusion.

There are numerous exercises to explore cruciate ligament injuries. The main ones are:

- ACL: anterior drawer test, Lachman test, jerk test, pivot shift, Dejour test, etc.
- PCL: posterior drawer test.

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Additional tests:

- Plain X-ray: it can show bony avulsions, osteochondral lesions, partial fractures or the joint can be forced by manoeuvres such as the drawer or Lachman test.
- MRI.
- Arthroscopy.

4.4. Degenerative or occupational pathology

Almost all injuries to the cruciate ligaments occur traumatically. There are rare cases of degenerative lesions, although there are two situations in which the ACL can be injured progressively. They are as follows:

1. Chronic laxity of the internal compartment (uncommon) and mainly in cases of total meniscectomy. In these cases there may be a rupture of the ligament due to “fatigue”.
2. Osteophytosis of the intercondylar notch. The ligament is subjected to friction which progressively injures it.

Chronic ruptures have not been described for PCL.

4.5. Previous condition

4.5.1 Anterior cruciate ligament

Damage to the ACL, whether partial or total rupture, is an acute injury (except in rare cases of chronic rupture). The clinical signs appear from the outset with varying intensity. The initial injury report is very important in these cases and should state some of the signs and symptoms described. It is very rare that a definitive diagnosis of damage to the ACL can be given at the time of basic medical attention since the physical examination should be done within the first hour after the injury. In most cases the pain and loss of function prevent exploratory manoeuvres so the diagnosis will be made a few days or weeks later after the process has cooled along with performing MRI.

As it is an acute process with abundant clinical signs, an ACL injury should not pose any problems in the field of legal medicine. The only

cases in which the injury may initially go unnoticed are multiple trauma patients who are bedridden for days and where the injury is not evident until they begin to walk.

4.5.2 Posterior cruciate ligament

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Damage to the PCL is also an acute injury, but its joint involvement may be minimal in some cases which may delay diagnosis for several months. Even so, and in the same way as with the ACL, the initial injury report is extremely important as there should be some kind of sign or symptom in the knee even if it is as imprecise as a bruise/erosion on the anterior side of the knee.

4.6 Treatment

4.6.1 Anterior cruciate ligament

It has very little healing capacity. Treatment should be individualised for each patient depending on age, sedentary lifestyle, sports, associated injuries, etc.

- Conservative: immobilisation and subsequent functional rehabilitation.
- Surgery (the most frequent option): replacing the injured ligament by tendon structures, cadaver cruciate ligament or synthetic tissue.

4.6.2 Posterior cruciate ligament

It has greater healing capacity than the ACL. The therapeutic option also will be assessed for each particular case.

- Conservative: in isolated PCL injuries with little joint involvement, immobilisation and subsequent functional rehabilitation will be the treatment of choice.
- Surgery: the same as for ACL.

4.7 Sequelae

The cruciate ligament sequelae are found in the knee section in the ligament injuries section:

- Cruciate ligaments (operated or not) with symptoms (1-15)

After a cruciate ligament injury, and depending on the lesion associations, there may be pain, functional impairment of the knee (laxity, instability, limited mobility, etc.), posttraumatic arthritis (if there is bone involvement), etc.

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5. Previous Condition and Collateral Knee Ligament Pathology

Dr. Juan Antonio González García

5.1 Anatomical and functional reminder

Lateral collateral ligament: it originates from the lateral femoral epicondyle and inserts in the fibular head. It is tight in extension and relaxes as the knee bends. It provides knee stability by preventing lateral outward movement of the joint.

Medial collateral ligament: it originates from the medial femoral epicondyle and inserts in the medial condyle of the tibia. It is tight in extension and relaxes as the knee bends. It provides knee stability by preventing lateral movement towards the inside of the joint.

5.2 Mechanism of injury

Either by direct blows to the knee or by rotation of the body with the foot fixed.

The LCL is injured due to pressure from the inside of the knee, causing greater strain on the outside of the joint (varus pressure).

When there are mechanisms associated with tibial varus-internal rotation, for example when falling forward forcing the knee into the varus position, injury to the lateral ligament is associated with cruciate ligaments and/or menisci.

The MCL is injured due to pressure from the outside of the knee, causing greater strain on the inside of the joint (valgus pressure).

When there are mechanisms associated with tibial valgus-external rotation, for example when the foot remains fixed to the ground while the leg turns outwards, injury to the MCL is associated with injury to the medial meniscus and/or cruciate ligaments.

5.3 Diagnostics and clinical examination of lateral and medial ligament lesions

Pain: immediate of variable intensity, involving the outer or inner side of the knee when it moves or resting on the foot. It can be explored by pressing the involved ligament with the knee bent at 30° to relax the cruciate ligaments and the posterior capsule. Laxity in full extension implies an associated injury of the cruciate ligaments and posterior capsule.

Inflammation or oedema: proportional to the damage to the ligament.

Stability of the knee: in case of partial rupture, forcing the knee to the opposite side makes it become taut. That way varus stress can be applied to explore the lateral collateral ligament and valgus stress to explore the medial collateral ligament.

Instability of the knee: in case of total rupture, presenting joint opening, forcing the knee to the opposite side, it does not become taut. Thus if varus stress is applied to explore the lateral collateral ligament or valgus stress to explore the medial collateral ligament and the joint opening is greater than in the other knee, there will be a ligament rupture that may be:

- Grade I: opening < 5 mm
- Grade II: opening from 5-10 mm
- Grade III: opening > 10 mm

Hematoma: frequent in the days following the injury

JOINT INVOLVEMENT: ruptures of the lateral or medial ligaments do not present with haemarthrosis although there may be some joint effusion.

5.4 Additional diagnostic examinations

MRI.

5.5 Degenerative or occupational pathology

The collateral ligaments of the knee present little degenerative pathology and traumatic pathology occurs in direct blows such as traffic

accidents or sports (hockey, football, etc.) either due to twisting when jumping or falling when doing sports such as skiing, basketball or football.

5.6 Previous condition

Strains of the collateral ligaments, when properly treated and even in the most severe cases of total rupture, do not tend to lead to instability or limit physical activity once healed and do not result in osteoarthritis. Hence there are no problems in assessing the previous condition.

Pellegrini-Stieda disease or calcification of the medial collateral ligament of the knee: when this ligament is injured it may have a tendency to accumulate calcium inside it after direct trauma that produces a haematoma. This finding during the first weeks would imply trauma prior to the current injury.

5.7 Treatment

The treatment of sprains of the lateral ligaments is usually conservative, i.e., immobilisation of the knee with an elastic bandage or brace with lateral reinforcements in case of zero knee instability.

An articulated knee brace should be used in moderate or major instabilities, avoiding if possible plaster which will produce stiffness for longer.

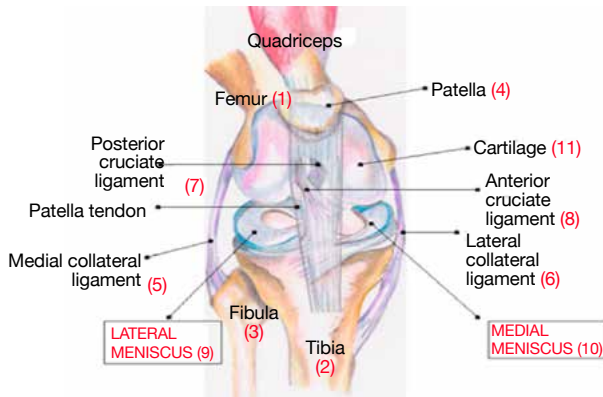
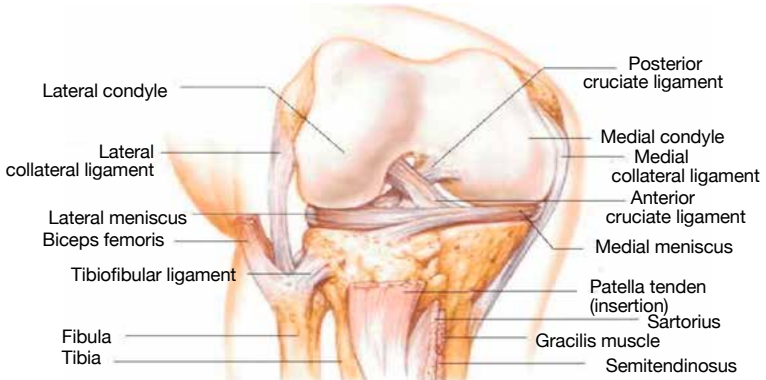
Surgery will be required only in cases of total rupture and extreme instability or when there has been avulsion of the proximal or distal inserts.

Rehabilitation will be required after immobilisation.

The medico-legal consideration will be medical treatment or medical-surgical treatment if they have been operated on. Only minimal strains which do not need rehabilitation should be considered basic attention.

5.8 Sequelae

Injuries to the collateral ligaments if treated properly do not lead to sequelae.



6. Previous Condition and Meniscal Pathology

Dr. Carmen Avila Molina

6.1 Anatomy of the knee

The knee is formed by the confluence of the femoral condyles, tibial plateau and patella: this means it is a joint with complicated mobility.

The knee also has great stability due to a complex system involving ligaments, joint capsule, menisci and musculotendinous structures.

The meniscus is a curved fibrocartilaginous lamina. There are two laminae, each situated on the tibial plateau:

- The inner or middle in a wider “C”.
- The external or lateral in a smaller “U” shape.

The meniscus’s functions are:

- Cushion the friction between the femoral condyles and tibial plateau

- Stabilise by accommodating articular surfaces
- Distribute synovial fluid forces.

6.2 Mechanism of injury

Rupture can occur in a previously healthy meniscus (traumatic rupture) or menisci where their structure has previously degenerated (tears as a complication in the process of meniscal degeneration).

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Pure traumatic ruptures are observed in young active adults, especially athletes.

Ruptures due to minimum trauma on a degenerated meniscus are found in adults over 45-50 suffering a progressive process of knee osteoarthritis. In other cases, meniscal degeneration that predisposes to rupture is the consequence of some previous ligamentous injury not treated properly, as often happens in ACL tears.

It is usually an indirect trauma to the knee; abrupt, fast, almost instantaneous and generally violent. The knee directly supports all the severe pressure of the trauma. In all circumstances in which the meniscus can be injured, the femoral condyle rotates at high speed, moves anteroposteriorly and rotates outward or inward, exerting violent compression on the internal or external tibial plateau and thus catching or pulling the meniscus between the articular surfaces.

Situations in which the injury may occur:

1. Semi-flexed knee; strongly supports the weight of the body with the leg in valgus (X) and external rotation; foot fixed on the ground. It is one of the most frequent mechanisms in football players.
2. Semi-flexed knees, carrying the weight of the body, with the leg in varus (O) and internal rotation.
3. Knees in hyperflexion and supporting the entire weight of the body; a sudden extension of the joint occurs. This is the case of a person who is squatting down and suddenly straightens up, supporting themselves on the foot fixed on the ground and usually with their leg in a valgus position: they generally tend to be elderly women who when in this position suddenly straighten up, supporting themselves on the foot fixed on the ground.
4. Knee in forced and violent hyperextension. "Air kick."

6.3 Type of injuries

1. *Vertical longitudinal ruptures*: known as a "bucket handle"
2. *Primary horizontal ruptures*: typical degenerative rupture extending from the end of the free edge and horizontally into the meniscus. The injury occurs due to a minimal traumatic event that often goes unnoticed. It does not always cause clinical diagnostics and in systematic

studies of autopsy material this injury has been shown in almost half of the population aged over 50.

3. *Radial vertical rupture (transverse or oblique)* perpendicular to the free edge of the meniscus. Other secondary ruptures may also be present in each of these ruptures causing flap or Mamelli detachment.

Finally, some compromises will be classified as:

4. *Miscellaneous*: composite ruptures, complete or partial degeneration, associated tears, discoid (congenital), fraying, etc.

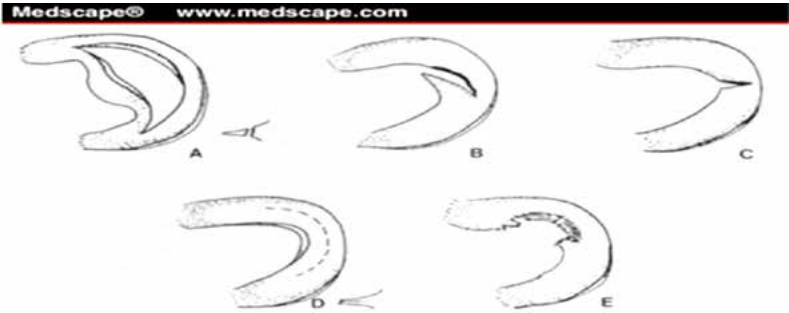


Diagram of meniscal lesion models: (A) Vertical or longitudinal (basket handle), (B) Flap or oblique, (C) Radial or transverse, (D) Horizontal, (E) Complex degenerative.

Meniscal ruptures can be located in the anterior, middle and posterior thirds of each meniscus. Tears at the level of insertion into the joint capsule are often associated with other capsular-ligamentous injuries.

The medial meniscus is the most frequently injured in the case of pure traumatic ruptures. The type of rupture varies: a longitudinal tear along the meniscus body (bucket handle); detachment of the meniscus in part or all of its insertion into the joint capsule; oblique tear on the meniscus body separating a tab on the inner edge; transverse rupture where the meniscus is divided into two; irregular rupture in which the meniscus is virtually crushed by the compression of the femoral condyle. Most of these lesions are located in the posterior third.

In the lateral meniscus the most common injury in the case of pure trauma is a cross tear and almost always in the middle third of the meniscal body. Radial ruptures of the posterior horn of the lateral meniscus are associated with ACL tears.

6.4 Symptoms

6.4.1 Acute symptoms

There is an anamnestic history. It starts with an intense, sharp pain, sometimes tearing, and noticing a crack or click. It presents functional

limitation, usually with the knee locked in flexion. Claudication. There may be joint effusion. If this occurs quickly, consideration should be given to haemarthrosis due to tearing of the meniscus in its capsular insertion. More often pain is noticed in the side of the knee for the torn meniscus when the patient twists the knee.

6.4.2 Chronic symptoms

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Weeks or months later there will be a series of varied symptoms that hinder interpretation:

There is a history of knee injury: pain, imprecise, not very severe, and discomfort that is hard to place. Insecurity in the use of knee and a feeling of instability. Joint effusion is occasional; feeling of a foreign body in the joint. Occasional clicks.

All these symptoms are maintained indefinitely.

6.5 Diagnosis

6.5.1 Physical examination:

Apley test: the patient lays prone and flexes their knee to a 90° angle, rotating the leg outwards and inwards with simultaneous axial compression against the knee from the leg and foot. There is pain in the internal and external articular interline depending on which meniscus is injured.

McMurray test: the patient lays supine and flexes their knee to a 90° angle, rotation of the leg on the joint inwards and outwards, gradually extending the knee. Pain appears in the articular interline for the injured meniscus.

Steinman test: when the knee is bent there is pain that moves backwards and forwards when the joint is extended.

Additional tests: plain X-rays make it possible to rule out other injuries. NMR will be used to assess the status of the meniscus. Arthroscopy is exploratory and therapeutic.

6.5.2 Treatment:

This may be:

- Conservative or alternatively
- Surgical arthroscopy for repair/suture or meniscal resection or transplantation.

Rehabilitation treatment will then be required.

6.6 Considerations

The meniscus in a person aged 45-50 and over will present meniscal degeneration. Major trauma is not required to produce large lesions and even articular overload can generate it.

Previous osteoarthritis may also affect the appearance of meniscal degeneration. An anterior cruciate ligament that is not treated properly will aggravate the arthritic process and meniscal degeneration at the same time.

Given a previous condition of degeneration, there is trauma to the knee that causes a meniscal injury, in which case we can consider the situation as aggravation of the previous condition caused by the accident. We may also find that the meniscus was already torn and the symptoms were minimal prior to the accident. This is the case in people aged over 50 and they are usually horizontal fractures or tears. In these cases MRI may help if it tells us the type of injury.

In young people severe trauma is required to produce injury; the meniscus is previously healthy and clinical signs will be immediate. There is a direct cause-effect relationship. If the clinical signs indicating it are chronic, the mechanism of injury is very important to see if it is possible to establish causation, although chronic symptoms usually follow acute ones.

6.7 Medico-legal description

Medical and rehabilitative treatment will always be considered when no invasive techniques have been necessary. If this is the case, medical and surgical treatment will be considered.

Healthcare is assessed as follows:

Admission: 1-3 days (although nowadays this surgery is being done in many cases without admission).

Approximately two weeks of rehabilitation (immediate start after surgery).

Lost work days: lost work days will be those required until crutches are no longer needed (about when rehabilitation is completed). About 20 further days are then required when only doing light work is recommended and work involving greater exertion should not be done for 38 days.

6.8 Sequelae

Likely sequelae are as follows:

Sequelae of meniscal lesions (operated or not) with symptoms: 1-5 points

Chapter IV

Assessment of Diagnoses Which Are Added During the Healing Time of an Injured Person. Causation

Authors: Dr. Jesús Gasque López*, Dr. Esther Amorós Galitó and Dr. Montserrat Pedrico Serradell

Often a medical forensic expert finds that the diagnoses in the report on basic attention in A&E are changed or new ones added. Sometimes this happens while the injured person is being monitored by forensic medicine, but on other occasions visits are begun subsequently when the entire process has stabilised/healed.

The medical forensic expert's role should be to consider in each particular case and at each particular time what the roles of the contributing causes, complications and individual factors are in each case.

To provide a proper assessment of these premises we need to review the concepts we will use:

- The *cause* is the necessary and sufficient condition to produce the damage, i.e. it is necessary and can produce the damage by itself, while
- The *contributing cause* is a necessary but not sufficient condition, i.e. by itself it cannot cause the damage. It has to be possible to establish the “quantum” or proportion/importance of the contributing causes to the outcome and whether or not the causal link has been broken when making the assessment.

Contributing causes are classified by time:

- *Pre-existent or precedent*: also called previous condition or health status prior to the injurious act. To be considered as such it must meet two conditions: be prior to the injurious act and not known by the injurious agent. The role of these contributing causes may be to extend the healing process (e.g. osteoporosis) or mean that the injury aggravates the previous pathological state (e.g. osteoarthritis).
- *Simultaneous or concurrent*: coincidence of two events or circumstances that influence the production of injury and increase its final

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importance (e.g. sharp force trauma with a contaminated object causing the wound to become infected and which together with a lack of medical care leads to tetanus and death).

- *Subsequent or consecutive*: events or circumstances that influence the damage once it has been produced but before final stabilisation. They are not the evolutionary complications of the injury but rather other causes or injurious agents that interfere with the course of the initial injury (e.g. medical malpractice in treating the initial injury, a new accident sustained by the injured person or a hospital infection).

- *Complications* which are additional medical problems which develop during the course of an illness or injury or after a procedure and/or treatment. In general complications are directly or indirectly related to a procedure, known as *risk of the procedure*, to a treatment known as *side effects or toxicity*, or occur in the course of an illness as part of its natural history or clinical course.

Hence complications may result from the underlying disease or initial injury, such as the onset of osteoarthritis in the final course of a fracture. However, they can also arise as a difficulty in or after surgery; an example would be surgical infection after surgery or a procedure.

From the healthcare perspective the problem of medical complications is whether you can predict their onset or the extent to which you can prevent their onset and development. However, from the medico-legal standpoint the problems are firstly to establish a causal link between the complication and the initial injury and between the complication and the final outcome, and secondly to assess the fact that complications may lengthen the healing period and/or change the criminal definition of the injuries.

When assessing *causality* we can follow the traditional *criteria* which are:

- *Topographic*: of concordance, which is based on establishing a relationship between the area affected by the violence and the one where the lesion has appeared, bearing in mind that there are mechanisms of injury that produce distant injuries.

For example: if a person suffers a fracture of the right wrist and their right arm is immobilised, at certain ages it is relatively likely that there will be limited mobility of the right shoulder when the immobilisation is removed, thus establishing a causal link. By contrast if the limitation occurs on the left shoulder there is no relationship.

Conversely, if the same event occurred in the legs any secondary pathology that appeared in the uninjured leg might be due to overload and therefore does meet the criteria of causality.

- *Chronologic*: it relates the time of violence with the time of onset of the damage or injury. It may be a simple relationship when the time is the same or the complication may occur after a while because certain pathological processes have an incubation or latency period before they emerge.
- For instance, and continuing with the previous case, shoulder mobility limitations or aches due to overload of the uninjured lower extremity appear when the initial injury has already been evolving for a number of days. Another example is the case of an injured person who has been resting in the supine position during the healing period and after a few days presents symptoms of thromboembolism.
- *Quantitative*: it takes into consideration the intensity of the violence or trauma with the severity of the injury caused. This relation can vary depending on the type of injurious agent, the nature of the injury and the length of the latency period. In theory, the greater severity of the initial symptoms means that complications are more likely to arise but it also means greater attention is paid to monitoring all the evolution factors. However, sometimes the small size of the initial injury means reduced monitoring of evolution and the beginning of complications goes unnoticed.
- *Symptomatic or pathogenic or anatomoclinical correlation continuity*: there must be some bridge symptoms between the time of trauma or action of the injurious agent and the resulting injury over time. The absence of bridge symptoms can be enough to exclude causation, bearing in mind that certain pathological processes have an incubation or latency period before they emerge. In any case there must be a possible pathophysiological explanation.
- *Exclusion*: every other possible cause of the injury or damage must be excluded. For this exclusion to be total the other cause must be complete and exclusive.

Furthermore, proper practice means also evaluating:

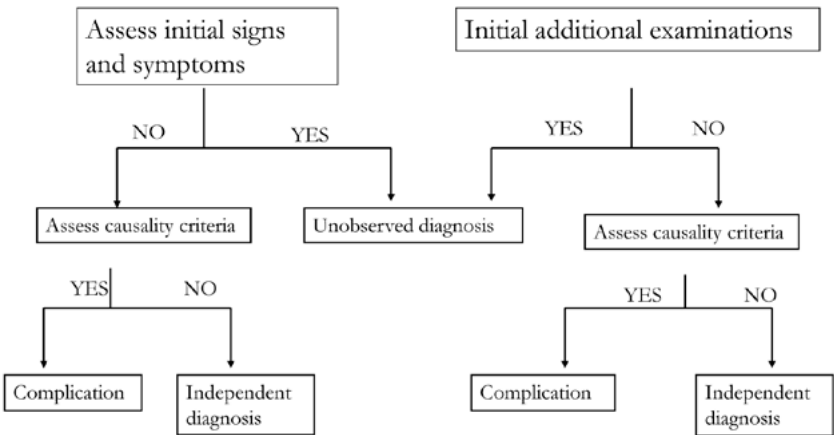
- Criterion of aetiology or reality of the trauma
- Criterion of prior integrity or absence of the previous condition.

Having clarified the different terms and concepts to be considered, we can *classify the various cases/situations that may occur*:

1. Diagnoses that are added to the initial diagnosis
2. Diagnoses that amend the initial diagnosis
3. Complications per se
4. Aggravation of the previous state
5. Concurrent contributing cause
6. Intercurrent contributing cause

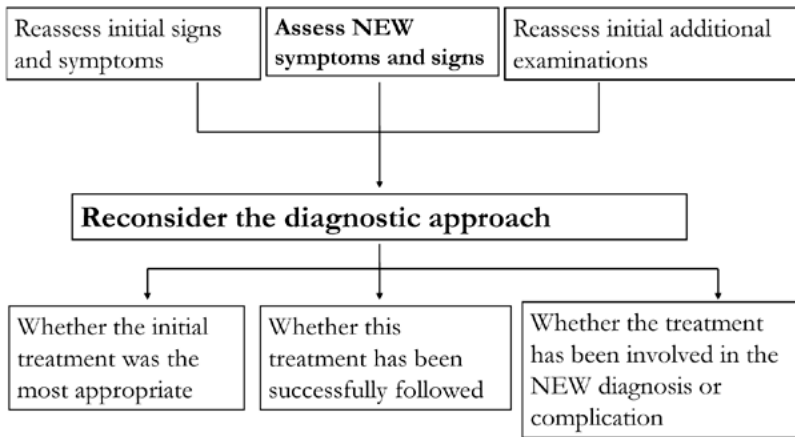
1. Diagnoses that are added to the initial diagnosis:

- Assess initial symptoms and signs.
- Review initial additional examinations in which it might have appeared based on the diagnosis.
- If there are no signs and/or symptoms of it based on the diagnosis, assess causality criteria.
- Assess whether it might be a complication of the first diagnosis or not have anything to do with it.



2. Diagnoses that amend the initial diagnosis:

- Assess initial symptoms and signs.
- Review initial additional examinations and reconsider the diagnostic approach and/or evaluate:
 - Whether the initially prescribed treatment was the most appropriate according to current medical science knowledge
 - Whether this treatment has been successfully followed
 - Whether the prescribed treatment or its application has been involved in the emergence of a new diagnosis or a complication



3. Complications per se:

3.1 Derived from the first diagnosis:

They present in the course of an illness or injury as part of its natural history or clinical course.

3.2 Derived from the application of treatment:

Treatment complications can be derived from:

- Its choice which is often determined by human and/or material factors
- Its indications. Treatments can be considered as indicated, contraindicated and not indicated. The latter two cases often prolong healing time and contraindicated treatment can also cause sequelae.

Another factor to consider is that treatment in the public health system tends to lengthen healing time and consequently the appearance of sequelae.

1.3 Derived from additional diagnostic tests

Whether these are indicated, not indicated or contraindicated needs to be considered.

4. Aggravation of the previous condition.

Which damage is due to the previous condition and which to the injurious act should be specified from the forensic medicine point of view but this is often difficult and sometimes only approximate conclusions can be established about:

- The probable course of the previous disease without the injurious act
- The probable course of the injurious act without the previous condition, and
- The consequences of the association of the previous condition and the injurious act.

The injurious act may have revealed a latent prior pathological condition not known to the patient.

5. Concurrent contributing cause:

The coincidence of two events or circumstances that influence the production of an injury increases its final significance and from the point of view of forensic medicine this translates into:

- The healing period is lengthened and/or
- The criminal definition becomes more serious and/or
- Sequelae are extended

6. Intercurrent or *subsequent or consecutive* contributing cause.

The events or circumstances that influence the damage once it has been produced but before its final stabilisation may also from the forensic medicine point of view lengthen the healing period and/or change the criminal definition and/or extend sequelae.

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Chapter V

Injury Forecast Reports

Authors: Dr. M^a Teresa Talón Navarro*, Dr. F. Xavier Llebaria Enrich and Dr. Antonio Soler Murall

Expert forensic medical work involves furnishing the courts with knowledge from a medical discipline about the assessment of bodily injury with the subjectivity typical of this discipline.

The legislative amendments which have been introduced involve changes in the practice and procedures normally used in our assessment work and particularly concern injury forecast reports in which we have to report on the future course of these injuries depending on their magnitude and features. These forecasts are made with all due caution and yet also the imprecision entailed by individual patient and event variability or the onset of complications, but which we are legally required to provide by recent regulatory reforms.

The imprecision of this expert function is accompanied by the degree of subjectivity of each expert which becomes more striking the greater the simplicity of the expert opinion that has to be provided.

In order to enhance the legal certainty required of us, we seek in the abstract and especially for the most basic cases of injury to reach a consensus about assessment items that will improve our work as experts in terms of providing greater predictability and uniformity in simple cases where this is possible.

The reforms introduced by Act 38/2002, of 24 October, on partial amendment of the Criminal Procedure Act with respect to fast-track and immediate trials for certain crimes and misdemeanours and amendment of the summary procedure, and by Act 8/2002 of the same date which adds to the former, affect three areas:

1. Fast-track trials for certain crimes with the setting up of a special procedure (Sections 795-803 – T.III / B.IV).
2. Amendment of summary procedure (Sections 757-794 – T.II / B.IV).
3. Immediate trial of misdemeanours under the new regulations in B.VI of the Criminal Procedure Act (Sections 962-977).

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The 3rd Final Provision of the above Act specifies it comes into effect six months after its publication, and since this took place on 28 October 2002 the legal reform will have come into force on 28 April 2003.

We think that 7 years on it is appropriate to take stock of the real impact of these reforms on our work as expert witnesses as opposed to the initial uncertainties they produced.

To that end we will examine the procedural occasions where expert forensic medicine intervention is provided for along with the specific features of such intervention.

1. *Fast-track trials for certain crimes with the setting up of a special procedure* (Sections 795-803 – T.III / B.IV).

Section 796 (Chapter II on the Judicial Police).

“Without prejudice to requiring the care referred to in Section 770 1, the health personnel who have attended the victim shall be asked for a copy of the report on care given to be attached to the police report. The presence of the Medical Forensic Expert shall also be requested when the person who is to be examined is unable to travel to the duty magistrates’ court in the period specified by Section 799”.

Section 797 (Chapter III on “Urgent Proceedings before the Duty Magistrates’ Court”).

1.2. b):

Once the duty magistrates’ court has received the police report, “It shall where relevant and reasonable order the Medical Forensic Expert, if they have not done so before, to examine the people who have appeared before the court and issue an expert witness report”.

2. *Amendment of summary procedure* (Sections 757-794 – T.II / B.IV).

Section 778 (chapter 3 on “Preliminary Proceedings”).

“2. In the event of injuries it shall not be necessary to wait for the healthcare of the injured person when closing or dismissing the case is appropriate. In any other case processing may continue without having obtained such healthcare if it is possible to draw up the charging document.”

3. *Immediate trial of misdemeanours under the new regulations in B.VI of the Criminal Procedure Act* (Sections 962-977).

Sections 964 and 967.

“...must appear with the evidence on which they intend to rely”.

Section 965.

“If it is not possible to hold the trial immediately... The summons will be issued (among others) ... to the experts who can give an account of the facts.”

There are two types of injury forecast reports:

1. *Reports in the light of the medical documentation.*

Section 796 (chapter II on the Judicial Police):

“...the health personnel who have attended the victim shall be asked for a copy of the report on care given to be attached to the police report.”

The Medical Forensic Expert has to assess the medical documentation provided together with the police report.

2. *Forecast reports after medical-forensic examination.*

Section 797 (Chapter III on Urgent Proceedings before the Duty Magistrates’ Court): “It shall where relevant and reasonable order the Medical Forensic Expert, if they have not done so before, to examine the people who have appeared before the court and issue an expert witness report.”

After the medical-forensic examination at two procedural points:

- Before the Duty Magistrates’ Court, once the police report has been received and as part of the urgent proceedings begun, if not done before and deemed relevant and reasonable by the court.
- Before the immediate trial of misdemeanours as an expert witness who can account for the facts (this case would be applicable only when we assess the documentation).

We will *analyse the medical documentation* available in court proceedings in relation to the facts giving rise to them as the basis our study.

Ideally this would mean that such documentation contained the objectification of the injuries by medical personnel with a description of the physical examination conducted for this purpose. However, the reality of our expert practise is often highly variable with respect to this ideal situation and other common cases:

- The health documentation that has been made available has been drawn up after care provided by paramedics.
- Medical personnel have not provided a description of the injury in the documentation.
- Medical personnel provide a description of the mechanism of injury as a diagnostic guideline without stating or detailing exploratory findings.
- Medical personnel provide a description of subjective symptoms (reported pains) as a diagnostic guideline.

We have to assess whether the examination leading to the medical documentation took place after the events leading to our intervention. This means taking extra precautions and checking the time lag between the events being analysed and the examination they gave rise to.

A specific report has to be drawn up including:

- *Identification*: name and age together with the number and type of judicial proceedings.
- *Dates* of the events and care.
- *Causal mechanism* / possibility of *contributing causes*.
- Description of the *examination and diagnostic guideline* together with any actual examination by the duty forensic officer if requested and it can be carried out.
- *Initial treatment plan*.
- *Average healing time* + *assessment of barriers* to performing usual tasks.
- *Type of treatment*.
- Whether the *sequelae* can be predicted.
- Potential *forensic medical considerations* concerning the case in question and which we think should be described in order to clarify or specify one or more points in our expert report.

In the case of simple injuries, and after reviewing the essential diseases and the expert experience shared at meetings on this issue and a clinical session open to all medical forensic experts at the Institute of Legal Medicine of Catalonia on the dates previously reported by the coordinator of the initiative and the Head of the Bodily Injury Assessment Section in the Forensic Medicine Clinic Service, we were able to agree on the items to be assessed:

Injury types	Length	Treatment type	Sequelae
<i>Erythema</i>	hours – 1 day	Initial care	NO
<i>Erosion</i>	1 – 3 days	Initial care	NO
<i>Abrasion / excoriation</i>	1 - 5 days	Initial care	Possible cosmetic
<i>Wound</i>	7 – 10 days	Initial care / treatment	Possible cosmetic
<i>Ecchymosis</i>	3 – 5 days	Initial care	NO
<i>Haematoma</i>	3 – 8 days	Initial care	NO
<i>Special haematomas</i>	8 – 15 days	Initial care	?

We would like to clarify that some diseases within the special haematomas (e.g. subungual haematomas or subconjunctival haemorrhage) require a longer period for resolution than that given above. This is because they involve skin pigmentation or a longer period of injury resorption which we believe will be resolved with the simple passage of time (e.g. a physiological process of nail replacement) and are therefore difficult to include as sequela symptoms in the *strict sense* of the term (the little used and questionable term *provisional sequelae* would seem to apply here). At any event they would require clarification in the considerations section in the expert report.

In the case of more serious injuries we believe we can:

- Report the type of treatment.
- Whether sequelae are foreseeable in general.
- Establish an average time for healing of the injury in question.

Here we refer to the periods set out in the Protocol of the Institute of Legal Medicine of Catalonia on the Scale in Act 34/2003, of 4 November.

Chapter VI

Rehabilitation, Basic Assistance or Medical Treatment

Authors: Dr. Maria Rifà Damunt,* Dr. Maria Victòria Bonastre Paredes, Dr. Josep Ramis Pujol and Dr. Joaquín Recio García

1. Introduction

In our work we often have to assess injured people who have completed a functional rehabilitation (FR) or physiotherapy programme after trauma. The problem arises when the person has done or is doing a huge number of FR sessions, which makes us wonder whether all these sessions have been or are necessary and especially when the trauma that resulted in the injuries was slight.

FR should be used to promote healing and reduce the time it takes. However, we often find, as is also confirmed by the literature, that injuries where FR was not used have a shorter healthcare period than those where it has been, with the most frequent example being whiplash syndromes.

Furthermore, physiotherapy and rehabilitation care designed to restore all or part of the functional deficit of a physical element often becomes a means whereby medical centres which live off insurers can use injured people to enrich themselves with the acquiescence of these injured people who see the chance to get much higher compensation.

To avoid this and to properly assess any injury, we need to consider in particular the initial injury so that the basic assistance report will guide us about its severity and progress. The time elapsed between when the injury occurred and the beginning of FR will tell us whether the latter has been effective since a major delay before starting FR will probably make it ineffective. Likewise, the emergence of new symptoms some time after the initial injury will suggest that there is no link between the former and the latter.

It also needs to be said that we should be surprised by a large number of physiotherapy or FR sessions for initially minor injuries which are indicated solely by subjective pain and dubious examination, a situation which continues on arrival at our consulting room where we

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find that the reported pain symptoms are not accompanied by genuine defence muscle spasms or real limitations on joint movement.

In a nutshell, as we shall see the number of days of FR should not determine our healthcare assessment as healing and process stabilisation may take place before FR finishes.

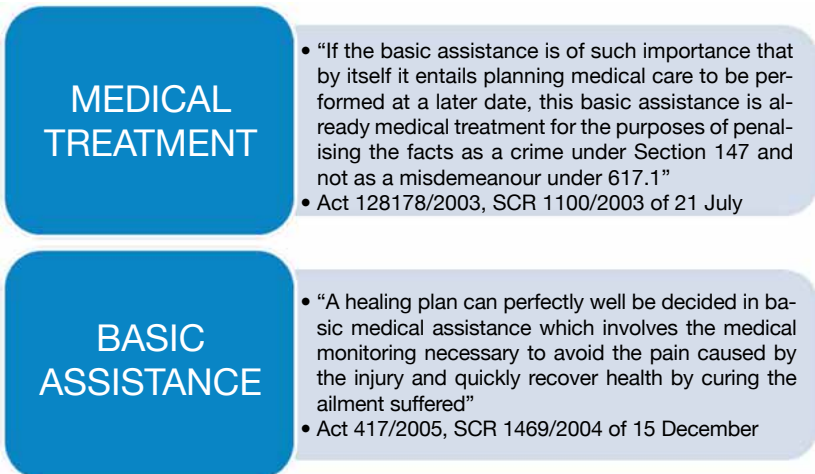
2. Medico-legal considerations

In all our injury reports we have to decide based on medico-legal criteria whether treating an injury has required basic assistance or medical treatment due to the different legal implications (misdemeanours or crimes) of each one.

Evaluating these concepts is sometimes problematic, for example in the case of whiplash, which means different professionals assess them differently.

To try to clarify the scope of these differentiation criteria we have reviewed the medical literature and found that we may sometimes fall into a trap which even led the State Attorney General in its Circular 2/1990 to set out differentiation guidelines. This has not however prevented experts becoming involved in disputes due to the evolution of both medicine and the law, as can be seen in Diagram 1, since these two concepts are defined differently based on time and knowledge.

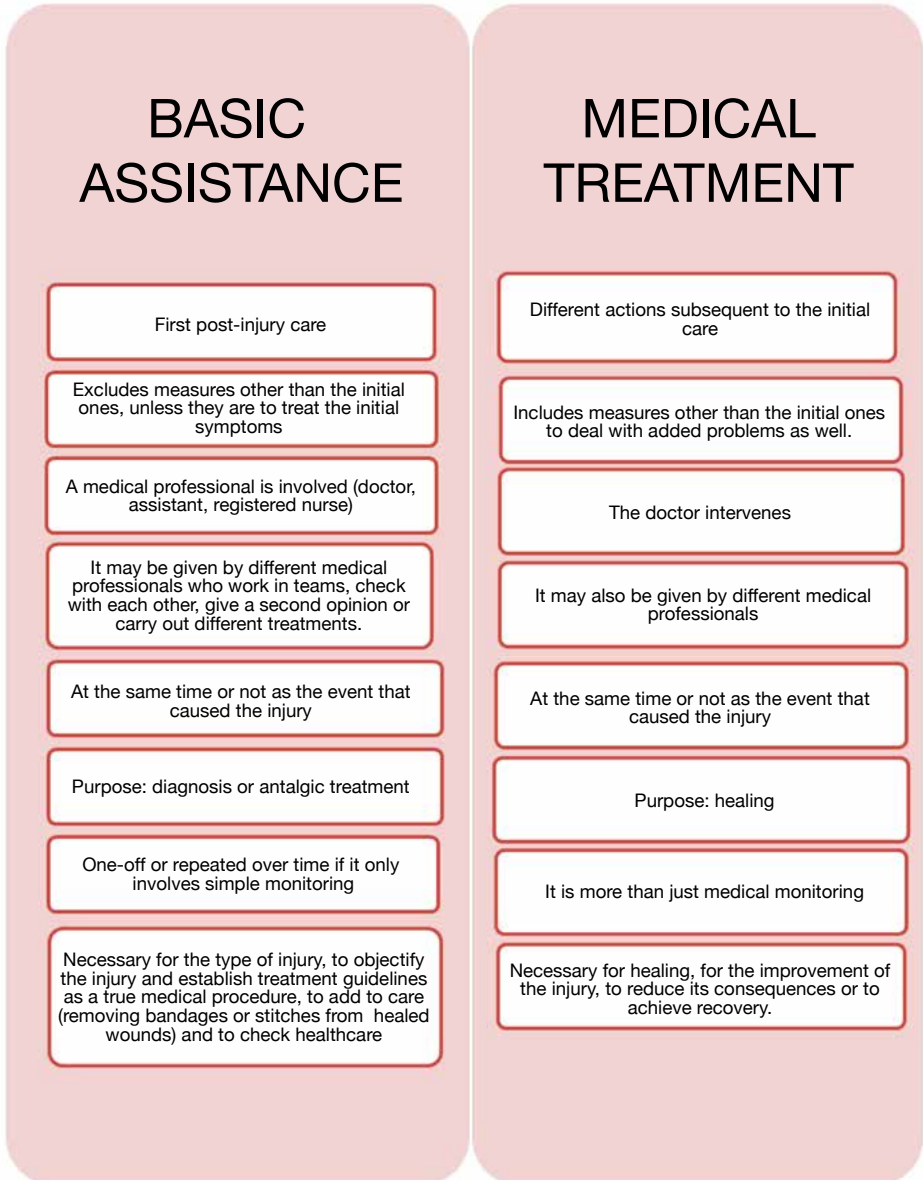
Diagram 1. Medico-legal controversies about the type of treatment. (Authors' own compilation)



Even so, we are left with the idea that, as shown in Diagram 2, the diagnostic or antalgic purpose versus that necessary for healing strictly defines the difference between the two medico-legal terms, between basic attention and medical treatment.

At any event, we believe it is always better to state in our report the type and features of the treatment: who decided on it, whether it was necessary, if it occurred at the beginning of the process, if it was part of the initial plan, if it has a symptom palliation or curative purpose and the type and amount of care received.

Diagram 2. Differences in medico-legal criteria in treating injuries. (Authors' own compilation)



3. Rehabilitation

3.1 History of rehabilitation

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Rehabilitation methods may seem modern, yet in fact this is not the case because if we look at the Roman *collegia* and medieval guilds we find that even then there were people who promoted treatment and healing of the injured (Diagram 3).

Pedro Ponce León made a major leap in the 16th century when far ahead of his time he set up the world's first school for teaching the deaf, thus beginning vocational training.

At all events, the word *rehabilitation* was not used until 1865 when it appeared for the first time with kinesiotherapy in a book by Dr. Busqué y Tormo, published in Madrid entitled *Gimnasia higiénica, médica y ortopédica*. Later on Lluís Vives laid the foundations for social medicine and rehabilitation.

Since then rehabilitation has evolved in lockstep with the needs of war, the wounded and their disabilities and different countries (USA, UK, etc.)

The Spanish Rehabilitation Society was founded in Spain in 1959 and the discipline was recognised as a medical speciality in 1969. From that time onwards it has been taught in a number of schools and centres.

Hence as we will see later on, today we cannot decouple rehabilitation treatment from the injury recovery process.

Diagram 3. Brief history of the evolution of rehabilitation. (Authors' own compilation)



3.2 Concept of rehabilitation

First of all we have to distinguish between the concepts of *rehabilitation* and *physiotherapy* as there are some basic specifications that need to be taken into account.

Often these terms are used interchangeably by the general public and even by health professionals themselves.

The AEF (Spanish Association of Physiotherapists) defines *physiotherapy* as the methods, activities and techniques which use physical means to cure, prevent and adapt both the disabled or people suffering from psychosomatic, somatic and organic disorders and those who wish to maintain an appropriate level of health. In a nutshell, physiotherapy's role is to prevent, treat and cure diseases and if there should be any sequelae to help the person adapt to their environment.

As for *rehabilitation*, the 20th edition of the dictionary of the Royal Spanish Academy of Language defines it as the methods that are intended to recover a function or activity that has been lost or impaired due to trauma or disease. From a more specialised standpoint the Stedman dictionary defines it as restoration after illness or injury of the ability to function as normal or nearly normal. Kessler establishes the concept of *rehabilitation* as the use of all medical instruments that can promote recovery geared towards treating people with a physical deficit. Thus rehabilitation, according to this definition, is the restoration of the disabled to their maximum possible physical, mental, social, vocational and economic limits. These medical means largely consist of using physical measures used to *supplement* medical and surgical treatments.

In addition the definition of the World Health Organisation (WHO) says that rehabilitation includes all measures aimed at reducing the impact caused by disabling and handicapping conditions and enabling people affected by these processes to achieve social integration.

Hence rehabilitation is an extremely broad concept which describes a process which has five main parts: clinical (physical and mental), psychosocial, educational, professional and recreational. In this respect Narbona states that physical and autonomy impairment entails loss of physical and also psychological and social capacity and affects both the individual and their family and community.

Hence rehabilitation is a conceptually broad yet also dense and deep process which does not dovetail with many of the procedures and practices of physiotherapy. Examples to underline this point would be sports physiotherapy and physiotherapy for various acute and chronic ailments in physiotherapy clinics.

3.3 Basic considerations about a rehabilitation programme

The medical fields of application of physiotherapy are largely orthopaedic and trauma surgery, rheumatology, gynaecology, respiratory medicine, neurology, sports ailments, cardiovascular disease, plastic surgery and psychiatry.

The techniques used in all of them include:

- *mobilisation*
- *physical agents*: electrotherapy, thermotherapy, cryotherapy, hydrotherapy, magnetic therapy, light therapy, pulley therapy, mechanical therapy, etc.

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When studying mobilisation it should be borne in mind that the amplitude of movement depends on the person's age, sex and morphology, training, presence or absence of disease, temperature, time of day and even mental state. However, it is very important to know the normal mobility of any movement and compare it with the healthy side.

Medical forensic experts and doctors who assess bodily injury need to know how to properly perform a progressive study of the **joint movement** of each joint during all the time the injured person is being monitored. Hence the most useful tools are a goniometer and tape measure in order to check the initial values prior to treatment, the same values during it and their final assessment.

The *Daniels' scale* (Diagram 4) and **Foreman and Croft's classification** for whiplash injury (Diagram 5), one of the most frequent pathologies in our practice, and which in turn can be extrapolated to any device or system, can help us to make a more accurate assessment during physical examination by defining the type of movement performed and relating it to the injury suffered.

At the same time we also need to relate Foreman's injury grades with the evolutionary stage of the process: acute, subacute, recovery or chronic (Diagram 6).

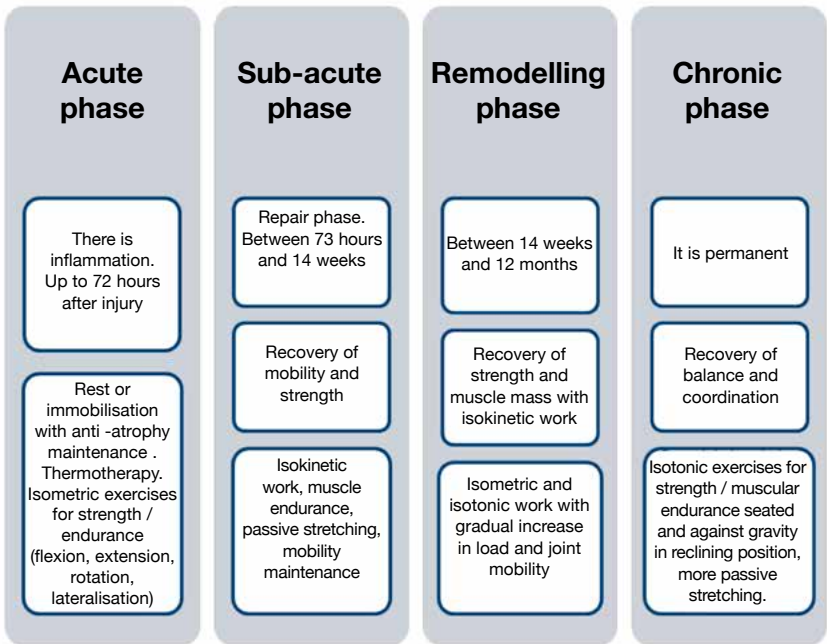
Diagram 4. Daniels' Scale. (Authors' own compilation)

- | |
|---|
| <ul style="list-style-type: none">0: No contraction detected1: Contraction detected without joint movement2: Complete movement but with no pressure or gravity3: Movement against gravity4: Movement against moderate resistance5: Movement against maximal resistance |
|---|

Diagram 5. Foreman and Croft's classification (1995). (Authors' own compilation)

- *Grade I:* No limitations of motion or ligamentous injury. No neurological findings.
- *Grade II:* Limitations of motion. No ligamentous injury, no neurological findings.
- *Grade III:* Limitations of motion. Some ligamentous injury, neurological findings may be present.
- *Grade IV:* Limitations of motion. Ligamentous instability; neurological findings present. Fracture or disc derangement.
- *Grade V:* Injury which requires surgical management or stabilisation.

Diagram 6. Evolutionary stages of whiplash. (Authors' own compilation)



However, it should be remembered that each case is individual as each injured person is different and requires analysis and treatment that are different as well.

Hence it will be necessary first to assess the patient's medical history where data and assessments are recorded which indicate the need to devise a comprehensive rehabilitation programme and the additional tests that have been done to prescribe FR while guaranteeing the safety of the injured person or patient.

Based on these parameters, in our view a patient will only require rehabilitation when they present a deficiency in at least one of the following: bodily functions, range of motion, pain, muscle weakness, coordination deficits or physical ability (Diagram 7)

Impairment in any of these areas will immediately lead to a greater or lesser change in their usual activities of daily living in various aspects such as mobility, handling, grasping, washing, taking care of themselves, cooking, dressing or going shopping. This means that their participation in work, hobbies, free time and social life or in family relations will also be affected to the extent that it does not allow them to act independently.

Diagram 7. Assessment of disability before, during and after healing/stabilisation of injury. (Authors' own compilation)

1. Joint function deficit: range of motion, lack of coordination and balance, impaired handling capacity and precision and gait and walking analysis.
2. Decline in muscle strength: electromyography assessment.
3. Involvement of pain: by sensory evaluation.
4. Analysis if executive, sensory and communication functions before, during and at the end of the process
5. Consideration of activities in daily living: washing, taking care of yourself, cooking, dressing, shopping.
6. Also social and family relations: leisure, free time or social life.

In the case of the musculoskeletal system, which is the one we assess most, rehabilitation goals include:

- Relieve pain.
- Reduce inflammation.
- Maintain or restore joint and muscular function, walking ability, balance, coordination and performance of basic and instrumental activities of daily living.
- Prevent deformities, joint stiffness, muscle and sensory deficits.
- Minimise disability, which may be temporary or permanent, enabling the person to fully perform their personal, professional and social role.
- Restore the maximum possible functional autonomy and independence.
- Improve quality of life.

It should be borne in mind that rehabilitation acts on the deficiency (anatomical, physiological or psychological), disability (when there is impaired performance of the person's tasks) and handicap (if there are problems with interaction and adaptation to the group and environment).

Hence we need to mention the following clinical assessments in our report:

- Muscle strength (it is advisable to ask for an electromyography study, EMG).
- Joint movement, goniometer.
- Balance and coordination.
- Gait and walking.
- Executive functions in brain damage.
- Sensory and communication functions.

We will also specify the intervention plan drawn up by the rehabilitation doctor, including medical measures and prescribing and/or using physical or occupational therapies such as those set out in Diagram 8.

Subsequently we will assess all the results and based on them and their physical examination (repeated during the evolutionary period to the extent possible) the medical forensic expert will draw up the discharge report and determine the assessment of bodily injury as complete recovery or with sequelae.

Diagram 8. Types of treatment. (Authors' own compilation)

<p>MEDICAL MEASURES</p> <ul style="list-style-type: none">• Support in drug treatment aimed at improving body structures (with analgesic, anti-inflammatory and tone regulation functions) and improving physical fitness and cognitive wellbeing, including treating anxiety and/or depression.• Practical procedures: steroid injections, hyaluronic acid, botulinum toxin, arthrocentesis.• Assess and review procedures.• Draw up a prognosis forecast and specify treatment targets.
<p>PHYSICAL TREATMENT</p> <ul style="list-style-type: none">• Manual therapy techniques for reversible rigidities and related soft tissue dysfunction. Spinal manipulations.• Kinesiotherapy and exercise therapy.• Spinal traction.• Making functional casts.• Orthopaedic reductions and making spine casts.• Electrotherapy (analgesia, electrical stimulation).• Other techniques: magnetic therapy, ultrasound, light therapy, thermotherapy, hydrotherapy, balneotherapy, massage, manual lymphatic drainage, extracorporeal shock wave, biofeedback.
<p>OCCUPATIONAL THERAPY</p> <ul style="list-style-type: none">• Prescription and review of prosthetics and orthotics.• Cardiac rehabilitation.• Pulmonary rehabilitation.• Vesico-sphincter rehabilitation.• Vestibular rehabilitation.• Language and communication therapy.

3.4 Different forms of therapy

Nowadays it is well known that rehabilitation treatments are personal and combine the different forms of therapy to influence and counteract joint functional impairment (joint mobility), increase muscle strength and reduce pain.

Below is a description of the most common forms of rehabilitative therapy, leaving others to one side as they are not usually used in our practise.

3.4.1 Kinesiotherapy

It is a therapy based on the benefits of movement rooted in Cyriax and Mennell's technological approach and Kabat, Bobath and Brunnstrom's methodological approach.

This kinesiotherapy can be part of manual therapy or active physical exercise.

In the former case, manual therapy (Diagram 9) can be performed *passively* through massage or mobility. It will then use isometric exercises with muscle contraction without joint movement (resistance is applied to prevent joint mobility).

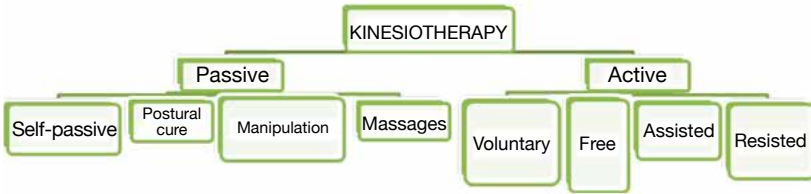
It can also be done *actively-passively* via corrective action by the physiotherapist and the patient's voluntary exercise. Isokinetic exercises are used with muscle contraction maintained and muscular elongation (the patient contracts the muscle and a machine or the physiotherapist produces the joint displacement).

Both isometric and isokinetic exercises increase muscle volume and maximum strength, although isokinetic ones are used more in muscle training programmes for healthy people and athletes while isometric ones are used in patients with bone and joint and/or trauma problems since they are less aggressive (Diagram 10).

Both techniques give very good results in the treatment of fractures, severe multiple trauma and after long periods of bed rest with muscle wasting, atrophy and joint stiffness.

In the second case of active exercise, which can be done at home or in a gym, exercises are done that are guided by mobility. It has beneficial effects on muscles, joints and osteoarticular structures, improving muscle extensibility and elasticity, enhancing and increasing muscular strength, articular range, cartilage lubrication and helping reduce oedema by improving blood circulation.

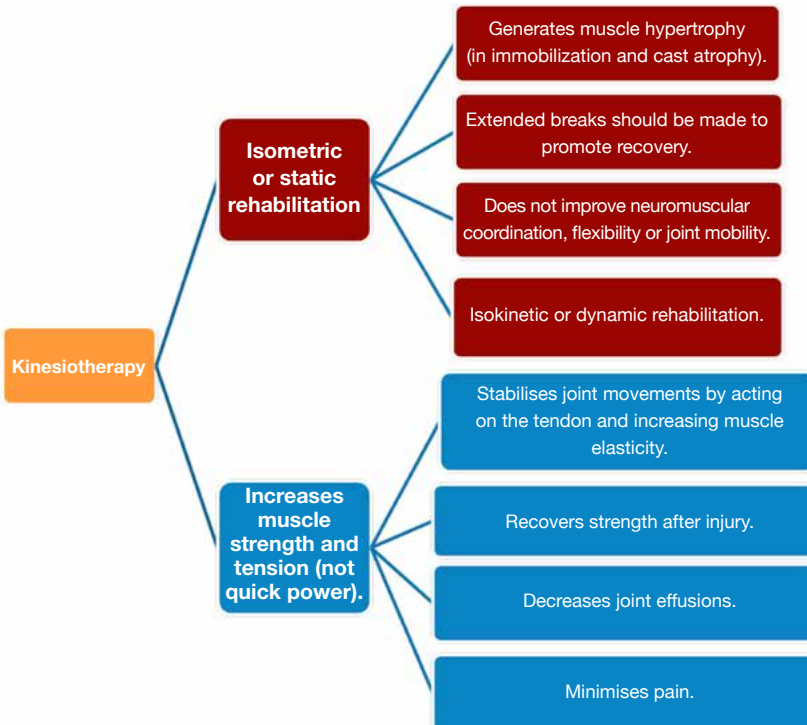
Diagram 9. Types of manual therapy in kinesiotherapy. (Authors' own compilation)



Kinesiotherapy is indicated in cases of skeleton deformities (especially spinal), soft tissue injuries or abnormalities, joint injuries or deformities, organic diseases and their sequelae (long-term bedridden), cardiorespiratory diseases or CNS diseases.

However, it is contraindicated in passive mobilisation if the joints are very painful, in recent soft tissue injuries, hypermobility in the joints (except flaccid palsy) and abundant joint effusions. Active mobilisation will also be contraindicated in ankylosis, recent fractures, inflammation (infectious osteitis, evolutionary tuberculosis foci) and herniated disc.

Diagram 10. Isometric and isokinetic rehabilitation work. (Authors' own compilation.)



3.4.2 Hydrotherapy, hydrokinesiotherapy, crenotherapy (hot spring)

There are many rehabilitation techniques that use water, some of them quite elaborate, and which are almost always used in combination.

They are little used in hospitals and almost exclusively in spinal injury and large paralysis rehabilitation centres. In the latter they are more accepted due to the beneficial effects of exercise in water as removing much of the effect of gravity makes movement easier and enables early ambulation and mobilisation, thus soothing sore muscles and improving venous return.

3.4.3 Electrotherapy

This technique is based on the effect of low, medium and high frequency galvanic currents applied depending on the patient's pathology.

They are applied using:

- Microwave devices.
- Laser for fixed or sweep treatment.
- Electric shock devices (enthesopathy, calcifications and consolidation delays).
- Biometric devices (assessment and training of the various functions of the hand and fingers).
- Isokinetic device (Con-Trex for the assessment and training of the lumbar spine and all limb joints).

3.4.4 Thermotherapy

It works through the properties of heat. It has moderate analgesic effects and increases blood flow in the region.

It helps with analgesia by facilitating joint mobilisation and resorption of oedemas.

There are two types: *surface* with infrared, hotpacks (hot sandbags) and paraffin (liquid wax) and *deep* through microwaves.

3.4.5 Cryotherapy

Treatment is based on the application of local cold (ice packs, aerosols).

It is useful in acute injuries because it has some very mild analgesic and anti-inflammatory effects.

3.5. Different types of exercises: isometric versus isokinetic rehabilitation

3.5.1 Isometric or static rehabilitation

Isometric exercises involve static muscle actions, i.e. those in which the length of the muscles activated does not vary while generating tension and therefore there are no changes in the angular position of the joint.

Such exercises are performed against a stationary object or when working a hard muscle group against a weak muscle group.

Isometric contraction programmes are used with some training symptoms, particularly in the rehabilitation of muscle groups affected by injury or surgery (for example where a limb has been immobilised by a cast) as they maintain muscle tone, thus reducing the scale of the atrophy and loss of muscle strength which facilitates the recovery process (Diagram 11).

Programmes with resistances using isometric exercises in multiple series are frequently used, including static contractions lasting at most from 3 to 6 seconds a minimum of three times.

Since isometric training enables maximum voluntary muscle contractions, it leads to remarkable gains in muscle strength and hypertrophy.

Diagram 11. Isometric or static exercise conclusions. (Authors' own compilation)

Static or isometric exercises bring about an increase in muscle strength and hypertrophy.

They do not train fast power strength.

They are very useful for developing stable and prolonged muscle tension.

They lead to a decrease in neuromuscular coordination, kinesthetic sense, flexibility and joint mobility.

It is recommended to combine them with the other exercises that make up for their shortcomings.

Breaks in isometric training should be lengthy to facilitate recovery.

They are indicated in muscle atrophy due to cast immobilisation of the legs.

3.5.2 *Isokinetic or dynamic rehabilitation*

This treatment involves the possibility of studying the whole musculoskeletal system. Basically it is a process by which a body segment moves at a preselected fixed speed against resistance determined by the maximum force generated by the patient.

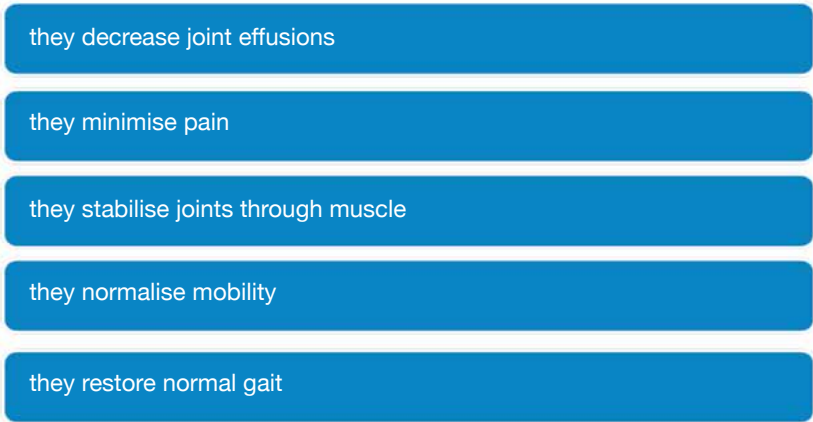
The use of isokinetic exercise in rehabilitation is due primarily to the need in the field of occupational medicine to obtain objective measures of impairments. Usually their assessment is determined by the loss of joint movement.

These isokinetic exercises, like isotonic ones, objectify dynamic strength while isometric exercises objectify static strength.

Isokinetic rehabilitation is to be used for injury prevention (identifying strength impairment or involvement), post-injury strength recovery, treating muscle and tendon and/or insertion injuries and achieving muscle elasticity (Diagram 12).

However, it is important to note that isometric exercises are performed first during the rehabilitation process to be followed by isokinetic exercises.

Diagram 12. Isokinetic exercise conclusions. (Authors' own compilation)



3.5.3 *Exercises by systems*

To conclude this practical section, immobility entails a series of pathophysiological consequences for haemodynamics and the cardiovascular, respiratory function, nutritional, metabolic, nephrology and urology, skin, muscle (by atrophy and hypotrophy), bone, joint and neuropsychological systems.

Based on these involvements we have classified the different types

of rehabilitation by systems in Diagram 13 below.

Diagram 13. Different types of rehabilitation. (Authors' own compilation)

- Functional rehabilitation (FR) for upper extremity peripheral nerve injury: brachial palsy.
- FR for lower extremity peripheral nerve injury.
- FR for quadriplegia, paraplegia, neurogenic bladder, spinal hyperreflexia.
- FR for central nervous system lesions. Head injury, hemiplegia.
- FR for fractures: fractures of the upper extremity, lower extremity, spine fractures without neurological complication.
- FR for soft tissue pathology: scapulohumeral peri-arthritis, tendonitis, enthesitis, more frequent muscle injuries.
- FR for osteoarthritis: knee osteoarthritis, hip osteoarthritis, joint replacement
- FR for brain damage: severe head injury or stroke
- FR in traumatic spinal cord injury
- FR for spinal pain (cervical and lumbar)
- FR for posttraumatic peripheral nerve injuries
- FR for muscle tendon injury
- FR for shoulder pathology (aggravation of degenerative processes prior to injury due to trauma, joint replacement)
- FR for knee pathology (ligaments, meniscus, joint replacement)
- FR for ankle and foot pathology (ligament injuries, heel pain, plantar fasciitis, metatarsalgia)
- FR for amputees (upper and lower extremity)
- FR for spine and pelvis
- FR for upper extremity and shoulder girdle
- FR for legs
- FR for burn patients and amputees
- Respiratory FR
- Cardiovascular FR
- Neuropsychological FR
- FR in transplant patients
- FR for sick children
- FR for geriatric patients
- FR at home.

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3.6 Assessment of the result of rehabilitation

When in the end we have to assess the rehabilitation process we may come across a situation that we analyse here for the case of spine and pelvis rehabilitation as an example that can be extrapolated to other systems.

Our physical examination of the spine prior to our report first assesses the attitude of the neck, looks at how the patient walks and evaluates their freedom of action.

It also is also a good idea to systematically examine the spine just as with any joint as this will help us to make an overall assessment of the person's anatomy and determine how far it matches the symptoms reported by the patient.

We begin with a general inspection (kyphotic neck, scoliosis, congenital malformations), followed by palpation (especially the cervical, dorsal and lumbar spinous processes to rule out the existence of an exaggerated interspinous step-off, which may signify the presence of posterior ligamentous instability, and we also assess the paracervical and trapezius muscles) and local and distant percussion.

Then we determine active and passive mobility, joint movement of the involved and contralateral area and walking.

Next we study reflexes and neurological examination of cranial nerves if need be.

We also study of muscle sensitivity and strength which may be appropriate depending on the case.

The determination of functional pain (non-organic): cutaneous hyperalgesia and exaggerated overreaction in cases of simulation will also be useful.

Our examination will be rounded off by studying the image diagnosis tests the patient normally brings with them.

3.7. Rehabilitation periods

Based on the abovementioned idea that rehabilitation aims to restore good physical and mental, psychosocial, vocational and recreational quality that the patient has lost as a result of the injury sustained, the medical forensic expert should regularly assess the injured person to see in each session whether the injury has been healed or not yet achieved injury stability.

Hence we will always need to be cautious and wait a sufficient time for the rehabilitation to achieve maximum joint mobility.

As an example we can take the case of a middle-aged patient who has had an open fracture of the tibia, so in the first month they begin to evolve favourably and after six months begin knee and ankle rehabilitation. If we monitor their evolution we see that after two months of rehabilitation, i.e. eight months of evolution, they have achieved injury stability leaving a certain sequela.

Hence it is advisable that when the injured person is visited we note down the degrees of joint movement they gain over time to compare how much they gain week by week. Then in each statement of condi-

tion we will objectively point out the degree of joint movement they are gaining and the evolution/gains in other symptoms. Nonetheless, we know that as rehabilitation treatment time continues the effect of rehabilitation becomes smaller which means in the last stages of treatment gains are much lower than in its early stages, and it is now when we can consider injury stability has been achieved.

It is at this point when there is no progress or change that they have to be given medico-legal discharge.

This medico-legal discharge is obviously completely separate from medical discharge, and may even be given without the planned rehab sessions having been completed. This is something we commonly come across in whiplash cases with injured people who have done rehabilitation for 6 months or more, added to the fact that rehabilitation began three or four months after the accident thus reducing the efficiency and success of rehabilitation.

In fact, when joint mobility is almost complete or full muscle strength is also almost restored (4/5), FR will no longer be curative and the injury has stabilised.

However, it is sometimes difficult to specify the number of rehabilitation sessions in advance due to factors connected with the injured person or patient, the centre where treatment is given or the attending doctor.

We have basically evaluated two types of tables, Tebex and the Social Security, to establish a criterion for guidance on the minimum and maximum number of sessions which may be required per condition or involved joint, although we would repeat that each case is individual (there are patients and not diseases). Thus in Diagram 14 we set out in detail rehabilitation sessions by involved joint.

By way of summary, injuries to these structures require:

- | | |
|--------------------|-------------------|
| • Shoulder girdle: | 15 to 30 sessions |
| • Arm: | 20 to 30 sessions |
| • Elbow: | 15 to 25 sessions |
| • Forearm: | around 45 |
| • Wrist/hand | 20 to 30 sessions |
| • Pelvis | 30 to 50 sessions |
| • Thigh | around 50 |
| • Knee | 20 to 50 sessions |
| • Leg: | 35 to 55 sessions |
| • Ankle: | 20 to 35 sessions |
| • Foot: | 20 to 50 sessions |
| • Spine: | 15 to 45 sessions |
| • Neuralgia: | 20 to 30 sessions |
| • Joint pathology | 35 to 55 sessions |
| • Rheumatisms | 30 to 65 sessions |

In general when a leg joint is affected you need a higher number of rehabilitation sessions (compared to an arm). This is because regardless of the injury sustained, these joints have to withstand body weight and require a period of immobilisation and reduced load that delays the start of rehabilitation thus leading to greater loss of mobility and strength.

It also has to be borne in mind that according to data from various rehabilitation centres, 30% of patients receiving rehabilitation should not be doing it because it is not justified by their clinical signs, because it is too late or because it does not lead to any improvement.

Diagram 14. Number of sessions by joint and associated pathology (Authors' own compilation)

SHOULDER GIRDLE	Collarbone fracture Humeral fracture Dislocated shoulder	15 to 30 sessions
ARM	Diaphyseal humeral fracture Rotator cuff tendinitis Bicep tendon tear Scapulohumeral periarthritits	20 to 30 sessions
ELBOW	Supra-inter-condylar fracture Epitrochlea fracture Epicondyle fracture Olecranon or radial head fracture Dislocated elbow	15 to 25 sessions
FOREARM	Diaphyseal radius and ulna fractures	around 45 sessions
WRIST AND HAND	Colles' fracture Barton joint fracture Bennett fractures Phalange or metacarpal fractures Carpal bones (scaphoid) fractures	20 to 30 sessions
PELVIS AND HIP	Stable or displaced fractures Sacrum, acetabulum, isquiopubianes branch fracture Traumatic hip dislocation	30 to 50 sessions

THIGH	Upper femoral epiphysis fracture Diaphyseal femur fractures Lower femoral epiphysis (upper, lower, intercondylar) fracture	around 50 sessions
KNEE	Patella fracture with/w.o. displacement Collateral or cruciate ligament tear Tibial plateau fracture Patellar tendon rupture Meniscal injuries	20 to 50 sessions (with arthroscopy 50% less)
LEG	Tibia and fibula fractures displaced or not, 35 to 55 sessions open or closed, with/without substance loss	
ANKLE	Supramalleolar fracture Uni or bimalleolar joint or pilon fractures	20 to 35 sessions
FOOT	Achilles tendon rupture Calcaneus or talus fracture (with / without subtalar inv.) Tarsal and metatarsal fracture Ligament ruptures Sudeck's atrophy	20 to 50 sessions
SPINE	Muscle or ligament trauma (whiplash) Deformities (kyphosis, scoliosis) Herniated disc Stable or unstable vertebral fractures	15 to 45 sessions
NEURALGIA	Neck pain, back pain, lower back pain Carpal Tunnel Syndrome Quadricipital or patellar tendonitis Dupuytren's Disease Talalgia	20 to 30 sessions

BONE AND JOINT PATHOLOGY	Osteoporosis, osteomalacia, Paget's D. 35 to 55 sessions Aseptic necrosis of the femoral head or scaphoid Inflammatory arthropathy Rheumatoid arthritis Ankylosing spondyloarthritis Gout
DEGEN. RHEUMATISMS	Osteoarthritis of the hip 30 to 65 sessions Knee osteoarthritis In total prosthesis (arthrodesis, arthroplasty, osteotomy)

In addition we can also sometimes be asked for guidance about the number of injuries by the type of structure injured.

Thus we have cases of *trauma without anatomical injury* whose course depends on individual response (applying the simile of a wide-spread saying in medicine that there are injured people and not injuries), but in which there is usually total restoration in a short time. It has to be borne in mind and excessive exertion should not be made.

These traumas include:

- *Contracture*: stabilisation or healing period between 1 and 3 days
- *Elongation*: stabilisation or healing period between 5 and 9 days
- *Fibrillar rupture*: stabilisation or healing period between 10 and 20 days

As for treatment, they heal themselves. By using treatment we speed recovery and reduce symptoms.

- *DOMS* (post-exertional muscle pain: plasters). In these cases it is advisable to do the same exercises that caused it. TENS and mild massage therapy can also be used to reduce pain as normally these types of injuries do not require immobilisation or rest.
- *Contusions*. The mechanism of production is often a traumatic agent acting against the muscle and compressing it against the deep planes. The severity of the injury depends on the characteristics of the traumatic agent and the state of the muscle (contraction, greater severity). If severe, it is treated as a muscle tear. Initial treatment is cryotherapy (ice) and rest and, later on, thermotherapy (improves circulation and healing), laser (regenerator by helping ATP metabolism), electrotherapy (enhancing muscle mass) muscular re-education and finally proprioceptive rehabilitation.

- *Muscle tear*: According to the classification given in Diagram 15, based on the number of broken fibres it can range from 10 days to over six months.

Diagram 15. Muscle ruptures (Authors' own compilation)

1. **Fibrillar rupture or tear**: small number of fibres affected. 10 days' immobilisation before beginning exercise.
2. **Partial tear**: large number of fibres affected, at least one fascicle. 8 weeks' rest before beginning exercise.
3. **Total tear**: complete interruption (total rupture) of a muscle. 6 months' rest.

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- *Tendon tears*. A tendon tear is more serious than a muscle tear due to the poor vascularity of tendons compared to muscles, added to the fact that adhesions occur more easily. Immobilisation is carried out so that the tendon is not stretched in order not to separate the ends involved.

The tear is accompanied by haematoma and bruising at a distance due to diffusion through the interstitial space. If the haematoma is trapped inside the fascicle resorption is hindered and pain and inability to regain muscle contraction persists. Ultrasound is the ideal additional examination.

Repairing tears includes two counteracting processes: firstly muscle regeneration and secondly healing and therefore fibrous tissue, so that if the fibrous tissue predominates the possibility of recurrence is greater. If the progressive gain in joint amplitude stops or decreases, physiotherapy treatment should be temporarily halted, the patient should rest and the problem reconsidered.

As for treatment (Diagram 16), prolonged immobilisation is not recommended as it may lead to more rapid appearance of granulation tissue which delays muscle repair. Thus at first bleeding or haematoma has to be controlled by compression and cooling. Diagnosis and prognosis of the lesion is performed afterwards. Finally, in the first 2 or 3 days the inflammatory process is treated by elevation of the limb, complete rest and administration of anti-inflammatory drugs.

In the following days, and once the severity of the injury has been assessed, the method to be used is chosen ranging from rest to mobilisation.

Diagram 16. Tendon tears

1. Partial rupture injuries heal by themselves.
2. Incomplete rupture injuries are treated by immobilisation and subsequent mobilisation.
3. Complete rupture injuries are treated by immobilisation or surgery with subsequent function rehabilitation and strength recovery.

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- *Peritendinitis*. These lesions are most common in extensors, anterior tibial and Achilles tendon and are often assessed by crepitus in movement.

The most common production mechanism is overuse and its clinical signs are painful palpation, soreness in active mobility and against resistance.

Treatment involves rest with a splint for 3 weeks and physiotherapy.

- *Tendinitis*. Treatment will depend on the evolutionary stage as in the acute phase it consists of rest and immobilisation. In the post-acute phase it is administration of anti-inflammatory drugs and cryotherapy with subsequent thermotherapy and ultrasound and laser.

Analgesia, interferential current, TENS and ionization are used to treat the pain.

- *Sprains*. If it is a *mild sprain*: the pain and oedema is treated with proprioceptive rehabilitation, recovery of mobility, increased strength and endurance, and rehabilitation for ADL and return to physical activity

If it is a *severe sprain*: surgery. If the reduction of the dislocation is quick and treatment correct, it can heal in 3-5 weeks. If the reduction is late or there are associated problems, the dislocation may become recurrent without surgery.

- *Synovitis*. The symptoms consist of swelling and heat in the joint area, pain and rapid muscle atrophy.

Treatment: heat the joint area if there is swelling, whereas compression, cryotherapy and rest are needed if there is pain. Later on gradual exercise will be used to prevent the onset of effusions.

- *Bursitis*. The production mechanism is a mechanical irritation or a bacterial infection.

Treatment: deep heat, ice, rest, kinesiotherapy (depending on evolution). If it is infectious, rest and pharmacological treatment are required.

4. Assessment protocol for rehabilitation treatment

1. Rehabilitation in medico-legal criteria:

- a. Basic assistance
 - i. If it is necessary
 - ii. If it is included in the initial measures
 - iii. When included to treat the initial symptoms
 - iv. If it is antalgic
- b. Medical treatment
 - i. If it is necessary
 - ii. If it has to be added to the initial treatment due to complications or different pathology
 - iii. If it is healing

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2. Assessment of rehabilitation in our report

- a. Mention whether or not it is necessary for healing
- b. Determine its medical purpose
 - i. Reduce pain
 - ii. Gain mobility
 - iii. Regain joint function
- c. And what the injured person's goal is
 - i. Go back to daily living activities
 - ii. Go back to work
 - iii. Go back to leisure pursuits

3. Determine the need for rehabilitation

- a. If there is any alteration in body function
 - i. Deficit in range of motion
 - ii. Muscle weakness
 - iii. Lack of coordination
 - iv. Lack of physical ability
- b. If there is pain
- c. If there is greater or lesser alteration in normal activities of daily living
 - i. Mobility, handling, gripping
 - ii. Daily living activity, family, social or work.

4. Establish what has improved

- a. In inflammation, pain
- b. Mobility, handling, grip and strength
- c. In balance and coordination
- d. In the deficiency (anatomical, functional or psychological), disability (in the performance of tasks as a person) or handicap (problems in adaptation and interaction with the group and the environment)

5. Start date of the rehabilitation

- a. As early as possible
- b. Reduce waiting lists and bureaucracy
- c. Shorten periods between surgery
- d. Finding the balance between rest for the fracture, stability of the bone callus and starting FR of adjacent joints.

6. Assess which type of rehabilitation

- a. All FR is personalised
 - b. It varies between a minimum and maximum number of sessions
 - c. Concerning the type of injury and where it is (assess medical history, functional diagnosis, previously set targets with the plan and means required)

7. What indicates a lengthening of the period of rehabilitation

a. Socio-economic factors

- i. By the health professional
 1. As defensive medicine
- a. Shutting the patient up or avoiding confrontation
- b. Doctors too accommodating with unjustified subjective discomfort
 2. To increase the revenue of the rehabilitation centre
- ii. By the patient
 1. If a victim (expects compensation)
 2. If a businessperson or freelance (can extend sick leave)

b. Medical factors

- i. Pain when doing rehabilitation
- ii. Coexistence of different diseases at the same time
- iii. Previous condition of the injured person
- iv. Complications (delayed bone consolidation, pseudarthrosis, infection, fistulas, reoperation)
- v. Possibility of improvement

8. When there is suspicion of unjustified extension

- a. If there is a long time between the recommendation for FR and when it actually begins
- b. If discharged just before the medical forensic expert's visit
- c. If discharged when the medical forensic expert asks for a report
- d. Sometimes if FR is restarted after returning to work

9. Date of discharge from rehabilitation

- a. When joint gain decreases to the point of joint stability
- b. If mobility is almost complete or muscle strength is 4/5, FR will no longer be curative and the injury has stabilised.

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Chapter VII

Assessment of aesthetic injury: evaluation criteria

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1. Introduction.

The assessment of aesthetic injury has become particularly important in recent years, mainly due to the attention given in today's society to beauty, aesthetics, body harmony, etc., as great significance is placed on these factors in the social and employment spheres as well as in almost all aspects of intimate relationships. As Arimany [1] points out, case law has held that human morphology has a significant impact on the economic, social, individual, psychological and even psychiatric aspects of a person irrespective of gender, age or profession.

In daily practice, appraisals must often be performed on people with injuries from road accidents, assaults, workplace accidents, chance accidents, etc. These include different types and severity of injuries, with occasional severe residual damage and often with sequelae affecting the individual's aesthetic heritage. These sequelae should be described in detail and their circumstances, location, visibility and intensity should be assessed. An attempt should be made to quantify them in terms of the particular ways they affect a person, and they should eventually be rated on a scale.

We believe that from a medical perspective it is more correct to speak of aesthetic injury, since the term damage is legal in nature, since a dictionary definition of the word *damage* gives the following meaning: "Something lawfully possessed that is no longer possessed; depreciation or expenses resulting from an act or omission of another person, who must provide compensation for the harm or material loss they have directly caused. The term *aesthetic* is defined as "that belonging or related to the perception or appreciation of beauty" and is contrasted with the term *ugliness*, meaning "devoid of beauty" [2].

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In accordance with Alonso, we define aesthetic injury as “Any physical irregularity or external body irregularity which is visible, permanent and ostensibly ugly to the naked eye”. To these characteristics the author adds that “another of vital importance must be added, which is that the affected person should feel this as such and their feelings are basis of the injury and therefore the damage” [3]. The fundamental characteristics of aesthetic injury are thus: 1. A physical irregularity or external body alteration. 2. The fact that it is permanent. 3. Its visibility. 4. The existence of ugliness, i.e. disfigurement of the individual’s morphology. 5. The existence of suffering by the victim which constitutes genuine moral harm [3,4]. Finally, the current scale of road accident injuries laid out in Royal Decree 8/2004, of 29 October, defines aesthetic injury as: “Any harmful change affecting image of the person and that constitutes a dimension of the physiological damage that serves as substrate in regards to both their static and dynamic expression” [5].

In the following pages we will discuss the different types of cosmetic damage, methods of assessing them and the evolution of Spanish scales for assessing aesthetic injury. We will conclude by examining its assessment from the medical forensic expert’s perspective.

2. Types of aesthetic injury

Aesthetic injury can be classified into two groups: static and dynamic aesthetic injury [3]. Static injury would include that which is perceived by the naked eye from a single examination of the victim. For example: simple scars, burns, pigmentation, mutilation, loss or reduction of a profile or contour as well as the loss of substance, limb differences, etc.

Dynamic aesthetic injury is noticeable from the person’s movements or changes in attitude. For example: gait disorders (limping), facial expression (facial palsy), abnormal gestures (ataxia, apraxia, hemiplegia, tremor, etc.), speech (dysarthria, stuttering, hoarseness, etc.) and disorders related to body odour (halitosis, or urinary fistulas or stercoraceous, colostomy discharge, etc.).

3. Current methods of assessing aesthetic injury.

In accordance with Rouge et al. [6], assessments of aesthetic injury begins with a detailed description of injuries visible on the patient dur-

ing an examination (injury description). Secondly we must assess the injuries described in the previous section in comparison with a scale or reference system (injury classification or rating).

As for the description of injury, we will first review the key components of one of the most frequent sequelae assessed in the study of aesthetic injury: scarring. The following types of scars can be present [7,8]: 1. *Scars in good condition*: they have almost the same tone as the skin, they have no outward or inward texture and thus provide good aesthetic results. 2. *Hypertrophic scars*: they stand out with respect to the dermal plane and can present reddish or pink tones. They are generally not painful, may be itchy and remain in the area of the original scar. 3. *Tattooed scars*: they are the result of foreign objects in the skin. 4. *Dyschromic scars*: they present skin colour alterations or modifications in the scar area and therefore may be hyper or hypo-pigmented. 5. *Cicatricial alopecia*: they occur in areas where there is no hair such as the scalp, beard area and eyebrows, with an absence of hair in the area of the scar. 6. *Retractile scars*: they are habitually secondary to loss of cutaneous substance and cause fibrous retractions and hence deformity, limited movement and limitations of certain functions such as arm mobility when located in the armpits, the forearm if located on the elbow, the knee if in the popliteal fossa, retraction of the eyelids, lips, etc. 7. *Keloid scars*: they present an enormous growth over the surface of the adjacent skin. They have a reddish colour, are painful, itchy and tend to recur following surgical treatment.

As for scars or other injuries that constitute aesthetic injury, we must describe their situation, number, size and shape, colouring, prominence, elasticity and texture, behaviour in detail with regards to nearby structures, changes with body movements in general and adjacent structures in particular and especially in regards to their visibility. In regards to visibility, it is important to describe whether the area or areas are permanently visible or not, and whether they are visible from short or long distances. Visibility is a criterion that was previously given much importance, but this situation has changed in recent years. Hence at present there is no requirement that the injury affects only the face, nor is it necessary for injuries to always be visible, as injuries affecting almost any area of the body, except those hidden by components of our own anatomy such as hair or the inside of the lips, are considered potentially visible, etc. [4,9,10]. Classically, certain regions of the body such as the face and hands have been considered as being of greater importance in terms of attractiveness [3]. A ranking of the regions of the body in their order of importance to aesthetic function are [11]: 1. Face: central part of the forehead, orbital region, nose and lips. 2. Face: sides of the forehead, ears, chin, and submental region. 3. Neck and areas of sexual attraction. 4. Limbs. 5. Trunk.

As for the overall assessment of cosmetic damage, different assessment procedures are available. According to Alonso [3], there are generally two types of methods: descriptive and quantitative.

1. *Descriptive method*: It consists of explaining the degree of ugliness or loss of attractiveness of the injured person based on the complete description of their aesthetic alteration with emphasis on a number of factors: aesthetic condition prior to injuries in terms of shape, size, location, visibility, whether it is static or dynamic aesthetic injury, personal factors (age, gender, occupation), suffering of the injured person because of their aesthetic injury. In addition, the possibility of reconstructive surgery should be reported.
2. *Quantitative methods*: We provide a brief, basic description of the Thierry-Nicourt, Barrot, classical, distance, Rechar and Rodriguez-Hinojal methods. In recent years, other methods have been described: the Bermudez method, the aesthetic prejudice scaled assessment method by Sánchez Rodríguez and Hernández Cueto, a rating system for aesthetic injury from amputations by Puig et al. and finally a method for medical and legal assessment of disfigurement from scarring by Rodes et al.
 - a) *Thierry-Nicourt method*: published in 1982 [12] it consists of ranking aesthetic injury on numeric scale from 1 to 7, depending on the intensity of the aesthetic injury with 1/7 indicating slight injury and 7/7 indicating very severe aesthetic injury [3,7].
 - b) *Barrot method*: based on the Thierry-Nicourt method in which each of the degrees of severity is assigned a percentage from 14% (equivalent to a slight degree, 1/7) to 100% (equivalent to a severe degree 7/7) [3].
 - c) *Classic method*: published in 1972 by GREF (Groupe de Recherches d'Esthétique Faciale), used for the assessment of aesthetic facial injury. based on multiplying two coefficients, one of location and the other of identification. The location coefficient is obtained from a map of the face by aesthetic areas, rated on a scale between 1 and 4 points. The coefficient is calculated by valuing diverse identification characters of the scar: its size, relief, texture, colour and shape. Subsequently, after multiplying the two coefficients, an overall score from 0 to 100 is obtained, which can be converted into one of the seven degrees from the Nicourt Thierry method [3,7].
 - d) *Distance method*: described in 1996 by Rouge et al. [6] and used for the quantification of aesthetic injury to the face. It is based primarily on two viewing distances: 50

cm, or intimate distance, and 3 metres, or social distance. It uses descriptive criteria of injuries and a normative criterion (viewing distance). Examination of the patient is first performed at a distance of 50 cm from the front and profile, and then from a distance of three metres with normal lighting. Cross referencing normative (viewing distance) and descriptive criteria: trace of the scar, colour, relief, deformation and ulceration/distortion enable an assessment table to be carried out on the authors with seven degrees of severity of facial aesthetic injury from very slight to a very severe degree [3,6,7,13].

- e) *Rechard method*: described in 1990 by Dr. Rechard in his PhD thesis. It is based on two types of criteria: subjective and objective. There are three subjective criteria: social and academic factors (possible score of 0 to 0.7), family factors (score of 0 to 0.7) and previous state from the aesthetic or psychic perspective (score of 0 to 0.7). There are six objective criteria based on clinical examination: a) Weight and height of the injured person, from which we can calculate the body surface using a table. b) cm^2 of affected body surface area, measuring both dimensions of the scar or scar area (length and width). In cases of limb amputations, organ mutilations (nose, ear, etc.) or hemiplegia, this method cannot be applied [3]. c) Affected body area (a visibility coefficient is applied). d) Age of subject (following the principle that the younger the affected person, the longer they will have the aesthetic injury). e) Gender of the subject (coefficient of 1.2 for men, 1.4 for women, 1.6 for children, irrespective of gender). f) Visual perception distance, which represents the distance at which the expert can see the lesion (a coefficient is also given). After the criteria have been described, the scores for subjective and objective criteria are summed and the following formula is applied: affected body surface area per thousand, divided by the subject's total body surface area, multiplied by the area involved, added to the result of multiplying the age coefficient by the gender coefficient, and finally the result is added to the visual distance perception coefficient. Finally, the sum of the subjective and objective criteria is calculated, yielding an overall score from 1 to 100, which can also be equated to the assessment system of Act 34/2003 [3,7,13].
- f) *Rodriguez-Hinojal method*: proposed in 1998 [14,15], modelled on the classic or reference method for facial

- scars. The coefficients for identification and location are extrapolated from this method to other parts of the body, ultimately obtaining a disfigurement coefficient. Subsequently the score obtained is applied to the scale of Act 30/1995 of 8 November. Finally, these authors have also proposed a rating system for dynamic aesthetic injury [14].
- g) *Bermúdez method*: described in 2004 [16], it takes into account a scar visibility coefficient, a morphological coefficient and a coefficient for personal characteristics (age, gender and previous aesthetic state). A formula is applied that multiplies the visibility coefficient by the morphology coefficient and the result of simultaneous division of the personal characteristics coefficient. Finally, the author has designed a spread sheet that enables us to calculate the scar's aesthetic injury [16].
- h) *Proposal by Sánchez Rodríguez and Hernández Cueto* (2008) [17], who offer a new assessment criterion that meet all the necessary aspects, eliminating special chapter for quantification of disfigurement using the current scale and including the rest of the measurable consequences in different sections of Table VI. They therefore propose a new rating scale for the evaluation of aesthetic injury to different parts of the body: head and neck, thorax and abdomen, lower limbs and upper limbs. They finally introduced a specific scale for the assessment of scars on different parts of the body, skin pigment changes, grafts and loss of tissue, facial and neck burns [17]. It is hence a totally different assessment system to that used in the current scale.
- i) *Assessment of aesthetic injury from amputations by Puig et al.* (2011). A system fully adapted to the current scale of injuries from road accidents for formal assessment of the severity of injury from amputations of the upper and lower extremities and the complete loss of more than one limb [18].
- j) *Rodes et al. method* (2013), which introduced a proposal for assessing the aesthetic injury caused by a single scar [19]. It is based on three parameters: location, length in cm and the distance from which the scar is visible, which are assessed from a table with a maximum score of 100 points. The resulting score is then carried to another table that assesses the deformity and colour of the scar which are weightings (aggravating or reducing). The final score obtained is divided by two and according to the authors, the assessment of aesthetic injury in accordance with the current scale [5].

4. Evolution of Spanish scales to assess aesthetic injury from road accidents.

In Spain the first scale for injuries resulting from road accidents was published in the Royal Decree of the Spanish Prime Minister of 4 July 1980 amending the Motor Vehicle Modified Liability Insurance Regulation which included the scale as an annex for compensation for permanent disabilities resulting from road accidents [20]. The ruling of June 1, 1989 of the Directorate General of Insurance, in which the scale of compensation for personal injury [21] was approved, was subsequently used to assess injuries from motor vehicle accidents. It was similar to the above scale, quite imperfect, and was broken down in terms of compensation in accordance with the degree of disability (four subtypes), and then into seven categories. It outlined compensable amounts as well as temporary disability compensation for the victim for their habitual work. The sequelae included in each of the seven categories are explained below, ranked from highest to lowest degree of severity. This scale does not consider aesthetic injury per se, and only included sequelae from burns considered serious insofar as they affected “deep organs”. It also assessed different degrees of burns in accordance with the percentage of affected body surface area, ranging from injuries affecting more than 30% of total body surface area to those affecting more than 10%. Less serious categories of keloids and hypertrophic scars are also assessed considering their size and “notably unsightly nature” [21].

Subsequently, the Ministerial Order of March 5, 1991, which for the first time included a special chapter to assess aesthetic injury [22], was published. In this chapter aesthetic injury was assessed in six categories, from mild to significant, with a different scoring system for men and women. The maximum aesthetic injury (considerable) had a minimum score of more than 16 for men and 20 for women, but there was no maximum score. The following was also stated therein: “The score should take into account the age and gender of the person, as well as the impact on their image for their habitual occupation” [22]. It is also stated that the costs of needed restorative plastic surgical interventions should be assessed.

Act 30/1995 was subsequently published on 8 November [23], wherein the first application of its Annex “System for the assessment of the damages caused to people in road accidents” had a binding character. A special section for aesthetic injury was also included, with a six-level scale from mild to significant. The distinction between men and women was eliminated, but the infinite upper limit for considerable aesthetic injury (higher than 20 points) was maintained.

Finally in the new Table VI (Classifications and assessment of sequelae) of the Annex to Act 34/2003 of 4 November [24], ratified by Table VI of Royal Legislative Decree 8/2004 of 29 October [5], many changes were made in regards to aesthetic injury assessment: nine rules of use are established and hence it is made clear for the first time that aesthetic injury must be assessed independently of anatomical or functional considerations [25,26]. In the usage rules there is no gender discrimination as stipulated by Act 30/95. Age is not considered either as it is reflected in Table III of economic valuation of points by sequelae. The occupation of the injured person should not be considered as a weighing factor, since rule nine stipulates that such circumstance, if it occurs, must be assessed through the permanent disability correction factor [5,24]. The names of the two most severe degrees were also changed from “very important” to “fairly important” and from “significant” “highly important” aesthetic injury.. The upper limit was set at 50 points, which as we know had previously been an indeterminate limit. In addition, rule six says that compensation for disfigurement is compatible with the cost of reparative plastic surgery and the impossibility of repair is a factor that intensifies the importance of the injury [7]. It also clarified the most serious injury category, which is highly important in this scale: large burns, large losses of substance and major alterations to facial or body shape [5,24].

We believe that although the system is not perfect, there have been notable improvements in the scales from 1980 to the present. With the help of all the professionals involved we will still be able to improve it for the good of the system and especially for all of our patients.

5. Approach to the assessment of aesthetic injury.

As mentioned throughout this paper, there is nothing simple about the assessment of aesthetic injury. Let us review some basic guidelines that seem indispensable for the purposes of assessment.

Firstly, we must note that the assessment of aesthetic injury always has subjective aspects, which are very difficult to eliminate completely. Thus, authors such as Medina Crespo [9] speak of a triple subjectivity: that of the injured person (own experience), the medical expert, who expresses it objectively without making it impersonal, and that of the judge who makes the definitive assessment. The medical forensic expert or assessor must begin with the history or examination of the injured person, a study of all medical and legal documentation provided, as well as an assessment of the circumstances of the injury and

its evolution, in addition to incidents that occurred during this evolution, a complete physical examination of the patient, assessment of complementary tests carried out and medical and legal monitoring until the injuries are healed or stabilised. Before issuing the relevant health report the previous and contributing causes must be assessed, and the causal relationship between events, injuries and current sequelae must be established to determine the sequelae and measure them and evaluate their potential impact on the activities that the injured person typically engaged in, as well as the possible impact on their social, personal and family life. It is also advisable to take pictures of the injuries at the time of their healing/stabilisation and include them in the report [27].

With regards to aesthetic injury, it is very important that in cases of road accident injuries we scrupulously follow the “nine rules of use” of the special chapter of the scale of Act 34/2003, which describes all sequelae that constitute the aesthetic injury [24] in detail. The following deserve mention: assessments of the aesthetic injury are always comprehensive; the physiological assessment of aesthetic injury must be assessed without any overlap; the aesthetic injury to be assessed is that which existed at the time the injured person became healed; it should be indicated whether the aesthetic sequelae would respond well to reconstructive surgery and its possible result. We must also remember that neither the age nor gender of the injured person are taken into account in measurements of the intensity of aesthetic injury. If work activities are affected, these effects should be assessed through the permanent disability correction factor (Annex IV table).

In accordance with Cobo and Aso [25], we would like to comment on cases where there may be contamination between the assessment of physiological and aesthetic injury. Firstly, aesthetics may be construed as being part of the assessed physiological deficiency. For example, the authors present the case of a mastectomy which on the one hand must be assessed as a “mastectomy” using Table VI and then separately in terms of its aesthetic consequences. Another similar example is the assessment of a full or partial amputation of a limb [18,25]. Another possibility mentioned by the authors and which is used daily practice is the possible neglect of physiological damage in cases where aesthetic injuries have an impact on essential functions, an example being a severe case of depression in response to severe aesthetic injury [25].

Cobo and Aso [25, 28] also propose a “Major Axis of Cosmetic Damage Assessment” to evaluate the effect that aesthetic injury has on people who see it. It would require five levels: a first level of visual witnessing, a second level for the observer’s tendency to view the injury, a third level of visual memories of the injured person, a fourth level consisting of the resulting emotional disturbance and finally the type of emotional response caused by observing the aesthetic injury.

We believe that in many cases the use of any of the valuation methods described in our previous section may be useful to quantify and rate aesthetic injury. Determining which system to use will depend on the usefulness of each method, the practical knowledge available and the type of case to be assessed [3, 27].

Finally, we would like to mention at this point that the assessment of aesthetic injuries is a difficult expert speciality, and medical forensic experts must rely on objective factors to describe and assess them. Adequate and continuous forensic and medical monitoring helps us to better evaluate the case, and a complete physical examination from different planes, angles and distances will always have to be carried out. We must identify the aesthetic injury, quantify it (extent and visibility) and attempt to rate it on a scale. In the most difficult cases, joint observation with another forensic expert will undoubtedly help to reduce our subjectivity.

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Chapter VIII

Medical and legal assessment of non-displaced fractures and cracks

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In the assessment of bodily injury, the medical care received by an injured person is assessed from a legal perspective. Thus, when injuries occur the need of lack thereof of medical or surgical treatment will determine the criminal definition of an action as well as the legal basis for the classification of assault and battery or that of misdemeanour assault.

Thus, in the Penal Code's Act 10/1995 of November 23, 1995, Book II, Title III, on injuries (sections 147-153), specifically Section 147.1, states: "Whoever by any means or procedure causes injury to another person that impairs their physical integrity or their physical or mental health shall be punished with imprisonment for from six months and a day to six years, provided that the injuries require medical or surgical treatment for their cure in addition to basic medical or surgical attention. Mere medical surveillance or monitoring of the progress of the injury is not considered medical treatment. Misdemeanour injuries are covered in Book III, "Misdemeanours and their penalties", Title I, Offenses against persons (sections 617-622); any injury not classed as an offense under this Code is misdemeanour assault.

Case law defines *basic medical attention* as a medical act in which care is provided to a person injured by a violent mechanism to clinically assess the injuries sustained, which may require additional analytical tests involving imaging or functional assessments etc. and to prescribe and administer the necessary preventive or curative treatment.

Moreover, *medical treatment* is therapeutic procedures following injuries and necessary to bring about their improvement or healing. This continuum of medical procedures which are necessary for the injuries to progress properly is what defines the medical procedure as medical treatment.

In order to standardise criteria in medical and forensic practice regarding the consideration of medical care as medical treatment or as

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basic medical attention, a series of rulings by both the Supreme Court and various Sections of the Provincial Court of Tarragona have been used, and from them the following conclusion has been reached: in cases of fractures, even when displacement is not present, the need for medical treatment is considered as necessary and a legal basis exists for felony crime classification. However, in cases of cracks, the medical care received will be considered basic medical attention and the legal status of misdemeanour injury will be applied.

Fracture means a break which affects the entire bone. A *crack* is a break that affects the cortical bone, without restricting any bone fragment.

- Thus, in the Ruling of 5 April 2004, 26/2002, of the Provincial Court of Tarragona, Second Section, in section four of the legal bases, it is determined that the events (broken bones of the nose resulting from assault) constitute *assault and battery* pursuant to and punishable under Section 147 of the Penal Code, which punishes anyone who, by any means or process, causes injury to another person that impairs their physical integrity or their physical or mental health, provided that the injury objectively requires, in addition to basic medical attention, medical or surgical treatment. The Supreme Court has held that the term *medical treatment* applies to a planned healing system or medical scheme prescribed by a qualified medical professional with curative intent; *surgical treatment* is defined as the performance of any medical intervention of this nature (major or minor surgery) which is objectively necessary to repair the human body or to restore or correct any functional or organic disorder caused by injuries. Supreme Court Rulings 898/2002 of 22-5, 1689/2001 of 27-9 and 1556/2001 of 10-9, amongst others, recognise that medical treatment and basic attention are not contradictory expressions, since it is possible that a medical or even surgical treatment is prescribed, designed and implemented in a single treatment procedure. Supreme Court Ruling 908/2002 of 25-5 states that the existence of medical treatment should be considered so that based on legal considerations and in accordance with the statutory classification assigned to it depending on the type of injury: “In this regard, the important distinction between felony and misdemeanour assault is whether the injury is significant. Specifically with respect to the *fracture of nasal bones*, including those without displacement it has been often considered that they require medical treatment, as provided in Supreme Court Rulings 1333/2001 of 3-7 and Supreme Court Ruling 505/2000 of 28-3. Of particular significance is Supreme Court Ruling 195/1999 of 16-2: “Also relevant regarding the need for medical treatment is

whether the injuries not only require appropriate medication, but that during their evolution a subsequent medical examination has been necessary”.

- Supreme Court Ruling 730/2003 of 19-5, Criminal Division II, stated: the following injuries resulted from being struck by a vehicle: distal sacral fracture, pelvic trauma with an lower-left pubic ramus fracture, fracture of the left head of fibula. The healing of these injuries requires medical and surgical treatment and a healing period of 170 days, which will impede the performance of habitual occupations and leave the following sequelae: painful left elbow with limited extension in the last 25-30°, and coccygodynia. The offender was convicted of assault and battery.
- Supreme Court Ruling **1895/2000, of 11 December, Criminal Chamber II**, found: as a result of the assault, injuries occurred producing pain in the left scapular, supracondylar haematoma in right elbow, erosions in the back left hand and *non-displaced fracture of sacrum*, requiring 90 days for healing, and all of which will impede the performance of habitual activities. A single instance of medical assistance is required, which includes and symptomatic treatment and rest, and with a discrete coccygodynia remaining as sequelae. The legal basis for the ruling is based on the classification of the injury as a felony, noting that the injured person underwent two therapeutic treatments, the first aimed at alleviating the symptoms and the second to directly cure the fracture since rest is the only remedy that enables healing of the aforementioned injuries. This regimen was described by a medical practitioner and could be provided at either a medical centre or in the home. Apart from cases of partial immobilisation with the use of bandages, splints or casts, the prescription of bed rest could produce faster healing, improved convalescence with a decrease or elimination of annoying or painful symptoms, without complications or consequences requiring another therapeutic measure. In other words, rest itself can constitute the only acceptable treatment for some ailments, including certain fractures. The fact that rest does not involve the administration of drugs or other more aggressive interventions to the injured person should be irrelevant because it constitutes *lex artis* and consists of medical treatment as defined in relevant case law (Supreme Court Rulings of 02/06/94, 22.44 and 09.02.96, 21.10.97 and 05.26.98). Bone fractures are always considered to be injuries that require medical treatment for healing (rulings from 12 December 1996, 21 October 1997 and 8 June 1999 No. 929/99), since treatment exists from the criminal law perspective, for any subsequent activities concerned healing people under medical prescription, including the administration

of drugs or prescribing behaviours to be followed. This is what occurs in the present case with prolonged immobilisation and rest to heal such a significant injury as a fracture of the sacrum, which took 90 days to heal and required symptomatic treatment and rest.

- Other rulings related to this matter are: Supreme Court Ruling of 6 February 1992, Supreme Court Ruling 1089/99 of 2 July.

1. Nasal cracks/fractures

Fractures that occur most often in the facial region.

The types of nasal fractures are:

- Fracture with lateral displacement without a septal fracture (septum).
- Fracture with lateral displacement with a septal fracture (septum).
- Fracture of nasal pyramid with septal fracture.
- Comminuted fracture (with involvement of different parts).
- Nasal crack.

1.1 Aetiology

Direct trauma: assaults (punching or heading) and falling on hard objects. In road accidents they associated more frequently with other facial fractures.

Fractures with lateral displacement that affect bones and the nasal septum are produced by a medial or lateral impact on the nasal region, and also by anterior-posterior (front) trauma.

Comminuted fractures are produced by high-energy trauma, whether to the front or side.

1.2 Previous condition

After septal deviation, respiratory failure due to rhinitis.

1.3 Clinical

Pain, periorbital oedema and nasal region, swelling, bilateral palpebral ecchymosis, epistaxis, asymmetries (front and profile), rhino deformation (deviation), bony crepitus, breathing disorders.

1.4 Diagnosis

X-Rays. Less frequently, rhinoscopy (septal deviation) and fibre-optic endoscopy.

1.5 Average healing time

From 15 to 35 days for nasal fractures.
For nasal cracks, 14 days.

1.6 Average disability period.

5-10 days. Time to full healing if surgical treatment has been implemented.

1.7 Treatment

Nasal fracture: surgical treatment if reduction or immobilisation with splinting or septoplasty is necessary. Otherwise, medical treatment is prescribed.

Nasal crack: Basic medical attention.

- Ruling 26/2002 of 5-4, of the Tarragona Provincial Court, Second Section (nose fracture from an assault: classified as assault and battery).

1.8 Sequelae

Limited sequelae. It is important to have a photograph taken prior to the injury. Disfigurement due to septal deviation or alteration of the nasal respiratory function. Possibility of septoplasty during the sequelae period.

2. Cracks/fractures of the zygomatic bone

The zygomatic bone, with zygomatic arch, is located in the lateral region of the face and is the second most prevalent fracture occurring in the mid-facial area. Both structures are part of the lateral orbital wall and floor, which is why a non-negligible percentage of these fractures damage the orbital region.

Classification of fractures of the zygomatic bone:

Non-displaced fractures

- Disjunction fractures
- Partial Fractures
- Comminuted fractures
- Zygomatic fractures associated with other bones
- Zygomatic crack

2.1 Aetiology

Multiple-trauma fracture resulting from road or workplace accidents. Assaults with blunt objects.

2.2 Previous condition

Bone malformations of the face.

2.3 Clinical

They are often fractures which result in flattening or collapse of facial contours (pathognomonic). Unilateral facial swelling accompanied by periorbital ecchymosis (eyelid/conjunctival area) of the side affected by the trauma.

The signs of fracture/collapse of zygomatic bone are: stepped shoulder. If the fracture compromises the suborbital canal and causes compression of the infraorbital nerve, anaesthesia or hypoesthesia in the area innervated by that nerve (genial, anterior vestibular mucus membrane, upper dental nerve, and nose ala region) will occur. In the event of collapse of the orbital floor, a palpebral and periorbital emphysema will exist, as also will diplopia.

2.4 Diagnosis

Clinical with confirmation by radiography and special views.

2.5 Average healing time

30-45 days.

2.6 Average disability period.

30-45 days.

2.7 Treatment

Surgical treatment. Fracture reduction is required.

2.8 Sequelae

Few sequelae if the fracture is reduced: deformities, limited masticatory function.

No reduction: crushed cheekbone, anaesthesia or paraesthesia in the infraorbital nerve region, exophthalmos or possibly diplopia.

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3. Non-displaced cracks/fractures of the zygomatic arch

3.1 Aetiology

Patients who have experienced multiple facial traumas from road accidents and assaults.

3.2 Previous condition

Bone malformations of the face – infrequent.

3.3 Clinical

Pain and difficulty opening and closing the mouth.

3.4 Diagnosis

Palpation of bone collapse, usually in the central area, and radiographic confirmation.

3.5 Average healing time

30-45 days for isolated fractures.

3.6 Average disability period.

30-45 days.

3.7 Treatment

Surgical treatment. Reduction through an intraoral approach. Exceptional osteosynthesis.

3.8 Sequelae

Masticatory dysfunction. Limited mouth opening.

4. Rib cracks/fractures

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The ribs are flat bones and are thus formed by a plane of spongy bone between two thin planes of compact bone. They have a flattened and not rounded form. This type of bone has marrow but not a marrow cavity.

Rib fractures can be:

- simple (rare) or multiple (common)
- simple (split into two fragments) or complex (split into more than two fragments)
- compound (breakage of both cortical structures occurs) or incomplete (cracks)
- unilateral or bilateral
- can affect the frontal, side or rear parts of the ribs
- rib cracks

Flail chest: occurs when three or more adjacent ribs are fractured at two or more points resulting in an unstable chest and a paradoxical movement of the affected region of the chest wall.

4.1 Aetiology

Direct or indirect thoracic trauma from road accidents, workplace accidents, home accidents, sports accidents, assaults, accidental falls, precipitation, stab or gunshot wounds.

4.2 Injury mechanism

- Direct trauma or direct compression: rib fracture at the point of impact by grinding the rib arch in the location of action of the traumatic agent.
- Anterior-posterior chest compression: breakage occurs in the lateral area of the rib cage.
- Crushing: the fracture occurs due to stress on the rib arch at a point away from the contact area.

4.3 Previous condition

- Bone diseases such as osteoporosis or bone cancer
- Corticotherapy.

4.4 Clinical

Pain in the fracture area, which is exacerbated with respiratory movements, especially with deep inhalation, movements and when applying pressure to the fracture.

The pain associated with chest wall lesions contributes to the appearance of respiratory failure due to limited airflow and ineffective clearance of secretions by coughing.

4.5 Physical examination

Upon touching, pain in the centre of the fracture, deformity, functional impotence.

Auscultation may be normal or popping can be heard with movements of the chest wall. Secondly, there may be accumulations of tracheobronchial secretions.

4.6 Diagnosis

Chest x-ray: this is a highly effective and rapid diagnostic method, and imaging features will show more or less obvious fracture lines, depending on the degree of separation of the ends of the fracture.

Sometimes rib fractures are not visible in chest x-rays; in these cases, clinical and other complementary tests (TAC) will inform the diagnosis.

4.7 Prognosis

Rib fractures have a good prognosis. However, signs of severity include the following:

- fractures in five or more consecutive ribs
- fracture of the first and second rib (and the possibility of the associated neurovascular injury)
- presence of flail chest

4.8 Treatment

Rib fractures: medical treatment, comprising:

- Rest.
- Control of pain using an appropriate analgesia.

Regional analgesia by intercostal blockage, extrapleural, or with epidural analgesia that have demonstrated effectiveness in improving respiratory mechanics, with a productive cough, conducting efficient incentive spirometry and chest physiotherapy and early mobilisation is favoured.

- Securing the chest with a compressive or elastic bandage.
- Rib fractures can require surgery.

Rib cracks: basic medical attention, consisting of rest and analgesia.

4.9 Healing/wound stabilisation time

- Rib cracks: 21 days.
- Rib fractures: between 30 and 60 days, depending on whether it is a simple or complex rib fracture, with neuralgia or endothoracic complications (60 days).

4.10 Restricted-activity days

During the wound stabilisation period.

4.11 Sequelae

- Pathological consolidation with vicious calluses which cause aesthetic injury due to deformity.
- Presence of osteosynthesis material.
- Intercostal pain.
- Thoracic-mobility disorders.
- Involvement of respiratory function in some cases of complex fractures.

5. Sternal cracks/fractures

It is not common for them to be isolated injury. Generally rib or even to cervical vertebrae or ridges fractures are, as well as visceral pericardium and heart lesions.

5.1 Production mechanism

By direct mechanism, impact on the frontal chest wall (anterior chest trauma):

- in road accidents consisting of head-on crashes with direct impact with the steering wheel or injuries associated with the use of seat belts
- crush injuries from vehicles and workplace accidents
- by direct mechanism: forced flexion or extension of the trunk

5.2 Previous condition

- Bone diseases such as osteoporosis or bone cancer
- Corticotherapy
- Sternal malformations: *pectus carinatum*; *pectus excavatum*

5.3 Clinical

Sternal pain area, aggravated with pressure.

The protrusion of the fracture can be felt on palpation.

5.4 Diagnosis

Anteroposterior chest and profile x-rays: only 15% of sternal fractures are visible on an initial anteroposterior chest x-ray. The diagnosis is usually made from a lateral chest x-ray.

In 40% of cases, the sternal fractures are associated with rib fractures.

in cases of sternal fractures, it is necessary to rule out related lung and or cardiac maladies and in the case of manubrium sterni synchondrosis, related spinal injuries must be ruled out.

5.5 Treatment

Sternal fractures: medical treatment:

- Rest and analgesia.
- Cases of complex sternal fractures are amenable to surgical treatment.

Sternal cracks: basic medical attention

5.6 Healing/wound stabilisation

Average healing time: 45-90 days, depending on whether it is a simple or complex sternal fracture with endothoracic complications.

Sternal cracks: 20-30 days.

5.7 Restricted-activity days

During the wound stabilisation period.

5.8 Sequelae

- Asymmetrical thoracic deformities causing disfigurement.
- Presence of osteosynthesis material - exceptional.
- Respiratory function may be temporarily affected.

6. Cracks/fractures of the pelvis (without fragment displacement)

Tile A, in accordance with the Marvin Tile classification.

- Tile A: Stable (fracture lines, fractures without displacement or bone avulsion at the level of the iliac bones, the ischial tuberosity, pubic symphysis or sacrum and coccyx).
- Tile A1: No damage to the pelvic ring. Spine avulsions or ischial tuberosity.
- Tile A2: fracture of the iliac wing or pelvic engagement ring without displacement.
- Tile A3: transverse fractures of the sacrococcyx without damage to the pelvic ring.

6.1 Aetiology

The most common cause of pelvic fractures are road accidents (vehicle impact, motorbikes, patients thrown from vehicles) and workplace accidents (falls).

Pelvic fractures without displacement often occur to multiple-trauma patients and rarely occur in isolation.

Risk factors

- Bone diseases such as osteoporosis, Paget's disease or bone cancer.
- Use of corticoids.

6.2 Clinical

Bone pain which is contused, intense and localised.

No problems with segment displacement or joint disjunctions.

Often associated with multiple trauma patients.

6.3 Diagnosis

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Anamnesis: History of trauma.

During the examination, look for pain, deformity and loss of function. We will feel for pubic symphysis and branches, iliac crests, sacrum, ischial tuberosity. Mobilised iliac crests are a clear sign of instability.

90% were diagnosed with a simple anteroposterior and oblique x-ray (the fracture may be missed by anteroposterior projections).

The TAC (both simple and 3D) is excellent for viewing the sacro-coccyx complex and to diagnose occult fractures of the sacrum.

6.4 Time for healing or stabilisation of injuries

The prognosis for pelvic fracture without displacement is good, and healing is complete within 45-60 days.

6.5 Restricted-activity days

Complete rest is necessary for a full healing. Restricted-activity days are hence required for injury stabilisation.

6.6 Treatment

Medical treatment: Treatment consists of bed rest and an analgesic regimen.

Unlike unstable fractures, surgical treatment is rarely needed.

6.7 Sequelae

Chronic pain, even in the sitting position.

In unstable injuries there may be:

- Mismatches in limb length (frequent in fractures that have not been successfully reduced, particularly vertical fractures).
- Nerve sequelae may affect the sciatic and obturator nerves.
- Post-traumatic osteoarthritis in cases of cotyloid injuries.

7. Sacral cracks/ fractures

Depending on the fracture line, DENIS classifies sacral fractures as follows:

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- Fracture type 1 or ala: lateral to the neural foramina.
- Type 2 fracture: affects the neural foramina.
- Type 3 or lateral fracture: affecting the area medial to the neural foramen.

In each of them there will be a certain degree of nerve involvement (5-9%, 24% and 56%).

Other authors such as YOUNG make classifications depending on the mechanism of production: lateral compression, compression anteroposterior, vertical shear or combined mechanism.

Roy-Camille speaks of transverse fracture in the upper sacrum due to falls from high places.

7.1 Aetiology

- Simple fractures of the sacrum: due to direct backwards falls or direct impact.
- Complex sacral fractures associated with major trauma and associated with fractures of the spine and pelvis, with dislocations and major musculo-ligamentous injuries.
- Coccyx fracture: infrequent.

All cause highly intense pain that increases with any effort.

When the last spinal roots are injured there may be disturbances of the sphincters and saddle anaesthesia.

7.2 Previous condition

Susceptibility to pain is significant in fractures of the tailbone.

7.3 Clinical

Pain located in the region of the fracture that increases with anteroposterior compression of the pelvis.

7.4 Diagnosis

Anteroposterior x-rays and sacral profile.

7.5 Average healing time

- Simple fractures of the sacrum: 20-25 to 40 days for healing, with restricted activity throughout. Almost never require hospitalisation. In elderly people, pain may persist for 9 weeks.
- Complex sacral or related fractures: 180 days for healing, 20 of hospitalisation and 180 days of restricted activities.

7.6 Treatment

- Simple fractures: bed rest followed by sitting and analgesic drugs for 20-25 days. Medical treatment:
- In isolated transverse fractures with neurological involvement, reduction is attained through surgery. If the fracture involves the cauda equine, the prognosis is worse.
- In displaced fractures of the fragment external reduction measures or tactile measures via the rectal area will be necessary. Surgical intervention and laminectomy may be necessary. Medical and surgical treatment.
- Complex fractures: hospitalisation (medical and surgical treatment).

Supreme Court Ruling 1895/2000 of 11 December.

7.7 Sequelae

Coccyx fracture: long-term coccygodynias which may not improve with local infiltrations.

In simple fractures of the sacrum: anatomical sequelae due to angulations or infrequent deformities.

In complex fractures:

- Anatomical: consolidation defects, vicious calluses, osteosynthesis material and recurrent dislocations.
- Functional: lameness, pelvic tilt, unstable standing, hip joint limitations, resistant pains, ulcers and fistulas.
- Aesthetic: limping, unsteady gait, surgical scar.
- Psychological: in young women, depression.
- Significant pain and alterations in sexual relations due to impossibility of the same during the months of treatment.

8. Non-displaced cracks/fractures. Distal phalanx of the fingers or toes

Non-displaced fractures of the distal phalanx consolidate at 4-5 weeks but can cause imbalances in bending-stretching mechanisms of the fingers. Therefore, joint dynamics are not affected, nor are the strength nor dexterity of fingers affected.

The consolidation process for distal phalanges does not usually result in problems, even distal, because of excellent blood circulation in the fingers.

8.1 Aetiology

They are produced by blunt trauma or pinched fingers. They are common in workplace, home and sport locations.

They are usually associated with comminution and nail injuries. The nail has to be maintained, since it acts as a splint.

8.2 Previous condition

A range of historical factors can also be pathological factors:

- Over the age of 50 with systemic metabolic/rheumatic diseases: the most common is diabetes.
- Chronic treatment involving corticoids.

8.3 Diagnosis

Pain

Nail deformity.

The following factors should be considered:

- Level of professional/sport /leisure activities.
- Location in the dominant hand.
- Degree of involvement of the soft tissues: neurovascular and tendons.
- Septic/underlying disease processes.

8.4 Average healing time

Days for healing/stability of injuries: 30-40 restricted-activity days. If surgical treatment is needed (soft tissue or tendon reconstruction), its progress must be must estimated.

8.5 Treatment

Fractures: medical treatment consists of immobilisation, justified for the sake of pain relief.

Only in the event that the fracture lines affecting the articular condyles and soft tissue (tendon, pulp, nail) have been highly affected, they must receive medical-surgical treatment and finger splint immobilisation for 3-4 weeks and/or reconstructive surgery.

Cracks: basic medical attention.

8.6 Sequelae

In this type of fracture, splints should be used to immobilise for as little time as is necessary and only involving the distal interphalangeal joint, to prevent rigidity that would be considered as sequelae.

The assessment of sequelae must be performed after a minimum injury stability period of 7-8 weeks. an assessment must be made of how soft tissues are affected and the associated complications that could result in sequelae such as the loss or alteration of feeling (loss of pulp tissue), neuromas, rigidities or nail deformities.

8.7 Special cases

Hammer toe (Bush, Mallet Finger fracture): when the fracture has an interarticular line at the base of the distal phalanx with detachment of the extensor tendon (avulsion/traction/pulling fracture), the mechanism of the injury is sudden hyperextension and the healing period is 4-6 weeks. Medical-surgical treatment required with an immobilising splint extension (4-5 weeks), functional rehabilitation and even surgery and treatment in some cases.

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Chapter IX

Assessment of posttraumatic stress disorder as sequelae within the forensic medical field

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1. Introduction

Sequelae associated with road accidents are often a subject of discussion in regards to their economic scope and quantification. In this regard, the legacy of psychological and psychiatric injuries or sequelae pose particular difficulties in terms of diagnosis, and anxiety-related disorders such as Post Traumatic Stress Disorder emerge as benchmark tools of the present study.

We suggest the need to study this pathology mainly due to (a) the casual way in which we believe such symptoms are diagnosed - with little rigour or an increasing tendency to over-diagnose them, and (b) the low levels of compensation provided for the disorder under the scale of Act 34/2003, when chronic forms of the disorder can result in severe limitations on a person's daily activities. In this regard, this section aims to expand knowledge of and diagnostic confidence with regards to Post Traumatic Stress Disorder (henceforth: PTSD), precisely define the circumstances under which it can be diagnosed, and finally consider stabilisation schemes which are economically adapted to the realities of the disorder and the limitations it places on individuals.

2. Post-traumatic stress. General

PTSD is an anxiety disorder which is a pathological response to extreme environmental stress. According to the DSM-IV-TR [1] (APA,

* Group Leader

2000, it can be defined by an individual's exposure to a traumatic event)¹ in which:

- They have experienced, witnessed or have been told about one or more events characterised by death or threats to their safety of that of others (eg wars, terrorist attacks or disasters).
- They have responded with fear, helplessness or intense horror.

The traumatic event is persistently re-experienced in one or more of the following ways:

- Recurrent and intrusive memories of the event which cause distress and include images, thoughts or perceptions.
- Recurring dreams about the event which produce discomfort.
- The person acts out or experiences feelings as if the traumatic event were recurring (eg, a sense of reliving the experience, illusions, hallucinations or flashbacks).
- Severe psychological discomfort when exposed to external cues that symbolise or resemble an aspect of the traumatic event.
- Physiological reactions upon exposure to internal or external cues that symbolise or resemble an aspect of the traumatic event.

Persistent avoidance of stimuli associated with the trauma and numbing of the individual's general responsiveness (not present before the trauma), as indicated by three (or more) of the following symptoms:

- Efforts to avoid thoughts, feelings or conversations associated with the trauma.
- Efforts to avoid activities, places or people that arouse recollections of the trauma.
- Inability to recall an important aspect of the trauma.
- Significant reduction of interest in participating in social or work activities.
- Sense of emotional detachment or alienation from others.
- Restricted emotional life (eg, inability to experience feelings of love).
- Sense of a foreshortened future (eg, feeling hopeless about finding a partner, starting a family, finding a job, living a normal life).

Persistent signs of increased alertness (absent before the trauma), as indicated by two or more of the following symptoms:

- Difficulty falling or staying asleep.
- Irritability or outbursts of anger.
- Difficulty concentrating.
- Altered startle response.

1. Those traumatic situations to which the person is exposed, such as serious accidents, natural disasters, rape, violent crimes, wars, sudden death of a loved one, as well as victims of terrorist attacks and those who, without being victims, observe such events

These alterations last longer than 1 month and cause significant distress or impairment to social relationships, work, or other areas of life that are important to the person.

PTSD, especially when arising from road accidents, includes an assessment of (a) the severity and intensity thereof, and (b) factors of individual susceptibility and vulnerability related to the trauma. It can be acute (one to three months), chronic (more than three months) or delayed onset (appearance up to six months after the trauma, rarely later).² Symptomatic continuity and the adequacy of treatment and psychological/psychiatric follow-up [3: 552-3] must be objectively adjusted to the severity of the case.

According to ICD-10 (WHO[4]), in cases of PTSD unique criteria are proposed for assessing the stressful nature of given situations or events, stipulating that they should have an “exceptionally threatening or catastrophic nature, likely to cause permanent discomfort in virtually any individual.” The ICD-10 diagnosis differs from that proposed in the DSM-IV-TR, as Criterion D of the latter (ie, symptoms due to increased activation) is not considered essential and can be replaced by an inability to recall significant aspects of the trauma. Unlike the DSM-IV-TR, diagnostic criteria for ICD-10 research do not set a minimum duration of symptoms.

3. Causes and contributory causes

In addition to the characteristic symptoms of the disorder, the evaluation must include two key moderating variables: the specific type of stressor and personal *individual factors*[5].

It will therefore be important to distinguish between two concepts: the main cause and contributory causes. The main cause is, in the case of the disorder, the traumatic event while the contributory cause(s) is/are vulnerability to suffer the disorder. These contributory causes differ from the main causes in that each of them, by itself, cannot trigger the disorder, and this only occurs when the main cause appears [6]. In this case previous trauma or pre-existing mental illnesses could be considered as contributory causes.

2. The onset of symptoms may be delayed in time, appearing during a latency or “cold” period during which the person is apparently normal or maladaptive [2].

3.1 Causes. Stressor

The event must be extremely stressful and traumatic and present danger or threats to life or safety. We can rule out, in principle, minor vital events³.

The unique nature⁴ of stressful events means that, to some extent, symptoms differ amongst individuals (in regards to sexual assault, violence, serious domestic violence or serious road accidents). In some cases they can be apparently mild but have posed an obvious risk to the life or safety of the victim. Hence the key factor must be psychopathology, that is, the presence of intense feelings of fear, threat and helplessness, in cases of direct or indirect victims.

The traumatic event must be memorised. There is no traumatic event without subjective perception. When there is loss of consciousness, PTSD does not present.

Predictors of severity [7] in cases of road accidents (from now on, PTSD-RA) include cognitive processing during the accident, negative interpretation of intrusive memories, and alienation or dissociative feelings about others [8]. We can divide them into two groups:

I. Intentional: higher stress capacity

- (a) Sexual assault in adulthood, especially with physical violence
- (b) Sexual abuse in childhood
- (c) Persistent domestic abuse, especially physical
- (d) Terrorism, kidnapping (unlawful detention) and torture
- (e) Violent death of a child
- (f) Road accidents

II. Non-intentional:

- (g) Accidents.
- (h) Natural disasters

3.2 Contributory causes. Vulnerabilities

As for personal factors, it is clear that not all people who have been exposed to a traumatic event develop PTSD. Individual vulnerability is very important, especially in cases in which the traumatic event has a lower objective weight.

3. "It must be of the utmost gravity, for example, a 'life threatening' situation." Seemingly banal or "normal" cases such as neighbourhood fights in premorbid personalities. Highly stressful contexts may leave lasting and severely maladaptive sequelae in some specific cases.

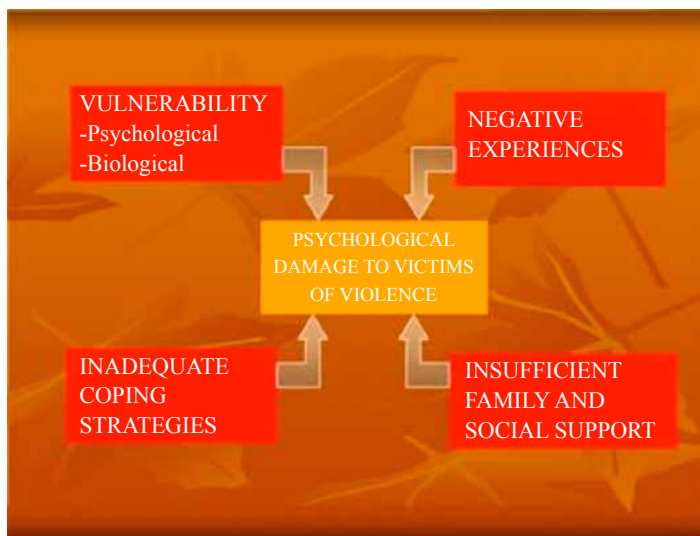
4. Experience the accident and the possible damage, harm or severity to the integrity or life of the person.

Vulnerability is divided into *psychological* and *biological*:

3.2.1 Psychological vulnerability

Psychological vulnerability includes general factors (low intelligence, emotional instability, low self-esteem, inability to adapt to changes in general (low resilience), history of family or personal problems⁵, low levels of social/family support⁶ (more likely to suffer disorder, more severe presentations, and longer healing possible), psychiatric history, dependence on alcohol and toxic substances [10], borderline personality traits, paranoid, antisocial or dependent [11,12], genetic vulnerability to psychiatric illness, or perception of an external (natural causes) rather than internal (human causes) *locus of control*⁷.

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5. Life events, with particular interest on the infant stage, assessing: other violent crimes, cumulative stress, family psychiatric history, depression, divorce, teenage parents, violent death of a child or domestic violence.

6. Síndrome del estrés postraumático. Enrique Fernández Rodríguez. Estudios Jurídicos. Plan de formación continuada para médicos forenses, n.º 1, 2003, pag. 683-696. Revisión de casos en la Clínica Médico Forense de Madrid; 1999-2000.

7. Ie, interpret control of the traumatic or stressful situation as alien to oneself (and therefore not controllable by one's will), thereby applying inadequate coping or maladaptive response to stress (persistent feelings of guilt or anger, substance abuse, or rentism, etc.).

Psychological vulnerability

- Low intelligence
- Emotional instability, low self-esteem
- Inability to adapt to changes in general (low resilience)
- History of family or personal problems: 1) other violent crimes; 2) cumulative stress; 3) family history of psychiatric disorders; 4) parental divorce during adolescence; 5) violent death of a child; 6) VIDO
- Poor social support (more likely to have the disorder in its more severe presentations and with longer healing periods)
- Psychiatric history
- Dependence on alcohol and toxic substances
- Borderline personality traits, paranoid, dependent or antisocial
- Genetic vulnerability to psychiatric illness
- Perception of an external (natural causes) rather than internal (human caused) locus of control; ie control of the situation is interpreted as alien to oneself (and therefore not controllable by one's will), hence inadequate coping strategies or maladaptive responses to stress are applied (persistent feelings of guilt or anger, substance abuse, or rentism)

Specific factors

- *Road accidents.* Negative effects 1-3 months after the trauma increase the risk of PTSD from road accidents [13,14], as do post-traumatic amnesia and a slight loss of consciousness [15]. Extraversion (insight damage) and neuroticism (anxiety), cognitive factors and subjective characteristics related to the trauma (trauma assessment biased by the victim) predict severity of PTSD (AT) and symptoms (intrusion, avoidance and high arousal, respectively). In addition, maladaptive coping styles that do not enable the functional containment of stress also maintain symptoms [16]. Elements such as cognitive intrusions are associated with an increased risk of suicide amongst road accident survivors [17].
- By *gender*, a greater likelihood of symptoms in women classified in Cluster C (avoidance, drowsiness) and Cluster D (arousal) is postulated; dissociative peritraumatic symptoms at the time of the accident increase the risk of PTSD M>H PTSD [18,19]. The risk of acute PTSD (AT) is doubled in women compared to men [20], but there is no risk inequality for chronic PTSD. The history of serious anxiety disorders is a good predictor of both acute and chronic PTSD.

- The prevalence of PTSD (AT) ranges, in *children and adults* respectively, from 23% to 25% [21] at the age of three months, and 11% to 18% [22] at 6 months. A prevalence of PTSD (AT) ranging from 15% to 10-46% [23]. is postulated worldwide. Echeburúa estimates sexual assault in childhood (6-7, 10-12 years of age) with acute PTSD-emotional symptoms at over 70%, while the figure for chronic symptoms is 30% [5] (14%).
- The prevalence of PTSD triggered by *serious assault* is 20%, and that of acute stress disorder is 19% [24]. Increases in the prevalence of PTSD are noted as follows: in cases of life-threatening injuries [25], PTSD is higher (42%) compared to situations of life-threatening events without injury (34-38%), and these in turn are more frequent than traumas resulting from events without actual threats (9-15%) [26]. Other authors dismiss the importance of previous trauma with respect to the occurrence of PTSD (AT).
- *Rape*. one third of rape victims have symptoms associated with PTSD, with 19% (USA) of them experiencing persistent symptoms throughout their lifetimes [27].
- *Domestic violence*. Two factors, chronicity and the fact that the perpetrator is within the victim's trusted environment. 46% of women abused by their partners with chronic PTSD presented a more severe form of the disorder if they had been sexually coerced (humiliating and degrading acts), if the abuse was more recent (immediacy), if coexistence with the aggressor continued and in the absence of social support systems. For the purpose of proper validity and diagnostic reliability, external data from the patient must be obtained via VIDO, avoiding minimising or maximising symptoms that they may hide or be confused with PTSD, when they are caused by acute stress, adjustment disorders, depression with anxiety, or simulation.
- *Children*. In children and adolescents [28], reported symptoms include repetitive dreams of the event, nightmares with monsters, stomach-aches, headaches and regressive behaviours. They are characteristic of "traumatic play" which consists of acting out repeatedly the traumatic event through play activities. Older children engage in "re-enactment", which consists of imaginary acts of revenge or intervention. Sexual behaviours, the use of mind-altering substances, acts of crime are common amongst children exposed to potentially deadly sporadic events (war, kidnapping, illness or severe burns, bone marrow transplants or disasters) or continuous trauma (sexual or physical abuse). Amongst child victims of sexual abuse or witnesses to the same, high levels of psychiatric comorbidity have been observed.

3.2.2 *Biological vulnerability*

- Hyperactivity in the sympathetic nervous system (SNS) through endorphin and noradrenergic neurotransmitters affects and modulates structures such as the hippocampus, the hypothalamus, the limbic system, the cerebellum and the ascending reticular system in chronic conditions affecting working memory, sleep-wake functions, concentration, hypersensitivity, or physical and cognitive fatigue falter when the system fails due to overloading. In PTSD chronic deregulation of the hypothalamic-pituitary-adrenal axis [29] occurs. Its characteristics are: lower levels of urine cortisol concentration, increased noradrenaline cortisol ratio in urine, flattening of the adrenocorticotrophic hormone response in the corticotropin releasing factor, greater suppression of cortisol after the administration of dexamethasone, which implies a model of hypersensitivity to negative feedback from HHS in PTSD.
- The *theory of learned helplessness* (Seligman), which involves the blocking of coping mechanisms due to unpredictability, includes a neurological substrate of conscious feelings found in the cerebral cortex (cingulate gyrus, parahippocampal gyru feeling and emotion perception and hippocampal formation, and orbitofrontal cortex) and emotional states affected by a group of peripheral, endocrine and skeletal motor system responses, which involve subcortical structures (amygdala nucleus, hypothalamus, and brainstem). The neurobiological response to acute stress involves the release of various stress-related hormones that enable the body to respond in an adaptive way. In PTSD, because of a serious or repeated trauma, the response is poorly regulated and a chronic autonomic hyperactivity sets in, manifested by positive symptoms of the disorder, ie, hyper-arousal, intrusive memories and negative symptoms [30] such as behavioural blunting or inhibition.

4. Clinical and psychometric assessment

The assessment of PTSD requires an accurate case history, preferably not planned, with the aim of collecting the following data:

- General: medical history and symptoms associated with DSM criteria
- Specific: assess the existence of the aforementioned vulnerabilities

We recommend not starting the examination focussed on the traumatic event, but rather seeking out other medical data to create a proper transfer and finally treating the cause of the disorder.

To complement the clinical examination, we selected a psychometric instruments that could give us more data to assess the personality of the person and their active symptoms. The tests give us information about your personality, your current symptoms and also show the response style to the test (desirable, magnifier, etc.) they are: MCMI-III and MMPI-2 [31].

In addition, there are a number of specific scales to assess symptoms which have the limitation of only having minimum reliability and validity ratings (while others are completely lacking in them) and are easily manipulated.

These scales could be used, but would have to be analysed very carefully with great care. If you decide to use any of the scales, it is recommended (a) that the person take the test and for each of the items for which they have answered positively questions should be asked and examples requested, or (b) the person should be asked to write all of the changes or situations perceived as bothersome on a blank piece of paper. The professional will then try to supplement the test with this information.

- To assess general symptoms, SCL-90 (Derogatis, 1977; TEA, 2002; provides symptoms from the previous week).
- To specifically assess PTSD: symptom severity scale (Echeburúa et al., 1997), Traumatic Experiences Questionnaire (TQ, Davidson, 1990) and Davidson Trauma Scale (DTS, Davidson, 1997 [32]).
- To assess anxiety: Hamilton Scale or STAI (State-Trait Anxiety Inventory).
- To assess depression: BDI, Beck or HRS.

Psychometric vulnerability (II)

- To assess general symptoms: **SCL-90** (Derogatis, 1977; TEA, 2002), which provides symptoms from the previous week
- To specifically assess PTSD: **symptom severity scale** (Echeburúa et al., 1997), **traumatic experiences questionnaire** (TQ; Davidson, 1990) and the **Davidson Trauma Scale** (TQ; Davidson, 1997)
- To assess anxiety: **Hamilton scale** or **STAI** (State-Trait Anxiety Inventory)
- To assess depression: **BDI, Beck or HRS**

Some thoughts from the perspective of personal injury assessment in France [33], which has long been concise and clearly protocolled, suggest:

- Starting with a biographical study, with the unique story behind the traumatic event, very important in terms of the existential and clinical elements, medical history and personality traits.
- Recollection of the traumatic event for the victim is useful in assessing the symptoms, the emotional intensity during the explanation and memory retention of the event.
- It takes a classic psychiatric examination, with special attention to signs of PTSD such as anhedonia, the acceleration of verbal expression, under-excitation.
- To determine the absence of a previous psychiatric condition, objective [34]: data are needed: hospitalisation history, healing in covalence centres, specialist consultations or psychiatric treatment and the scrupulous reconstruction of the biography, with searches for hospitalisations or releases. The type of medication and duration must be determined: antidepressants and tranquilisers are often prescribed for trivial reasons. The most delicate aspect is the personality of the individual. Well defined pathologies do not present diagnostic problems (psychosis, mental retardation, etc.).
- Assessment of the duration of temporary disability: assess the intensity of treatment with psychotropic medicines with their effects of drowsiness and slow movements, the inability to leave home due to a phobia, panic assaults, stress, difficulty concentrating and anxiety symptoms. These are all elements to consider when evaluating a temporary disability, with *stabilisation* at 18 months at the most.
- Experts in bodily injury call a psychiatrist in cases of overstimulation, or unclear evolution.
- In regards to an accurate diagnosis of *sinistrosis*, two tips are provided: do not adopt a contradictory attitude with a patient with compensation neurosis and set a mandatory date for stabilisation or consolidation. Note that compensation neurosis is a disease; it is not the same as a simulator.

5. Differential diagnosis

- 5.1. Acute stress reaction: appears during the first month after trauma and lasts 2-30 days.
- 5.2. Adjustment disorder: if it does not meet criteria for PTSD, or the PTSD symptomatology presents in conjunction with triggers of no great importance, with a mild psychiatric pathology. It can be acute (up to 6 months) and chronic thereafter.
- 5.3. Simulation in the presence of financial compensation, forensic selections or examinations.

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Differential diagnosis

- **Adjustment disorder:** the severity of the stressor in an adaptive disorder is differentiated in magnitude from that which is observed in PTSD, as the stressor in an adaptive disorder is usually less severe is among common human experiences. However, it is not always possible to predict the relationship between the severity of the stressor and the type of symptomatology
- **Acute stress:** DSM-IV, in recognition of the spectrum of disorders that arise after severe stress, added acute stress disorder to the anxiety disorder. It is similar to PTSD both in regards to the traumatic event which triggered symptoms, but is self-limiting in time, since it can appear up to a month after the traumatic event. It is a highly reliable variable for making prognoses regarding the development of PTSD
- **Simulation**

6. Treatment

Psychiatric treatment of PTSD is based on two pillars:

1. *Pharmacotherapy:* Mainly SSRI for at least a year, and anxiolytics when the intrusive re-experiencing or hyper-arousal are prevalent.
2. *Psychotherapy:* when avoidance behaviours or numbing are prevalent. Above all, cognitive-behavioural or social cognitive therapies, using stress management techniques that are more effective in the short term and event exposure techniques, which are more durable in its effects, or reformulation of cognitive distortions.

In untreated patients, about 30% recover completely [35], 40% continue with mild symptoms, 20% continue with moderate symptoms and 10% remain unchanged or worsen.

7. Prognosis

Indicators of a good prognosis are the rapid onset of symptoms, their short duration (less than six months), a proper premorbid state, appropriate and early treatment, strong social support and the absence of other psychiatric medical or disorders related to substance abuse.

In general, the vulnerability factors mentioned above will adversely affect the prognosis.

Children have more difficulties overcoming the disorder (because they do not possess adequate mechanisms to cope with the trauma experience) as do the elderly (because they present less flexible coping mechanisms, in addition to somatic illnesses such as heart failure or stroke).

8. Case law⁸

After review of the rulings related to Posttraumatic Stress Disorder (PTSD), the Supreme Court of the National High Court and the High Courts of Justice, the following concepts seem relevant in regards to medical and legal practice:

- The Supreme Court case law on Posttraumatic Stress Disorder considers bodily injury as follows: “Any disruption to the integrity of the human body, in both physical and psychological aspects, is considered as an injury” (STS, 516/2002, 30 May).
- Posttraumatic Stress Disorder is also considered amongst the psychological consequences of violent assault which produce psychiatric disorders: “The legislator, though no mental harm has been required of the victim in regards to the offense (sexual assault), considered that as a rule the crime would have to produce them” (STS, 1590/1999, 13 November, on sexual assault).

8. Sources:

- Database Westlaw.es, Editorial Aranzadi.
 - Database laleydigital.es (Wolters Kluwer España pub.).

The legal issue is whether Posttraumatic Stress Disorder is assault and battery with autonomy as a violent crime or whether it is included in the typology of violent crime if the psychic or emotional consequences of psychic disturbance that psychology and psychiatry classify under various names such as PTSD, adaptive disorders such as depression, harrowing, anxiety, etc., are a consequence of the aggression that is part of a violent crime. Case law fluctuates, although most of the time it is favourable to subsume assault and battery within the violent crime being tried.

Amongst the rulings of the High Courts, most of those revised are issued in the social courts, in some cases via contentious-administrative proceedings, and most of the revised rulings concern claims for work disability, being denied or granted to a lesser degree for various pathologies including PTSD from road or workplace accidents, without specifying the origins of the disorder. Most rulings dismiss the claims.

Other revised cases include those dealing with workplace harassment with claims of post-traumatic stress disorder, where the revised rulings dismissed the claims.

From revisions of court rulings, the importance of accurate diagnoses of PTSD, injury or sequelae can be deduced, in any legal field, as beyond the compensation value, this diagnosis is understood as being related to the severity of causal factor (which is as it should be) then sometimes used in different processes or in a “wilful” manner”.

(Also taken into account were the following Supreme Court Rulings: STS 1400/2005 of 23 November on sexual assault; 629/2008 of 10 October on kidnapping, offenses against moral integrity and injuries)

9. Medical and legal conclusions

- 9.1. PTSD, especially in road accidents, includes the assessment of (a) severity and intensity thereof, and (b) factors of individual susceptibility and vulnerability related to the trauma. It can be acute (one to three months), chronic (more than three months) or delayed onset (appearance up to six months after the trauma, rarely later⁹). Symptomatic continuity and the adequacy of treatment and psychological/psychiatric follow-up must be objectively adjusted to the severity of the case [3: 552-3].

⁹ The onset of symptoms may be delayed in time, appearing following a latency or “cold” period in which the person is apparently normal or maladaptive [2].

9.2. Under criterion (a), the event must be potentially or factually serious, unrelated to usual human experience, and that undermines the integrity (may include physical and/or psychological) aspects of the person. The experience of trauma may be direct (personal experience) or referred (induction). There is no direct relationship between abnormally intense trauma and being subjected to PTSD. However, seemingly minor traumas¹⁰ may lead to chronic PTSD.

Medico-legal conclusions (I)

- PTSD is a psychiatric disorder associated with direct or indirect experience with a stressful event
- It may be acute, chronic (lasting more than three months) or delayed onset (appearance up to six months after the trauma, rarely later)
- Assessment should include two key variables:
 - The particular type of stressor (cause)
 - Personal individual factors (contributory causes) (external data should always be collected)
- Difficulty in establishing a causal relationship:
 - Because of inherent difficulties associated with psychic injuries
 - Due to lack of expert familiarity with the person's previous state
- It is necessary to obtain a complete medical history and perform psychometric examinations

9.3. The criterion *b* must be assessed in each case, with attention to individual and contextual factors for pre-post-trauma, which will limit the prognosis and severity of their impact.

9.4. In addition to the characteristic symptoms of the disorder, the evaluation must include two key modulator variables: the specific type of stressor and personal individual factors. It will therefore be important to distinguish between two concepts: the main cause and contributory causes. The main cause is, in the case of the disorder, the traumatic event while the contributory cause(s) is/are vulnerability to suffer the disorder. These contributory causes differ from the main causes in that each

10. While the severity and intensity of trauma carries a proportionally greater risk of PTSD, the subjective-personal experience of the same is what determines the magnitude and consequences for each potential victim.

of them, by itself, cannot trigger the disorder, and this only occurs when the main cause appears. In this case previous trauma or pre-existing mental illnesses could be considered as contributory causes.

- 9.5. Establishing a causal relationship [36: 145] is more difficult than for other types of psychic injuries and because of general unfamiliarity on the part of experts of the subject's previous state, but it would be necessary to describe to the extent possible the existence of vulnerability factors.

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Medico-legal conclusions (II)

- Medico-legal symptom stabilisation period: DSM-IV-TR stipulates three months as the disorder's chronicity period, bearing in mind that it is a clinical, not a forensic or expert's manual
- Assessment of the duration of temporary disability: assessment of the intensity of treatment with psychotropic medicines with their effects of drowsiness and slow movements; the inability to leave home due to a phobia, panic assaults, stress, difficulty concentrating (anxiety symptoms) are elements to consider when evaluating a temporary disability
- Experts in bodily injury call a psychiatrist in cases of overstimulation, or unclear evolution
- From a medico-legal perspective, the type of treatment is considered as a medical treatment

- 9.6. The treatment applied must be considered as a "medical treatment" from the medical and legal perspective, and not merely a symptomatic treatment, if it is adequate and its monitoring is accredited, since under such conditions the prognosis improves substantially.

- 9.7. With regard to the medical and legal stabilisation period for the symptoms and consideration of consequences, an extension of the follow-up of between 3 and 6 months is recommended, with the healing period nonetheless being stabilised at 3 months based on the chronicity criterion of the DSM-IV-TR symptoms. Factors such as emotional immaturity, the severity of the stressor, the lack of treatment or previous psychiatric disorders will worsen the prognosis.

Medico-legal conclusions (III)

- Scale: possibility of assessing PTSD as a “worsening or destabilisation of other mental disorders” in premorbid unstable personalities (1-10 points)
- Consider secondary victimisation (delayed remission period for anxiety symptoms due to added anxiety during legal proceedings, the wait for trial and attending the same)
- In the event of an accurate sinistrosis diagnosis, the following is recommended: a) not to actively confront a patient with compensation neurosis b) set a mandatory stabilisation or consolidation date; it should be noted that compensation neurosis is a disease, not a simulation...



- 9.8. The sequelae with this diagnosis would be appropriate if there is a poor response to treatment with persistent severe symptoms (severe discomfort) and especially with regards to any change in personal functioning (work, family relationships, social, leisure, etc.). In the clinical setting, an alternative that is widely used in cases in which the stressful event is not very significant or when the clinical diagnosis of adjustment disorder is milder is the adaptive disorder diagnosis. But in our case, at least in regards issues of the road accident scale, the score charts, paradoxically, awards 1 to 5 points for neurotic disorders (where in theory the adjustment disorder would be placed) and only 1-3 points for PTSD.
- 9.9. We would have to consider a specific form of secondary victimisation 28, the court-evaluating system, considering in the case of PTSD-AT (and the rest of the so-called “neurotic disorders”) between 1-3 points on the road accident scale. The severity and chronicity of the sequelae could, in some cases, make even personal, social, occupational, or interpersonal rehabilitation impossible, and classify it within the post-concussion syndrome sequelae (5-15 points) with concussion interpreted in a “broad” sense. In premorbid unstable personalities, we propose a similar sequelae similar to “worsening

or destabilisation of other mental disorders” (1-10 points). In addition, secondary victimisation lengthened the remission period of anxiety symptoms added during the legal proceedings, the wait for trial and attending the same.

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Chapter X

Adjustment Disorder in Bodily Injury Assessment

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1. Introduction

Adjustment disorders as such are not covered by the classification and assessment of *sequelae* in Act 34/2003.

From a clinical standpoint this diagnosis is made very often but brings with it a series of conceptual and operational problems. On the one hand it relates stress with psychopathology, but then on the other there are no operational criteria for diagnosis, so this category is sometimes used as a catchall for cases which do not meet the criteria for other disorders.

2. Definition

According to the current classification systems (DSM-IV-TR and ICD-10), adjustment disorders are characterised by the presence of emotional or behavioural symptoms, always in response to a stressful event. We might say that this is a minor psychiatric disorder, falling between normality and mental disorders in their own right, as is the case of affective disorders and anxiety disorders.

The DSM-IV-TR Manual classifies six subtypes by predominant symptoms. The ICD-10 classification considers seven subtypes, some comparable to DSM subtypes and others slightly different, as shown in the table below:

* Group Leader

Table 2.1. Subtypes of adjustment disorders

DSM-IV-TR	ICD-10
F43.20 Adjustment disorder with depressed mood (309.0)	Brief depressive reaction (F43.20)
F43.28 Adjustment disorder with anxiety (309.24)	
F43.22 Adjustment disorder with mixed anxiety and depressed mood (309.28)	Mixed anxiety and depression reaction (F43.22)
F43.24 Adjustment disorder with disturbance of conduct (309.3)	Predominantly antisocial disorders (F43.24)
F43.25 Adjustment disorder with mixed disturbance of emotions and conduct (309.4)	With mixed emotion and antisocial disturbance (F43.25)
F43.9 Adjustment disorder, unspecified (309.9)	
	Prolonged depressive reaction (F43.21)
	Predominantly disturbance of other emotions (F43.23)
	Other adjustment disorder with specified predominant symptoms (F43.28)

The necessary conditions for establishing a diagnosis of adjustment disorder are:

- a) The presence of an identifiable stressful event.
- b) Emotional (depression, anxiety), behavioural, mixed (emotional and behavioural) and other disturbances entail greater than expected discomfort in response to the stressor, and
- c) Emotional, behavioural, mixed and other disturbances produce significant impairment in social or occupational (or academic) activity.

3. Adaptive disorder case law

We have reviewed the rulings of Provincial Courts and High Courts which contain adjustment disorder as a sequela of an accident.

One of the main features of these rulings is the absence of uniformity in relation to different parameters of forensic medical importance.

For one thing, we found different names for the adaptive disorder that do not dovetail with the medical terminology established by the DSM-IV-TR, such as for instance Ruling 890/2005 of 27 December of Alicante Provincial Court which refers to “adaptive personality disorder”.

Nor do they establish precisely whether treatment has to be prescribed by primary care doctors or specialists in Psychiatry. Thus for example there is Ruling 249/2006 of 11 September by Zaragoza Provincial Court where the treatment has been prescribed by a psychiatry specialist, whereas Ruling 91/2006 of 3 October by Murcia Provincial Court simply states that psychiatric treatment was required without specifying the professional who has monitored the evolution of the patient, and Ruling 957/2004 of 20 September by the Murcia High Court is much less specific in that it only mentions that there has been medical treatment.

As for the scoring of adjustment disorder as a sequela, we have found disparate data such as the score of 1 point in Ruling 91/2006 of 3 October by Murcia Provincial Court which classified it with “other neurotic disorders”, the 7 sequela points with no explanation in Ruling 249/2006 of 11 September by Zaragoza Provincial Court, and the 15 points in Ruling 66/2006 of 13 March by Guipúzcoa Provincial Court which specifies an “adaptive disorder due to severe chronic stress assimilated by analogy to severe posttraumatic neurosis”.

The only point on which there seems to be consistency of approach is that adjustment disorder, except temporary disability (TD), is not grounds for any other degree of permanent disability as set out in Ruling 942/2005 of 14 September by Murcia High Court, Ruling 890/2005 of 27 December by Alicante Provincial Court, Ruling 373/02006 of 13 April by Murcia High Court, Ruling 126/2003 of 27 January by Murcia High Court, Ruling 895/2004 of 6 September and Ruling 957/2004 of 20 September by Murcia High Court.

4. Controversial issues

According to the DSM-IV-TR, symptoms appear within 3 months from the stressful event. The ICD-10 argues that the period of onset of symptoms is the following first month. Irrespective of these time periods established by consensus, the issue of up to when the stressful event can be considered has to be addressed. The legal process often perpetuates the stressful experience, which suggests to us that the stressful event is maintained for the duration of the legal proceedings.

The DSM-IV-TR considers the disorder as acute if adaptive clinical signs last longer than 6 months after the stressor ceases. If the clinical signs continue after this time, it is considered a chronic disorder. By contrast, the ICD-10 does not address the acute-chronic subdivision but specifies that a prolonged depressive reaction could be extended up to two years.

The DSM-IV-TR refers to the prior existence of a stressor factor without specifying at any time the intensity and characteristics of the stressful event. The ICD-10 describes these disorders as reactions occurring in the period of adaptation to a significant biographical change or a stressful life event, but also notes that individual predisposition or vulnerability plays a more important role in these than in other stress disorders.

There are no operational diagnostic criteria based on specific symptoms or a diagnostic threshold to differentiate them from other pathologies. In fact, the DSM-IV considered the possibility of including them in other chapters (the depressive form in mood disorders, the anxious form in anxiety disorders, etc.) to treat minor forms of these pathologies. It also thought about grouping adjustment disorders with PTSD in a separate chapter but finally decided to keep the “adjustment disorder” category due to lack of empirical evidence to justify the change. The ICD-10, however, has put them all in the same section along with the response to acute stress and PTSD in the “Neurotic, stress-related and somatoform” chapter.

5. Diagnosis

There are no specific symptoms to provide a diagnosis. The diagnosis may be considered in the case of people who when confronted with stressful life situations do not react with a mental illness but also not normally. The evaluator needs to confirm the existence of a “disproportionate” discomfort.

To establish the diagnosis the following should be assessed:

- *Establishing a causal relationship with the stressor*; the latter may be a one-off event or situations sustained over time which may be single or multiple expected or unexpected life changes experienced by one person or shared (this does not mean that everyone who has undergone the same stressful experience will develop an adjustment disorder).
- *Assessment of the level of involvement* by comparing the reaction of the person concerned with the average in similar circumstances.
- *Ruling out other mental disorders*.
- *Establishing an approximate length for the process*.

6. Differential diagnosis

Firstly, adjustment disorders must be distinguished from non-pathological emotional reactions to stress which do not constitute a diagnosis, do not require treatment and do not cause higher than expected psychological distress or functional impairment.

An adjustment disorder diagnosis will not be made when the clinical symptoms are compatible with a diagnosis of:

- Mood disorders (depressive disorders).
- Anxiety disorders (generalised anxiety disorder, panic disorder with/without agoraphobia).
- Bereavement.
- Posttraumatic stress disorder (PTSD).
- Personality disorder.

It is possible to diagnose an adjustment disorder with prior psychiatric illness when there is no overlap of symptoms. Thus a person with obsessive-compulsive disorder presenting sadness and a tendency to cry in response to a stressor can be diagnosed as having an adjustment disorder but cannot be considered to be suffering from an adjustment disorder if what they present is the increased anxiety, obsessive ideation, rituals, etc. characteristic of their underlying disease process. Another example of adjustment disorder with underlying pathology would be a person with a paranoid personality disorder who following a stressful situation develops a clinical picture of anxiety and depression. In this case adjustment disorder may be diagnosed, which would not be possible if the reaction to the stressor was an aggravation of their personality traits (suspicion, mistrust).

It is important to identify simulation cases where secondary gain or external benefits (compensation, avoiding legal problems, etc.) are identified that may entail the presence of an adjustment disorder.

Table 6.1. Differential diagnosis

DIAGNOSIS PREVALENT DIAGNOSTICS	Adjustment D.	Depressive D.	Anxiety D.	Bereavement	PTSD
Severe traumatic/ stressful event	+	+ o -	+ o -	+	+++
Depressed mood	According to subtype	+++	+ o -	++	+
Anxiety	According to subtype	+	+++ Psychic and somatic	+ o -	+
Re-experiencing the stressor	-	-	-	-	+++
Dissociative symptoms	-	-	-	-	+++
Avoidance behaviours related to the stressor	+ o -	-	++	-	+++
Arousal	+ o -	-	++	-	+++
Affective blunting	+ o -	+ o -	+ o -	+ o -	+++

7. Treatment

Due to the characteristics of this disorder, the right place for treatment is the GP's surgery. The literature refers to brief psychotherapy as the treatment of choice, but the reality is that it often requires psychopharmacological medical treatment (anxiolytics for anxiety, antidepressants for depression, etc.) for a period of a few months.

They are considered as needing specialised treatment with referral to the psychiatrist when:

- There is previous psychiatric pathology complicating the course of the process: major depression, psychosis, suicidality, bipolar disorder, personality disorder, dysthymic disorder, etc.
- It evolves towards chronicity with no clinical improvement at 6 months.
- Symptoms persist 6 months after the stressor has disappeared.

8. Chronicity factors

8.1 Personality disorders or maladaptive personality traits.

Personality disorders undermine the resilience of people in stressful situations, particularly Cluster C, i.e., anxious and fearful people. Dependent personality disorder is most often associated with adjustment disorders that have become chronic. They are people characterised by an inability to make decisions and who delegate responsibility for their actions to others. Avoidant and schizoid personalities are also frequent who tend to isolate themselves, albeit for different reasons. Obsessive-compulsive personality disorders characterised by doubt and indecision may have a mental block when faced with a stressful situation.

8.2 Environmental factors

Environmental factors that favour the persistence of symptoms due to the persistence of the stressor or the appearance of a new stressor

8.3 Organic diseases, drugs or medicines

Organic diseases, drugs or medicines that can cause anxiety, depression, etc.

9. Medico-legal evaluation

Adjustment disorders have a tendency to symptomatic remission for full recovery without sequelae.

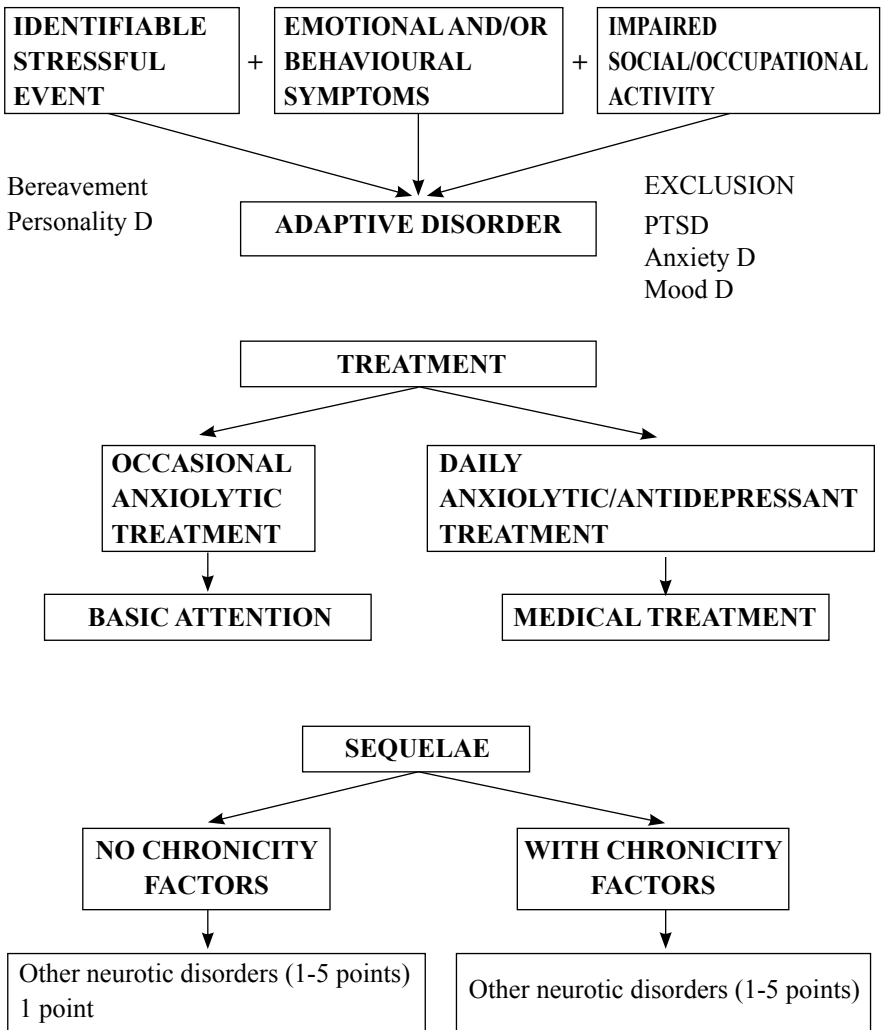
If residual or isolated symptoms are present but progress toward restoration and not chronicity is expected when the forensic medical health report is issued, it will be included in “Other neurotic disorders,

1-5 points” in Table VI of Act 34/2003, of 4 November, with a low score. If there is a chronic course, it will be regarded as a sequela and included in “Other neurotic disorders, 1-5 points’ in Table VI of Act 34/2003, of 4 November.

Days for healing/stabilisation and any lost work days will be taken into account.

If continued daily treatment with anxiolytics and/or antidepressants has been required, this is considered treatment. If, by contrast, only occasional anxiolytic medication has been required, it will be considered basic medical attention.

Table 9.1. Decision tree for diagnosis, treatment and assessment of sequelae



10. Conclusions

In the light of the above we contend that adjustment disorders:

- Are emotional and/or behavioural reactions to a stressful life event.
- Do not have their own or specific symptoms, the most common symptoms being depression, anxiety and, less frequently, behavioural disturbance.
- Are less serious disorders with a good prognosis and progress to full remission once the stressor that produced them has disappeared.
- Previous personality plays a very important role in the development of adaptive disorders as an individual vulnerability factor which in some cases can determine the chronicity of the psychopathological process.
- From a medico-legal point of view they require medical treatment and not basic medical attention.

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Chapter XI

Controversies in Forensic Dentistry: Basic Medical Attention/Medical Treatment

Authors: Dr. J. F. Ortigosa Ruiz*, Dr. F. X. Güerri Ripol and Dr. Marta Macharé Alberni

1. Introduction to the controversies

Controversies in the assessment of oral and maxillofacial injuries can be divided into three groups:

- Treatment.
- Duration of the lesions.
- Sequelae.

2. Treatment

In general we consider the existence of medical and surgical treatment based on the need for it.

Thus surgical extractions and root canals should be considered as medical and surgical treatment.

By contrast, we consider reconstructions and fillings and periodontal tooth extractions to be basic attention.

We have to bear in mind that some dental treatments are not treatments of initial lesions but rather of sequelae. Thus treatment with implants or orthodontic treatment should be considered as treatment of sequelae. This has an impact on both the type of offence and the determination of the duration of the lesions.

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3. Duration of the lesions

In general soft tissue injuries and dentoalveolar injuries are similar in duration to bruises and have a stabilisation time of between 7 and 21 days.

Maxillary and craniofacial fractures have a stabilisation time of between 60 and 120 days.

It should be noted here that implants and orthodontics are treatments of sequelae and therefore their duration should not be taken into account when calculating injury stabilisation time.

4. Sequelae

The assessment of sequelae affecting the osteoarticular system does not usually prove overly problematic.

Trickier is the evaluation of dental sequelae, as the scale in Act 34/2003 only awards 1 point for each “completely” lost tooth.

Given that this point assesses functional chewing loss, it would seem that some important aspects are therefore not assessed.

Thus incisors, canines and premolars are of obvious aesthetic importance and disfigurement needs to be considered.

These anterior teeth are also important for speaking and losing them will have an impact on occupations that involve using the voice such as speakers, teachers, musicians, singers and wind musicians.

Another controversial issue is the concept of *complete traumatic loss* of the tooth. Conservative dentistry treatments allow the survival of the teeth in the mouth by root canal and reconstruction treatment. Clearly under these circumstances this is not a “complete loss” of the tooth, but equally it is not a normal healthy tooth but rather a weakened one with more chances of complications such as tooth fractures or periodontal lesions which jeopardise its future viability.

4.1 Maxillofacial region trauma

The major cause of multiple injuries is road traffic accidents (40%) followed by domestic accidents (20%), accidental falls (14%), occupational accidents (3%) and other causes (8%).

The area most frequently affected by trauma is the head with the following predominant involvement:

- The maxillofacial area is the most involved at 72.1%.
- Meanwhile the cervical region is involved in 8.7% of cases.

Factors to bear in mind:

1. Dental trauma can affect the pulp directly or indirectly.
2. A very important factor in dental injuries is age. By age 14 approximately 25% of children have suffered dental trauma consisting of a lesion to permanent dentition. In this situation we do everything we can to try to preserve pulp vitality.
3. The ideal response of the pulp to injury is complete recovery after a complete lesion.

There are many international classifications of dental trauma. Below we set out the following extended Andreasen classification, chosen to enhance understanding of dental trauma.

4.1.1 Injuries to dental hard tissues and pulp

- a) Enamel fracture or incomplete enamel fracture without loss of substance.
- b) Uncomplicated crown fracture, limited to enamel.
- c) Enamel-dentin fracture or uncomplicated crown fracture, involving both enamel and dentine but without exposing the pulp or supporting tissues; everything is intact.
- d) Complex crown fracture with pulp exposure (enamel + dentine + pulp)

4.1.2 Injuries to dental hard tissues, pulp and the alveolar process

- a) Uncomplicated crown-root fracture: involves the enamel, dentine and cementum but does not expose the pulp.



- b) Complex crown-root fracture: involves the enamel, dentine and cementum and exposes the pulp.
- c) Horizontal root fracture → does not involve the crown. Treatment is virtually heroic as the tooth can be written off as lost: EXTRACTION. The prognosis depends on the level where the fracture is located; the more apical the better the prognosis, since mobility will be less and stability greater.
- d) Tooth socket fracture.
- e) Fracture of the alveolar process (whole socket) and avulsion fracture.
- f) Maxillary/mandibular fracture (including or not the tooth socket and teeth).

4.1.3 Injuries to periodontal tissues:

Stages of ascending involvement:

- a) Concussion: blow which inflames periodontal tissue. Feeling of long tooth, that the tooth is striking earlier because it has moved.
- b) Subluxation (loosening): loose tooth feeling. The changes between concussion and subluxation are very subtle.
- c) Extrusive luxation (partial avulsion): the tooth is somewhat displaced.
- d) Intrusive luxation (central dislocation): sometimes the tooth is lost to “view”. If not seen, check for intrusion or if the tooth has fallen out (intraoral X-ray).
- e) Lateral luxation: associated with fracture of the alveolar process.
- f) Avulsion (exarticulation): absence of the tooth. Consider whether to reimplant (temporary or permanent tooth, person’s age).

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Alveolar-dental injuries are more common in men. Dislocations are more frequent in deciduous dentition while fractures are more frequent in permanent teeth. The teeth most frequently involved are the upper central incisors, upper lateral incisors and lower incisors.

4.1.4 Gum and oral mucosa injuries:

- a) Laceration (lacerated contused wounds).
- b) Contusion.
- c) Erosion-abrasion.

4.2 Fractures

Below we describe the following fractures by their medico-legal interest:

4.2.1. Mandibular fractures

Classification by location:

- Symphyseal region: intercanine (distal of canine to distal of canine).
- Body region: from distal canine to anterior margin of the anterior insertion of the masseter muscle.
- Angle region: from the anterior insertion of the masseter muscle to the line from the ridge to the posterior insertion of the masseter muscle.
- Condylar region (they are the most common fractures).
- Ascending ramus region.
- Coronoid process region.
- Alveolar process region.



Physiopathology:

Mandibular fractures can be produced by:

- *Direct force*: at the point of impact. (10%)
- *Indirect force*: when force exceeds mandibular elasticity. At the contralateral level it breaks in the weakest area. (90%)
- *Muscle spasm*: epilepsy, electroshock, electrocution, etc. (no trauma).

When a fracture occurs, the muscular forces can push the fragments in different directions. This depends not only on where the jaw is broken but how it is broken, the number of fracture lines and whether they are favourable or unfavourable fractures.

A bruise on the chin can produce a subcondylar fracture.

An open bite after trauma which did not previously exist may suggest a **bilateral** subcondylar fracture.

By contrast, deviated midlines and a unilateral tilted mandible may suggest a *unilateral* subcondylar fracture.

4.2.2 Longitudinal tooth fractures of the crown and root

Horizontal fractures are normally much more common in the anterior sector due to the impact sustained from direct trauma.

Longitudinal (vertical) fractures can affect all groups of teeth and may be due to:

- 1) the action of occlusal forces, which exceed the limit of elasticity of the dentine and/or enamel, or
- 2) dental treatment of teeth subject to complex surgery involving restorative and endodontic treatment that removes part of the dentine, thus compromising the internal resistance of the teeth.

They can be classified into:

- a) cracks
- b) cusp fractures
- c) cracked tooth (crown and some root involvement)
- d) complete fracture of part of the crown and root
- e) vertical root fracture

Treatment is complicated because the diagnosis can sometimes be difficult (filling or cosmetic restoration, endodontics), while in other cases the affected tooth may need to be removed.

4.5.3 Fractures of the maxillary or mandibular alveolar process

The most important thing is that when these fractures affect tooth sockets, they may lead to pulp necrosis on involved or nearby teeth associated with the fracture lines. The diagnosis is suspected when there is very strong dental displacement, when several teeth are displaced or move as a block, when there are discrepancies in occlusal alignment or uninterrupted intraoral bleeding, trismus.

4.5.4 Malar fracture

Clinically it presents eyelid ecchymosis, subconjunctival haemorrhage, anaesthesia of the infraorbital nerve territory, infraorbital oedema.

4.5.5 Maxilla fracture: Le Fort I-II-III

Clinically it presents upper and lower eyelid haematoma in both eyes, long face and premature contact of the molars without contacting the anterior teeth, suggesting midface fracture.

- a) Le Fort I or de Guerin or transversal fracture of the maxilla: the fracture line is located above the tooth apex and extends to the pterygoid.
- b) Le Fort II or pyramidal fracture: the fracture line goes along the nasal root, the lachrymal bone, the infraorbital margin and the wall of the maxilla to the pterygoid.
- c) Le Fort III or craniofacial disjunction fracture: nasal root, lachrymal bone, frontal process of the malar bone, lateral and posterior wall of the maxilla to the pterygoid.

4.3 Physical examination of a patient with dental trauma:

The most important thing is to carefully review the medical and dental information received about the trauma which should state:

- a) The type of injury
- b) Its exact location
- c) The diagnostic tests used (periapical X-ray, bitewing, orthopantomography, etc.)
- d) Vitality tests and outcome.
- e) Prognosis and treatment to be performed.

The clinical examination is usually performed some time after trauma and may include:

- a) Examination of the lips, the soft tissues of the mouth and facial skeleton, teeth and supporting structures. Tooth count.
- b) The colouring of the dental crown.
- c) Whether or not there is mobility (carefully examine the involved tooth and its neighbours and assess the degree of mobility which has 3 grades: 0 (none), 1 (slight mobility <1 mm), 2 (marked mobility, 1-3 mm), 3 (excessive mobility > 3 mm, vertically and horizontally).
- d) Perform percussion, firstly on apparently healthy teeth and then gently on the involved tooth; listen to the sound or ask if it causes discomfort.
- e) Note the existence of perilesional and contused soft tissue lesions, where there may also be haemorrhaging and oedema at the level of the periodontal ligament with positive percussion.
- f) Existence of occlusal interferences (especially after mandibular or maxillary fractures).
- g) Previous dental state and the existence of pathologies that may be contributing causes of the appearance of lesions and sequelae should also be assessed.
- h) Subsequently assess dental occlusion, especially in denture wearers who have suffered mandibular or maxillary fractures.

The following physical and dental examination of the injured person will be needed:

- 1) Systematic: always following an order and from the 1st to 4th quadrant.
- 2) Assess the x-rays of the injured area taken at the time of injury or later as they reveal valuable information about fractures in bone and teeth and the state of development of the tooth root in childhood injuries.

Additional tests used depend on the type of fracture and the place where you are (outpatients, clinic, hospital, etc.).

- Plain x-ray of skull, face and profile à not very useful. Normally done in hospital by default.
- Orthopantomography: reports on mandibular/maxilla and dental injuries.
- Occlusal radiograph (for fractures of the alveolar process).
- Waters' view or occipitomental radiography (gives information of the orbits).

Inverted Waters' view. Patients with cervical pain... for malar, inferior maxilla, nasal fractures involving the zygomatic bone...

- Hirtz's view or defilade radiography of the zygomatic arch.
- Nasal bone radiography.
- Defilade jaw radiography with different angles.
- Towne radiography (ascending ramus of the mandible and subcondylar fractures). Opposite to Waters' view.
- Tomography.
- CT: Lefort I-II, malar and orbital floor.

4.4 Medico-legal considerations:

- Trauma on teeth acutely leaves obvious signs of the damage sustained. However, sometimes checks will have to be made over time to objectify any possible pulp necrosis which would result in loss of tooth vitality and subsequent treatment (endodontics or extraction).
- It is important that in all simple crown fractures with highly unlikely pulp involvement a check is made for other associated signs, positive percussion, dislocation, etc.
- The first treatment used for these injuries will be conservative, provided this is possible.

4.5 Treatment

1. Tooth fractures: dental treatment with antibiotic coverage if there is pulp exposure.

2. *Periodontal tissue injury:*

- a) Tooth intrusion (immature apex): spontaneous reeruption.
- b) Tooth intrusion (mature apex): orthodontic or surgical repositioning, prophylactic endodontic treatment.
- c) Tooth intrusion (deciduous dentition): assess whether it affects permanent dentition, and if so extraction.
- d) Extrusion and dislocation: socket replacement, fixation for 1 to 2 weeks, assess endodontics.
- e) Avulsion: preservation in optimal media: milk, blood, saline solution, saliva. Minimum time between trauma and treatment of two hours. Permanent teeth free of decay and periodontal disease or crowding or poor prognosis due to time elapsed and transport are reimplanted. Tooth cleaning without scratching the tooth root. Cleaning the socket, reposition the tooth in the socket, fix for 7 days and endodontics. Antibiotic and tetanus prophylaxis.
- f) Bone injuries: splint.

Note that any fracture in the arch is an open fracture. The ideal treatment in these cases is emergency surgery.

Normally intermaxillary fixation of teeth 3-4-5-6 will be used in a subcondylar fractures for 3 weeks, though not for more than 3 weeks as it could lead to ankylosis. RHB is started after 3 weeks involving mobility guiding the occlusion with elastic bands. In the rest of mandibular fractures, fixation is normally for 6 weeks unless osteosynthesis material is used, in which case the time will be shorter (3 weeks).

5. Conclusions

Dentistry is a constantly evolving speciality which must be continuously reviewed owing to technological developments, research and renewal of materials, the obvious greater expertise and training of new generations and the concept of total healing time for health or disability.

Even though forensic professionals call for clear solutions, static or at least stable criteria and a fundamentally useful tool for everyday work, and although in this paper we also present very clear criteria for carrying out expert examinations, we believe that the current perspective on expert assessment has to change drastically.

Thus the criterion of the speakers at the oral presentation was to reflect our shared vision that the current concept of medical treatment or basic attention and the time assessment of injuries in their various aspects are or should be a criterion of the last century. We seek to convey that these current criteria are not valid in a speciality such as ours and we believe they are not valid for the rest of medicine either. An injury

cannot be assessed in one way or another depending on the knowledge, expertise, ability or interest of a professional, as these are quite variable depending on the circumstances of place, person, resources or time.

We think that the criteria should be based only on the injury caused with the different circumstances surrounding it, criteria which may be a subject for further debate.

To this can be added the fact that, as already mentioned in the introduction, it is easy to include treatment of the injury with treatment of the sequela within the expert's work in our speciality. Determining the need for one or the other praxis to treat the patient must be separated from trying to treat the injury to repair the damage caused. In other words, it is for example perfectly possible to live with a broken tooth, with obturation being used to repair the sequela, and therefore it should not be included in healing time, whereas by contrast it is not possible to live with an injury involving dental pulp and this requires treatment by a specialist to relieve pain and seal the damaged tissue.

In conclusion, we argue that the body assessment system needs to be reworked by turning it into a much simpler system, one which is not affected by environmental and personal factors and which can become uniform and revolve around the injury. We do not know if we are laying a cornerstone, or whether everything is so monolithic that we shall have to wait for a new Pere Mata to streamline the current system of assessment.

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Chapter XII

Assessment of Ocular Trauma: Basic Medical Attention/Medical Treatment

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Collaborator: Isabel Bobes from the Language Normalisation Service for the Courts of Tortosa

1. Introduction and approach of the study

Ocular trauma is an important part of all body traumas since while the front surface of the eye makes up only 0.27% of the body's area, accidents in this area account for a little more than 10% of all body accidents.

Ocular trauma is the leading cause of anatomical loss of the eyeball as well as the multiple sequelae arising from it, loss of work hours, disability, etc. at a high cost not only in healthcare but also economically and socially.

This paper analyses ocular trauma in relation to assessing bodily injury and the various controversies that arise around:

- whether or not it has required medical and/or surgical treatment
- healing and disability days and hospital stay days
- sequelae or permanent disabilities
- mechanism of injury and medico-legal aetiology, as the injury may have taken place in an occupational, casual or sports accident, traffic accident or assault

* Group Leader

2. Mechanical trauma

2.1. Eyelid injuries

2.1.1 Haematoma

It is a type of bruise, meaning surface and deep traumatic injury to the tissue which may or may not have macroscopic manifestations but always has objectifiable microscopic signs, generally without interruption of the skin.

It is the most common manifestation of injury due to assault and occupational, traffic and casual accident.

Symptoms and signs. Painful swelling and variable ecchymosis (“panda eyes”).

Examination. Simple inspection. The eyeball, visual acuity and the whole ocular-adnexa should be assessed.

Complications. The prognosis is generally good unless there is some kind of associated orbit pathology.

Treatment. Basic medical attention: analgesics, anti-inflammatory drugs and local cold.

Healing. 8 days on average. Varies according to the intensity of the haematoma. Does not require hospitalisation or incapacity.

Sequelae. Not to be expected.

2.1.2 Lacerations

Eyelid injuries can easily occur in facial trauma. They can be simple superficial skin layer abrasions or puncture and sharp wounds and avulsions.

Superficial laceration (abrasion)

Symptoms and signs. Erosion, painful swelling.

Examination. Careful physical examination of the wound and the eyeball.

Complications. The prognosis is generally good unless there is some kind of associated eyeball and adnexal pathology.

Treatment. Basic medical attention: analgesics, anti-inflammatory drugs and local cold.

Healing. 5 days on average.

Sequelae. Not to be expected.

Laceration of the edge of the eyelid

Symptoms and signs. Defect of the edge of the eyelid. Painful swelling.

Examination. Evaluation by an ophthalmologist in OR. Careful physical examination of the wound. The eyeball, visual acuity and the whole ocular-adnexa should be assessed.

Complications. The prognosis is generally good. Cicatricial ectropion may appear. If on the inner edge it may involve the lachrymal system.

Treatment. Basic medical attention and medical and surgical treatment: wounds of the free edge of the eyelid need to be treated by an ophthalmologist in OR, as repair by levels has to be perfect and without tension to prevent cicatricial ectropion. Washing the wound. Removal of foreign bodies. Tetanus prophylaxis. Assess systemic antibiotics.

The extensive vascularity of the eyelid allows repair by primary intention even up to 12 hours after injury. If it does not affect the eyelid margin (free edge) it can be closed in one or two levels.

Healing. If it does not affect the free edge of the eyelid, stitches are removed after 4-6 days. If the free edge of the eyelid is affected, the average estimated time for removing stitches is 10-14 days.

Sequelae. If healing is not perfect there will be cicatricial ectropion.

Laceration with loss of tissue

Symptoms and signs. Defect of the edge of the eyelid. Painful swelling.

Examination. Evaluation by an ophthalmologist in OR. Careful physical examination of the wound. The eyeball, visual acuity and the whole ocular-adnexa should be assessed.

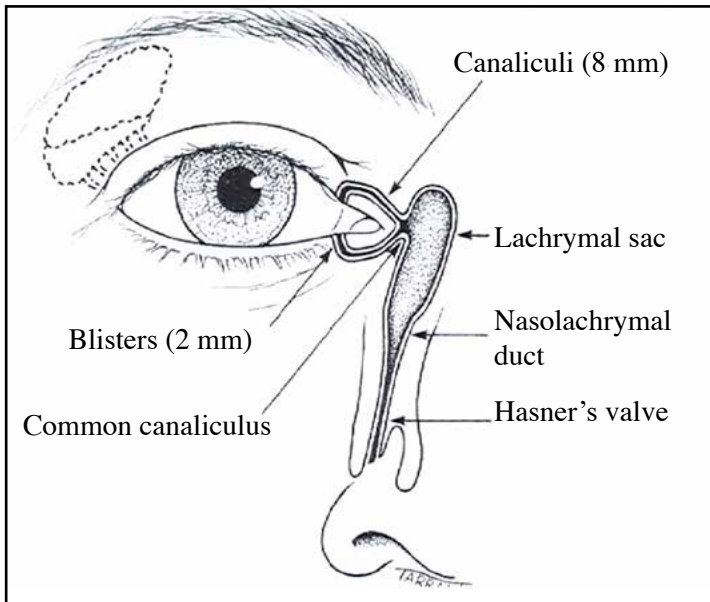
Complications. The prognosis is generally good. Cicatricial ectropion may appear. If on the inner edge it may affect the lachrymal system (canicular laceration).

Treatment. Basic medical attention and medical and surgical treatment: wounds of the free edge of the eyelid need to be treated by an ophthalmologist in OR, as repair by levels has to be perfect and without tension to prevent cicatricial ectropion. Washing the wound. Removal of foreign bodies. Tetanus prophylaxis. Assess systemic antibiotics.

The extensive vascularity of the eyelid allows repair by primary intention even up to 12 hours after injury. Usually prior lateral cantholysis is performed to increase the lateral mobility of the eyelid.

Healing. The average estimated time for removing stitches is 10-14 days.

Sequelae. If healing is not perfect there will be cicatricial ectropion and disfigurement.



2.2 Lacrimal system trauma

The lacrimal drainage system consists of the following structures:

The *lacrimal points* are at the far back of the eyelid edge. Generally they face slightly backwards and can be inspected with eversion of the medial aspect of the eyelids. The *canaliculi (tear ducts)* run vertically from the edge of the eyelid (blisters), then turn and run horizontally to reach the lachrymal sac. In most people, the upper and lower canaliculi form the common canaliculus which opens in the side wall of the lachrymal sac. The *lachrymal sac* is located between the anterior and posterior lachrymal crests. The lachrymal bone and frontal process of the maxilla separate the lachrymal sac from the middle meatus of the nasal cavity.

The *nasolachrymal duct* is the lower continuation of the lachrymal sac. It runs downwards and at a slight angle to the side and rear to open into the lower nasal meatus laterally and below the inferior turbinate. The opening of this duct is partially covered by a fold of mucous (Hasner's valve).

Trauma to the lacrimal system is a result of stab wounds and avulsions of the inner edge of the eyelids (in dog bites, for example, or injuries due to broken glass) and can damage lachrymal puncta and canaliculi. Traumatic lesions of lachrymal glands or the lachrymal sac can only occur in severe craniofacial trauma.

Symptoms and signs. They depend on the level of the lesion location and therefore of the anatomical structure involved: lachrymal puncta,

lacrimal canaliculi, the lacrimal sac or nasolacrimal duct. Hence if the injury is on eyelid structures (lacrimal punctum and canaliculi) it can be extrapolated to the entire clinical signs described for eyelid lesions with epiphora.

Examination. Evaluation by an ophthalmologist in OR. Careful physical examination of the wound and the whole ocular-adnexa.

Complications. Healing obstructions may occur more frequently in eyelid lacerations affecting the edge.

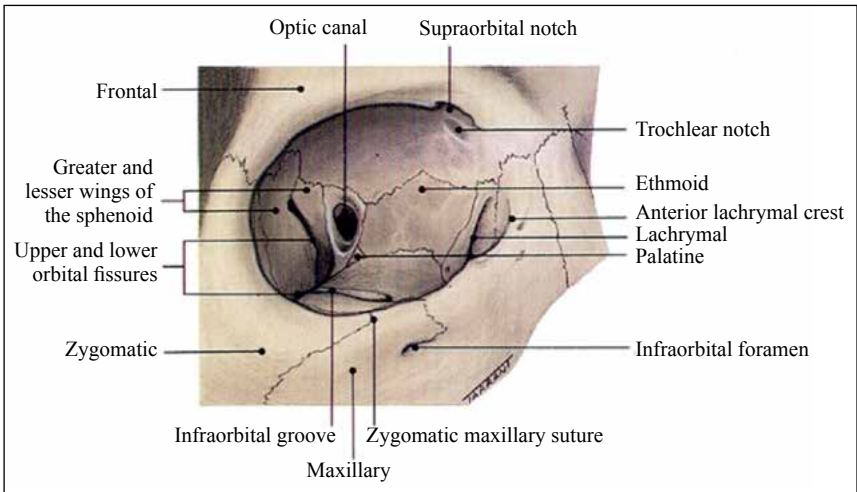
Treatment. Basic medical attention and medical and surgical treatment: they require microscopic surgery by a specialist ophthalmologist. A silicone probe is used to repair the canaliculi.

Healing. The estimated average time for removing the probe is 3-6 months.

Sequelae. If healing is not perfect a cicatricial obstruction of lacrimal drainage may be left along with epiphora which may constitute disfigurement.

2.3 Orbit trauma

The orbit is a pear-shaped cavity whose stem is the optical canal. The intraorbital portion of the optic nerve is longer than the distance between the back of the eyeball and the optic canal which allows significant forward displacement of the eyeball without causing excessive traction of the optic nerve.



The orbital cavity is formed by:

The *floor* consists of three bones: zygomatic, maxillary and palatine. The posteromedial portion of the maxillary bone is relatively weak and can be affected by a “depressed” fracture. The roof of the orbit forms the roof of the maxillary sinus.

The *medial wall* consists of four bones: maxilla, lachrymal, ethmoid and sphenoid. The lamina papyracea is a very thin part of the medial wall and is perforated by numerous orifices for nerves and blood vessels.

The *roof* of the orbit consists of two bones: the lower wing of the sphenoid and the orbital plate of the frontal bone. It is located next to the anterior cranial fossa and the frontal sinus.

The *side wall* also consists of two bones: the greater wing of the sphenoid and the zygomatic bone.

2.3.1 Fracture due to subsidence of the orbit floor

Traumatic injury produced by two mechanisms: the mechanism most frequently involved is a sudden increase in orbital pressure transmitted to the floor of the orbit by the eyeball on receiving the impact of a blunt object causing herniation and immobilisation of orbital fat and the inferior rectus muscle and its sheath. Another mechanism is traumatic impact on the inferior orbital rim which collapses the weak orbit floor.

Symptoms and signs. At the periocular level there is ecchymosis, oedema and subcutaneous emphysema in varying degrees. Anaesthesia of the infraorbital nerve (affecting the lower eyelid, side of nose, lip, upper teeth and gums). Vertical diplopia, enophthalmia. Intraocular lesions due to the bruising.

Examination. Expert ophthalmological examination is needed. Examination of the eyeball by slit lamp and ocular fundus. Examination by MRI and CT.

Complications. Vertical diplopia and enophthalmia.

Treatment. It will always be medical and surgical by an ophthalmologist. It will depend on the size of the fracture, herniation of orbital contents in the maxillary sinus and muscle entrapment.

Healing. Average healing time between 30 and 60 days depending on the degree of involvement of the inferior rectus muscle.

Sequelae. Permanent vertical diplopia and enophthalmia. Synthetic material to repair the defect of the orbit floor and disfigurement.

2.3.2 Nasoorbital or nasoethmoid fracture

Violent traumatic injury to the nasal pyramid, causing the subsidence of the nasoethmoidal complex which collapses the interorbital space and results in a fracture of the medial side of the orbit (lamina papyracea of the ethmoid).

Symptoms and signs. Unilateral or bilateral eye ecchymosis, eyelid crepitus, periorbital and nasal oedema, telecanthus (increase in the distance of the medial epicanthus), epistaxis, external dorsonasal flattening/excavation with elevation of the tip of the nose, epiphora (if tear duct is involved), enophthalmia with eyelid ptosis, CSF leak (liquor-rhea) and occasionally diplopia.

Examination. Expert ophthalmological examination is needed. Examination of the eyeball by slit lamp and ocular fundus. Examination by X-ray and CT.

Treatment. Treatment is always surgery, consisting of reducing and immobilising the fractures by open surgery and osteosynthesis with wire to repair the detachment of the medial canthal ligament and restore the nasolachrymal duct (dacryocystorhinostomy) if possible immediately.

Healing. The average stabilisation time of these skull fractures ranges from 30-60 days.

Sequelae. Epiphora, enophthalmia, diplopia, osteosynthesis material and disfigurement.

2.3.3 Orbital roof fracture

A very rare traumatic injury in ophthalmology. They are generally isolated fractures caused by minor trauma, such as falling on a sharp object or a blow to the forehead. They are, however, common in children. In adults they are complicated fractures caused by major trauma with significant involvement of craniofacial bones.

Symptoms and signs. Upper eyelid hematoma and periorbital ecchymosis, which occurs after a few hours and can extend to the opposite side. Inferior or axial displacement of the eyeball. In major trauma it may be associated with a pulsation of the eyeball due to the transmission of CSF pulsation.

Examination. Expert ophthalmological examination is needed. Examination of the eyeball by slit lamp, ocular fundus and tonometry. Examination by X-ray and CT.

Treatment. Treatment varies depending on the scale of the bone lesions: small fractures may not need treatment, although it is important to rule out possible CSF leakage that can lead to meningitis. Major bone defects may need reconstructive surgery.

Healing. The average stabilisation time of these skull fractures ranges from 30-60 days.

Sequelae. Disfigurement. In cases of reconstructive surgery with use of osteosynthesis material, this material will persist.

2.3.4 Fracture of the side wall

A very rare traumatic injury in ophthalmology and very often associated with extensive facial injuries. It is a type of fracture that predominantly affects the complex formed by the zygomatic bone and the malar bone.

Symptoms and signs. They depend on whether it is a stable fracture (1/4 of cases) or a fracture accompanied by greater or lesser displacement. Present with deformation of the cheekbone to varying degrees depending on the displacement. Subconjunctival and periorbital haematoma. If it affects other walls of the orbits, such as the floor, it proceeds with epistaxis and possible involvement of the infraorbital nerve.

Examination. Expert ophthalmological examination is needed. Examination of the eyeball by slit lamp and ocular fundus. Examination by X-ray and CT.

Treatment. Treatment is always surgery. In displaced fractures, treatment is open reduction and fixation of the displaced fragments with wire sutures. Semi-open reduction may be sufficient with minimum displacement by inserting an elevating instrument that levers the depressed malar bone.

Healing. The average stabilisation time of these skull fractures ranges from 30-60 days.

Sequelae. Osteosynthesis material. Disfigurement.

2.4 Eyeball trauma

2.4.1 Conjunctival lacerations

It is a type of injury that is caused by the action of sharp trauma, collisions with objects or flying foreign bodies.

Symptoms and signs. Red eye, moderate pain and foreign body sensation (with a history of trauma). Conjunctival redness or subconjunctival haemorrhage (hyposphagma) is observed in the area of the wound. Sometimes conjunctival dehiscence is only evidenced by fluorescein staining.

The possibility of penetrating wounds or intraocular foreign bodies must always be considered.

Examination. The entire eyeball has to be assessed, while always exploring the underlying sclera by applying a topical anaesthetic.

Treatment. Basic medical attention: subconjunctival haemorrhages do not need treatment. If there is interruption, antibiotic ointment or eye drops are recommended.

Surgical treatment. Only suture with absorbable material large dehiscence with hanging edges.

Healing. Average healing time is 5-7 days.

Sequelae. Not expected unless there is perforation of the eyeball (see penetrating injuries of the eyeball) or they involve the cornea (see corneal lesions).

2.4 Corneal and conjunctival foreign bodies

They are one of the most common ocular emergencies in medical practice.

They are generally airborne foreign bodies.

Symptoms and signs. Foreign body sensation in every blinking movement, pain, tearing, photophobia and blepharospasm, conjunctival hyperaemia depending on time present (hours to days).

Examination. All orbit structures should be assessed. Sometimes they are very small and can only be seen with a magnifying glass. When there is no foreign body, fluorescein staining will reveal the presence of corneal injury which stains. If in the fluorescein staining the cornea presents vertical "scratches" that are stained, this would suggest a sub-tarsal foreign body, so the conjunctival sac should always be examined.

Treatment. Basic medical attention: most non-fixed foreign bodies can be removed with the edge of a piece of cloth or gauze or a wet cotton swab under topical anaesthetic or just with irrigation. Never allow the patient to continue to apply the topical anaesthetic or establish it as a treatment because it can cause severe corneal injuries due to loss of the painful corneal reflex.

Medical and surgical treatment. If it is attached to the cornea it is removed with a spatula or 25 g needle. The bed left behind may be infiltrated or have oxide impregnation and should be carefully and fully polished.

After removal of the foreign body, the residual lesion is a corneal erosion and is treated as such.

Healing. Average healing time: 3 days - 1 week.

Sequelae. Since corneal erosion is left behind after removal of the foreign body, the same sequelae as described for this diagnostic entity may persist.

2.4.3 Corneal erosion

They may be caused by flying foreign bodies that are subsequently removed or various kinds of contusions (with nails, edges of sheets of paper, etc.).

Symptoms and signs. Pain, foreign body sensation (even when there isn't one), lachrymation, photophobia, hyperaemia, history of mild ocular trauma.

Examination. Topical anaesthetic, fluorescein staining to get the size and location of the lesion, eyelid eversion in search of injury or foreign body.

Complications. In the case of recurrent erosions, sometimes the epithelial cells adhere poorly to the Bowman layer resulting in the epithelium repeatedly breaking at the site of the initial trauma. Initial normal healing occurs in 24-48 hours, but relapses occur (often in the morning on waking when suddenly opening the eyes). This situation usually entails a heavy psychological burden for the patient. It is treated with antibiotic ointment and prolonged occlusive bandaging either every night or with a therapeutic contact lens and antibiotic eye drops. In some cases it may require hospital admission and putting on a bilateral ocular bandage. Phototherapeutic keratectomy and corneal punctures are other treatment options. These cases would come under the medico-legal concept of medical and surgical treatment.

Treatment. Basic medical attention to prevent infection by applying antibiotic ointment every 8 hours for 7-10 days. The pain also needs to be eased with homatropine eye drops or cycloplegic eye drops every 8 hours for 2-3 days. Eye occlusion for 24 to 48 hours (while it hurts).

Topical anaesthetics should never be used as treatment.

It does not usually require medical treatment by an ophthalmology specialist except in the case of embedded attached foreign bodies or large or central ulcers or recurring erosions.

Healing. Average healing time: 9 days.

Sequelae. It will not leave a scar (leukoma) if depth is less than the Bowman membrane.

Sight may be severely affected if the erosion affects the pupillary area.

2.4.4 Contusion of the eyeball

Overview

When a blunt object has a diameter less than the diameter of the orbit (a stone, hammer, stick, champagne cork or squash ball), the eyeball receives most of the impact and it tends to be more severe than if

the diameter of the object is larger as it would primarily hit the orbital edges and might produce other kinds of injuries.

Eye contusion injuries are very diverse, ranging from minor conditions with no consequences to complete loss of vision.

Entities that can develop after eye contusion:

Traumatic hyphaema

It means accumulated blood in the anterior eye chamber.

The source of bleeding is in the iris or ciliary body (anterior uvea).

It is the injury most frequently caused by blunt trauma to the eye.

It may be isolated or associated with more severe injuries of posterior segments.

Signs and symptoms. Pain, blurred vision.

Examination. Eyeball examination during which the following are to be assessed:

Visual acuity.

Anterior pole. Reddish clouds in the anterior chamber or blood deposited with a free upper level or anterior chamber haemorrhage that obscures the iris.

Fundus. RBCs characteristically settle to the bottom resulting in a fluid level whose height has to be measured and documented indicating the grade (grade I: $<1/3$ of anterior chamber; grade II: $1/3-1/2$ of anterior chamber; grade III: $> 1/2$ of anterior chamber; and grade IV: total hyphaema).

Intraocular pressure. Intraocular pressure (IOP) has to be assessed.

Decide on CT scan based on the intensity of trauma if it is not possible to see the fundus and other injuries are suspected.

Treatment. Basic medical attention: most traumatic hyphaema in which bleeding is minimal (microhyphaema grade I) are harmless and transient and simply require daily observation until they clear up on their own.

Medical and surgical treatment. Resting at a 45° angle, non-aspirin analgesics and control of intraocular pressure is recommended for grade II hyphaema. If intraocular pressure is >24 mmHg administration of atropine is indicated (if there are no more injuries). If intraocular pressure values do not come down surgical resorption of the hyphaema is indicated to prevent corneal pigmentations and deposits or damage to the optic nerve.

Healing. Average healing time is between 20-90 days.

Complications. Rebleeding may occur between the 2nd and 5th day of trauma. It is most common during the first 24 hours after injury.

Other possible complications include corneal pigmentations and deposits (due to secondary impregnation of the cornea with blood) and lesions of the optic nerve.

Another possible complication is traumatic uveitis.

Sequelae. They usually heal without sequelae except for the presence of associated injuries or complications (damage to the optic nerve or corneal pigmentations and deposits).

Pupillary involvement

Spastic miosis

Very frequent after trauma.

It is due to the imprint of pigment from the iris on the anterior lens capsule (Vossius ring) which corresponds to the size of the miotic pupil.

Symptoms and signs. Defects of accommodation and myopia.

Examination. The following should be examined:

Visual acuity.

Examine anterior pole: examination of pupillary reflexes.

Fundus.

Complications: Do not usually appear.

Treatment. Basic medical attention: tends to clear up on its own.

Healing. Short.

Sequelae. Does not usually leave any.

Mydriasis

It is due to a lesion of the iris sphincter.

Signs and symptoms. Dilated pupils, the pupil reacts slowly or does not react to light and accommodation, photophobia. Radial tears are common on the pupillary border.

Examination. The following should be examined:

Visual acuity.

Examine anterior pole: examination of pupillary reflexes.

Fundus

Complications: Do not usually appear.

Treatment. Basic medical attention: depending on the intensity of the tear of the pupillary sphincter or dilator muscles. Afterwards there is no treatment other than sunglasses.

Medical and surgical treatment. When tears are bigger and accompanied by more severe symptoms, an iris suture to tighten the pupil is recommended.

Healing. From several weeks to becoming a permanent injury.

Sequelae. Disfigurement, photophobia and accommodation problems.

Traumatic iritis

It is a pathological entity caused by the action of a severe blunt trauma which causes a temporary cessation of secretion of aqueous humour.

Symptoms and signs. Pain, photophobia and tearing, with a history of trauma, there may be also miosis (anisocoria with affected eye miosis), perilimbal conjunctival injection and decreased intraocular pressure.

Examination. The following should be examined:

Visual acuity.

The anterior chamber by slit lamp to evaluate the presence of a + Tyndall effect (intensity + to + + + is evaluated) which is the deposit of inflammatory white cells in the anterior chamber (if there is a large deposit it is called hypopyon).

Fundus.

Complications. Regular check-ups are needed as there may secondarily be late glaucoma due to angle narrowing or retinal tears.

Treatment. Basic medical attention: In cases where the Tyndall effect is slightly positive (+).

Medical treatment. In cases where the Tyndall effect is greater (+++/++++). It involves applying cycloplegic eye drops every 8 hours to break any irido-crystalline adhesions, corticosteroid eye drops every 1-4 hours, analgesics.

Healing. Average healing time: 20 days.

Sequelae: Does not usually leave any except for possible complications.

Iridodialysis (or angular recess)

It consists of the detachment of the iris root at a given level of the ring.

Symptoms and signs. Pupillary roundness is lost (pupil deformed in a “D” shape), glare increases. It may be asymptomatic if it is covered by the upper eyelid. Visual impairment when it is large and located in the eyelid fissure area causing a “double pupil”.

Examination. The following should be assessed:

Visual acuity.

Examine anterior pole: examination of pupillary morphology and pupillary reflexes.

Fundus.

Complications: unilateral diplopia and distellaments may occur.

Treatment. Basic medical attention: in most cases it does not require treatment especially if it is small.

Medical and surgical treatment in larger cases or suturing the base of the iris (iridopexy) in the case of “double pupil”.

Healing. Average healing time: from several weeks to becoming a permanent injury.

Sequelae. Disfigurement, photophobia and accommodation problems.

Traumatic aniridia

It is the complete detachment of the iris that “folds” to form a sphere and falls to the bottom of the anterior chamber. It will be secondary to the above described Injury, in which 360° iridodialysis occurs.

Signs and symptoms. Similar to the previous one.

Examination. Similar to the previous.

Complications: Glaucoma, unilateral diplopia and destellaments may occur.

Treatment. Basic medical attention to objectify the injury (total detachment of the iris). Symptomatic treatment consists of wearing sunglasses or the surgical implantation of an artificial black lens with an optical aperture of pupil size aimed at minimising the sequela.

Healing. Healing time ranges from several weeks to becoming a permanent injury.

Sequelae. Secondary to posttraumatic iris loss with accommodation disorder, loss of visual acuity and presence of osteosynthesis material (intraocular lens).

2.4.5 Luxation and subluxation of the lens

It occurs as a result of breakage of the zonule of Zinn.

It may involve partial or total breakage producing respectively subluxation or luxation of the lens which may be anterior or posterior (more rarely extraocular) depending on where the dislocated lens is sited.

Severe trauma is generated by the forward or backward pressure wave generated by the contusion.

It is more common in people with hypermobility (in fact spontaneous subluxation may appear in Marfan syndrome, homocystinuria or Ehlers–Danlos syndrome).

Signs and symptoms. It manifests in decreased visual acuity, monocular diplopia.

Examination.

Visual acuity.

Examine anterior pole: the lens is offset, iridodonesi (tremulousness of the iris) or phacodonesis (tremulousness of the lens).

Fundus.

Complications. Unocular diplopia, lenticular astigmatism due to tilting of the lens. Glaucoma.

Treatment. Medical and surgical treatment consisting of lens extraction and intraocular lens insertion.

Healing. Average healing time is 1-2 months after surgery if there are no complications.

Sequelae. Osteosynthesis material. Assess degree of accommodation. Can lead to uniocular diplopia, lenticular astigmatism.

2.4.6 Traumatic cataract

It occurs as a result of traumatic opening of the lens capsule through which aqueous humour enters which produces swelling of the lens and, secondarily, more or less severe progressive clouding of it.

Large breaks make the lens completely opaque within days, even hours. Smaller breaks which close on their own only cause circumscribed opacity, anterior or posterior subcapsular, in a rosette shape.

The largest are typically caused by penetrating injuries (see below).

Signs and symptoms. They are manifested by decreased visual acuity, monocular diplopia.

The period for establishment of a traumatic cataract varies by the severity of injury, and in cases of small breaks the clouding period is longer.

Diagnosis needs to differentiate between traumatic cataracts and other types of cataracts.

Examination.

Visual acuity.

Examine anterior pole: clouding usually occurs in the posterior subcapsular cortex along the posterior sutures (rosette cataract).

Fundus.

Complications. Resulting from surgery to improve vision (insertion of intraocular lenses)

Treatment. Medical and surgical treatment in cases of significant opacities.

Healing. Average healing time is 1-2 months after surgery if there are no complications.

Sequelae: Osteosynthesis material. Assess degree of accommodation.

2.4.7 Haemorrhage in the vitreous body

It occurs as a result of trauma or spontaneously. In 50% of cases the aetiology is associated with diabetic retinopathy.

It may be associated or not with other injuries (posterior vitreous detachment).

Signs and symptoms. Loss of visual acuity varies with the perception of floating bodies and a red or brown veil. Perception of light is preserved.

Examination.

Visual acuity.

Examine anterior pole:

Fundus: a blood clot and later a reddish-orange dispersed vitreous opacity are observed along with the absence of fundus reflex (red pupil is not observed).

Complications. Formation of membranes that can lead to traction retinal detachment.

Treatment. Basic medical attention: have to wait for spontaneous clarification in mild cases.

Medical and surgical treatment. In cases of large hemorrhagic extravasation, specific treatment required.

Healing. Average healing time: depending on the severity of the hemorrhagic extravasation and the existence of possible complications.

Sequelae. Loss of visual acuity which is variable.

2.4.8 Posterior vitreous detachment

State in which the vitreous cortex is separated from the inner boundary layer of the retina in the rear of the vitreous base. It is the result of liquefaction of the vitreous which starts in the rear and extends to the periphery. This creates mechanical instability.

It is common (2/3) in people over 65 and is also more common in people with myopia, perforating trauma, uveitis or genetic disorders.

Symptoms and signs. It is manifested by increased myodesopsia (perception of floaters, dark spots, threads, veils and “spider web” that move across the visual field when moving the eyes, more obvious when looking at a white background and correspond with vitreous opacities) and photopsias (perceiving flashes of lights).

Examination.

Visual acuity.

Examine anterior pole.

Fundus: small floating opacities are observed in the vitreous. The Weiss ring can be seen in the fundus, which was the area of adhesion of the vitreous to the optic nerve.

Complications. Retinal tears have to be ruled out which occur in 10% of cases. The possibility of retinal tears increases when associated with a history of retinal detachment, high myopia, head trauma or cataract surgery.

Treatment. Basic medical attention: does not need treatment in cases of mild symptoms.

Medical and surgical treatment. Variable depending on the complications.

Healing. Variable average healing time.

Sequelae. Depending on the complications.

2.4.9 Retinal disorder, retinal contusion or Berlin's oedema

It occurs is due to a mechanism of coup and contrecoup. Retinal and macular oedema in the posterior pole. May be associated with bleeding.

Signs and symptoms. Impaired visual acuity and whitening of an area of the retina (Berlin's oedema).

Examination.

Visual acuity.

Examine anterior pole.

Fundus: whitish retinal injury, sometimes with an iridescent appearance. It can be located on the posterior pole (Berlin's oedema) or retinal periphery.

Complications. If it involves the macula may be associated with intraretinal haemorrhage leading to posttraumatic macular changes including progressive pigmentary degeneration and the formation of a macular hole.

Treatment. Basic medical attention: watchful waiting and after a few days vision gradually recovers, although it may be delayed by pigment deposits in the macula.

Medical and surgical treatment. In case of oedema or complications.

Healing. Average healing time: from 2 to 6 weeks.

Sequelae. Heals with no sequelae, except for the complications described.

2.4.10 Retinal haemorrhages

They can go unnoticed. They are important because the presence of blood in the retina is toxic to the photoreceptors and retinal pigment epithelium which promotes detachment of the secondary retina.

Signs and symptoms. Impairment of visual acuity and perception of dark spots.

Examination.

Visual acuity.

Fundus: Tyndall effect haematic vitreous.

Complications. In case of major bleeding, fibrous proliferative vitreoretinopathy can occur and retinal detachment becomes easier. There is also the possibility of vitreous haemorrhage breakage.

Treatment. Basic medical attention: if there is not much bleeding, preventive administration of corticosteroids and cycloplegics is indicated.

Medical and surgical treatment. If there is a lot of bleeding, administration of corticoids and cycloplegics and posterior vitrectomy.

Healing. Average healing time: about 14-15 days, with no relationship between the size of the haemorrhage and final visual acuity.

Sequelae. Loss of visual acuity.

2.4.11 Ora serrata dialysis

Avulsion of the periphery of the retina (tear in the ora serrata) caused by traction of the vitreous gel along the rear face of the vitreous base. They can be caused abruptly by an anteroposterior mechanism or, secondarily, weeks or months later by vitreous traction on the retina.

Signs and symptoms. Photopsia, scotoma (fixed visual field defects) or blindness. Most frequent location superior or inferior temporal nasal.

Examination.

Visual acuity.

Fundus: sometimes the vitreous base is removed which leads to a “bucket handle” appearance.

Complications. Retinal detachment.

Treatment. Surgical treatment: surgery or laser photocoagulation.

Healing. Average healing time: 1-2 months.

Sequelae. Depends on complications.

2.4.12 Retinal tears and holes

They are caused by vitreous traction.

If they are tears they are often horseshoe or arrowhead shaped (operculated tears or holes). If they are holes they are usually round and oval (macular holes).

Signs and symptoms. Floaters or photopsia, more rarely scotoma (fixed loss of part of the visual field).

Examination.

Visual acuity.

Fundus: The retinal holes or tears are seen, sometimes associated with vitreous haemorrhage or intraretinal haemorrhage.

Complications. Retinal detachment.

Treatment. Surgical treatment: laser photocoagulation.

Healing. Average healing time 15-30 days.

Sequelae. Depends on the complications.

2.4.13 Traumatic retinal tear with detachment. Retinal detachment (RD).

This is a larger tear acting as a curtain.

Retinal tearing occurs either due to breakage and necrosis or vitreous haemorrhage traction.

There non-traumatic causes of retinal detachment, whether *predisposing diseases or conditions* that may cause it.

- Advanced age (the incidence of RD in the general population is 1:10,000 while in the over 70s it is 0.4%).
- Myopia > 6 diopters.
- Retinal degeneration: “palisade”, “snail slime”, “lattice” and retinoschisis.
- Collagen diseases: Marfan, Ehler-Danlos, etc. syndromes.
- Diabetes mellitus.
- Cotes disease (retinal vessel telangiectasia)
- Associated choroidal tumours.

Signs and symptoms. Scotoma and loss of visual acuity (“curtain”).

Examination.

Visual acuity.

Fundus. Detachment is seen, observing a horseshoe-shaped tear.

Complications. Loss of vision.

Treatment. Medical and surgical treatment: consists of one or a combination of these treatments:

- Laser photocoagulation.
- Surgery: cryopexy plus explant and/or scleral buckling. Vitrectomy and drainage of subretinal fluid.
- Intravitreal injection of expandable gas (SF 6).

Healing. Healing time: 120 days (varies according to the treatment applied).

Sequelae. Vision loss.

2.4.14 Traumatic choroid rupture

Choroidal rupture affects Bruch’s membrane and RPE (retinal pigment epithelium).

They can be direct (located anteriorly in the impact site and running parallel to the ora serrata) or indirect (occurring on the opposite side to the impact).

Signs and symptoms. It may be asymptomatic or cause loss of visual acuity when it affects the choroid in the macula.

Examination.

Visual acuity.

Fundus. If it is recent subretinal haemorrhage is seen. Within days or weeks of reabsorbed blood, multiple yellow or white subretinal formations are observed in the form of lightning bolts.

Complication. New vessels may grow (neovascular membrane) with vessels and connective tissue to the retina from the choroid.

Treatment. Medical and surgical treatment: according to the complications. Only the neovascular membrane is to be treated with laser if it forms and threatens the macula. Monitor for 3-6 months.

Healing. Between 3 and 6 months.

Sequelae. Visual impairment.

2.4.15 Optic nerve trauma

The mechanisms which may cause damage to the optic nerve are:

- Optic neuropathy. It is a rare cause, but devastating for permanent loss of visual acuity after blunt injury to the head (front).
- Avulsion of the optic nerve. It is rare and typically occurs when an object is inserted between the eyeball and the orbital wall (intraorbital foreign bodies, fractures of the orbit or other pathologies of the eyeball).

Signs and symptoms. Sudden decrease in VA, pain, afferent pupillary defect, visual field loss.

Examination.

Visual acuity.

Fundus. Typically the optic nerve head and fundus are normal at the beginning and the only objective fact found is a relative afferent pupillary defect. Over time you can see a whitish papilla, but not from the beginning.

CT; to rule out orbital foreign body or alterations of the orbital canal.

MRI.

Complications. Vision loss.

Treatment. Medical and surgical treatment; neither systemic steroids nor surgical decompression of the canal prevent the onset of optic atrophy in 3-4 weeks. If avulsion, there is no treatment.

Healing. The interruption of nerve fibres is irreversible.

Sequelae. Vision loss.

2.4.16 Retrobulbar haematoma

It is caused by severe blunt trauma that damages the retrobulbar vessels.

Signs and symptoms. Decreased visual acuity and pain. Associated with proptosis (exophthalmos) with resistance to retropulsion, eyelid ecchymosis, chemosis, increased intraocular pressure, conjunctival congestion and limited extrinsic ocular motility.

Examination.

Visual acuity.

Fundus.

CT of orbit.

Complications. Abscesses and infections.

Treatment. Basic medical attention consisting of controlling intraocular pressure to prevent complications.

Medical and surgical treatment. Methods to reduce intraocular pressure are used only if it increases. If there is a threat to visual acuity or it does not reduce, it then becomes necessary to hospitalise and perform decompressive surgery to prevent irreversible damage to the optic nerve due to occlusion of the central retinal artery because of the pressure.

Healing. Depending on the evolution and complications.

Sequelae. Assessment of vision loss.

2.5 Penetrating and/or perforating eyeball trauma

Penetrating eyeball injuries are defined as laceration of the full thickness of the ocular walls, usually caused by a sharp object and with no exit site. Such injuries are potentially very serious and so they need immediate diagnosis and treatment.

They typically affect the eyelids and also cause lacerations or penetrating injuries to the eyeball involving different structures that may range from large openings of the cornea and sclera with anterior chamber loss to barely visible small injuries which clear up on their own (they can sometimes be a gateway for small intraocular foreign bodies).

2.5.1 Eyelid involvement

It would be similar to the situation described for lacerations with tissue loss.

Symptoms and signs. Pain. Interruption.

Examination. Wound assessment and exploration by an ophthalmologist.

Complications. Eyelid involvement has a good prognosis. If it does not heal well, cicatricial ectropion may occur.

Treatment. Depending on its size, it will have to be aesthetically sutured (7-0 silk), so in these cases medical and surgical treatment will be needed.

Healing. Healing time will be approximately 7 days.

Sequelae. Ranging from scars that cause aesthetic damage to scars that cause impaired mobility of the eyelid, depending on the characteristics of the wound that has caused.

2.5.2 Lachrymal involvement (lower canaliculus)

Injuries affecting the lower lid may injure the lachrymal canaliculus, which requires immediate remedial attention to the extent possible given its important role in lachrymal drainage.

Symptoms and signs. See trauma to the lachrymal system.

Examination. See trauma to the lachrymal system.

Complications. See trauma to the lachrymal system.

Treatment. See trauma to the lachrymal system.

Healing. See trauma to the lachrymal system.

Sequelae. See trauma to the lachrymal system.

2.5.3 Eyeball involvement

Corneal lesions

Simple laceration (under 2 mm)

Symptoms and signs. These lesions may be symptomatic or cause pain.

Examination. Ophthalmoscopy or fluorescein staining.

Treatment. Clean the wound with saline solution, prescribe epithelialising ointment, antibiotic eye drops and apply an occlusive patch for at most 24 hours.

Healing. Approximate healing time from 48 hours to 5 days.

Sequelae. Except in cases where complications may arise, they usually heal without sequelae.

Larger laceration

Symptoms and signs. Present with pain, decreased visual acuity, photophobia, lachrymation and blepharospasm.

Examination. With direct and indirect ophthalmoscopy.

Treatment. Surgical suture (10-0 nylon, silk 8-0).

Healing. From 15 to 30 days.

Sequelae. Corneal scar that may cause decreased visual acuity.

Stellate laceration

Greater complexity required in care.

Symptoms and signs. See previous.

Examination. Seidel Test and application of fluorescein and slit lamp observation to check for aqueous humour leaks.

Treatment. Surgical repair and use of tissue adhesives such as cyanoacrylate are indicated when there is loss of substance.

These conditions must have a negative Seidel test.

Healing. Between 2 and 4 months.

Sequelae. Corneal scarring, which depending on severity may require corneal transplantation.

Penetrating wound with iris prolapse

2 types have been described: simple (in which maintenance of the anterior chamber and pupillary deformation is observed) and stellate (difficult to handle and complex).

Symptoms and signs. Photophobia, pain, tearing, blepharospasm and pupillary displacement.

Examination. Direct and indirect ophthalmoscopy.

Treatment. In both situations iris vitality and contamination will be assessed to decide whether to keep it (more likely) or section it. Any corneal wound must be referred with the urgency of the case due to the oedema it generates hampering suturing.

Healing. About 1 month where prolapse is minimal. In cases of greater prolapse, 2 to 3 months.

Sequelae. Pupil displacement, corneal scarring.

Penetrating wound with involvement of the lens

It is caused by sharp objects.

Symptoms and signs. The central wound can be observed. Traumatic cataract due to opening of the anterior lens capsule can occur in the anterior chamber occupied by crystalline masses.

Examination. Direct ophthalmoscopy, biomicroscopy.

Treatment. Need for surgery, corneal repair and treating the cataract and optical rehabilitation. Corneal transplant to be considered immediately and preferably.

Healing. 2 to 4 months.

Sequelae. Cataract, IOL placement.

Penetrating wound with vitreous loss

Probably involves lens injury.

Symptoms and signs. Acute pain, blurred vision, decreased visual acuity and pupillary abnormalities. The presence of the vitreous is recognized by its stretchiness when touched with a micro sponge.

Examination. Ophthalmoscopy.

Treatment. Will need suturing, vitrectomy, evaluation and final treatment for the retina and vitreous unit.

Healing. Between 4 and 6 months.

Sequelae. Retinal detachment, lens dislocation, retinal haemorrhages.

Sclerocorneal lesions

They can be simple wounds or sclerocorneal wounds which are usually associated with uveal tissue prolapse, vitreous.

Symptoms and signs. Acute pain, decreased visual acuity, blurred vision, photophobia and evidence of the place of rupture.

Examination. Biomicroscopy, ophthalmoscopy.

Treatment. Suture. In cases where there is an abundant vitreous haemorrhage, it will be necessary to perform vitrectomy to avoid traction on the retina leading to its detachment.

Healing. From 2 to 3 months if there are no complications.

Sequelae. Retinal detachment, corneal scarring, lens luxation.

Posterior scleral (behind the equator) or posterior scleral penetrating lesions

They are 10-20%.

Symptoms and signs. Decreased visual acuity. Alteration of anterior chamber depth (increase or decrease). Ocular hypotony, pupillary distortion (deformity of the pupil towards the site of deformation), uveal prolapse (dark brown).

Examination. When the medical history suggests a small injury and/or intraocular foreign body surgical examination will be required in addition to other diagnostic tests, CT scan, to assess the possible presence of a foreign body. MRI should never be used as the foreign body is probably made of metal.

Treatment. Handle as little as possible and avoid any pressure on the eye involving extrusion of ocular contents. Emergency surgery. Systemic and local antibiotic prophylaxis. Local corticosteroids and sterile ocular dressing. In cases of oedema is important to administer systemic corticosteroids.

Healing. 4 to 6 months.

Sequelae. Deformity of the pupil, retinal haemorrhage and retinal detachment.

Intraocular foreign body (IOFB)

May or may not be associated with a penetrating wound but with history of an indicative mechanism, for example, breaking rocks, hitting metal (usually an occupational origin).

Symptoms and signs. Moderate to severe inflammatory reaction depending on the nature of the foreign body: iron, steel (magnetic), copper or plant (non-magnetic) cause a severe inflammatory reaction.

Nickel (magnetic), aluminium, mercury or zinc (non magnetic) cause a moderate inflammatory reaction.

Carbon, glass, plastic, stone, silver and platinum are inert.

In the event of a puncture wound, proceed based on the involved structures.

Examination. Fundoscopy is not always helpful since the medium is often not transparent. Biomicroscopy, direct and indirect ophthalmoscopy to specify the path and associated ocular damage. Ultrasound or CT should be used. MRI should not be done.

Immediate treatment. Hospitalisation, sterile dressing, tetanus prophylaxis and systemic antibiotics. Cycloplegic agents.

If the visibility conditions are good, it has to be removed when repairing the wound. When there is heavy bleeding or an unclear situation, remove the IOFB after primary wound closure. Bear in mind that if it is made of copper or iron it always has to be removed, since its oxidation can cause siderosis/calcinosis with irreversible functional loss; if it is made of well tolerated material it will not always be necessary to remove it.

Healing. From 1 to 2 months.

Sequelae. Delayed consequences that must be monitored: formation of anterior synechiae of the iris in the anterior chamber angle and therefore secondary glaucoma, traumatic retinal lesion and secondary detachment.

Inflammatory reactions by type of material, uveitis and hypopyon, atrophy and hypotonia of the eyeball (*ptthisis bulbi*), fulminant endophthalmitis.

Irreparable penetrating sclerocorneal wound

There are cases which are difficult to repair caused by loss of tissue or extensive destruction of the eyeball.

In these cases attempts should always be made to repair or perform evisceration of the globe whenever the patient authorises it. Evisceration or enucleation has a preventive role in preventing the development of sympathetic ophthalmia in the healthy eye.

Treatment. Broad spectrum antibiotics administered parenterally, anti-inflammatory drugs, occlusive patch, tetanus toxin or gamma globulin (depending on patient history) and surgical repair or enucleation.

Healing. 30 days.

Sequelae. Eyeball avulsion with the functional impact it entails.

3. Chemical trauma

3.1 Ocular burn

They are substances that produce a burn on the tissue with which they come into contact. The harmful effect of ocular chemical burns depends on the nature and type of substance and the contact time. Limbus ischemia clinically marks the extent and severity of chemical burns.

They are produced by a wide range of elements including acids, alkalis, detergents, solution media, adhesives, irritants (tear gas).

The most common alkali agents are ammonia, lye, potassium hydroxide, magnesium hydroxide and lime. The most frequently involved acids are sulphuric, sulphurous, hydrofluoric, hydrochloric, acetic and nitrous.

In general, bases are more dangerous than acids. The main feature of acid involvement is their limitations, since they cause denaturation and coagulation of tissue proteins forming insoluble compounds constituting a barrier against thorough penetration of the chemical agent. For this reason most acid burns are limited to the epithelium forming shallow scars and their toxic effect is not progressive. Some acids do have profound effects comparable to alkalis (concentrated sulphuric acid, hydrofluoric acid and nitric acid).

The main feature of alkalis is diffusion as they dissolve tissue proteins with thorough penetration. They tend to react with cellular lipid fractions, forming hydrosoluble compounds that easily penetrate the corneal stroma and sclera. They produce tissue necrosis by vascular ischemia. Their adverse action depends on the concentration of the alkali and duration of exposure. From the point of view of prognosis, they cause more serious injuries that can affect the entire anterior chamber.

Symptoms and signs. Pain (from foreign body sensation to severe pain), blepharospasm and decreased visual acuity. The main signs range from conjunctival hyperaemia or corneal epithelial erosion, which will not leave sequelae in mild cases, to conjunctival ischemic necrosis and corneal opacities which may result in residual leucomas and possible corneal neovascularisation. In the most severe cases massive ischemic necrosis will occur with an opalescent white cornea that may involve perforation.

Depending on the degree of ocular involvement:

- Grade I: conjunctival hyperaemia and corneal epithelial defects without deep lesions of the eyeball.
- Grade II: conjunctival hyperaemia and chemosis with partial limbus ischemia.

- Grade III: ischemia affecting more than half of the limbus. Corneal opacity may be observed.
- Grade IV: extensive conjunctival necrosis that reveals white avascular sclera, “porcelain” appearance. Ischemic lesions exceed 75% of the limbal circumference. Very evident corneal opacity.

Direct and indirect biomicroscopy and ophthalmoscopy examination.

Complications. Injury to the iris, perforation of the eye and eyelid deformation. Severe burns of the cornea can result in the formation of a crust, perforation of the eye and blindness.

Treatment. Ocular chemical burns are treated immediately by cleansing the open eye with water (for at least 10 minutes); it is essential to eliminate the chemical as soon as possible. Treatment under local anaesthetic, mydriatic cycloplegic eye drops, antibiotic ointment and oral analgesics. Severe burns may require treatment by an ophthalmologist to preserve sight and prevent further complications.

Healing. Average healing time between 7-90 days depending on severity and extent.

Sequelae. Mild cases heal without sequelae, while in severe ones relapsing ulcers, scars, neovascularisation areas or glaucoma may persist which may require surgical repair.

3.2 Physical trauma

3.2.1 UV radiation. actinic or photoelectric keratitis

Corneal injury caused by the direct effect of ultraviolet radiation on the cornea.

Ultraviolet radiation is the most common cause of injury after sun exposure at the beach or in the snow. UV radiation only damages corneal epithelium which results in necrosis; its penetration ability is very poor.

Symptoms and signs. After a few hours without symptoms, it begins with foreign body sensation, tearing and photophobia which persist for 12-24 hours, pain, acute blindness, blepharospasm. It is usually in both eyes. The symptoms (severe pain) appear 8-12 hours after exposure and there is no relationship between the exposure time and intensity of clinical manifestations.

Examination. Clinical diagnosis based on history and complete eye examination.

Treatment. Topical cycloplegic eye drops. Topical antibiotic ointment and binocular occlusion. Oral analgesic. Sedative by mouth for easy night's rest.

Healing. From a few days to a month, depending on the severity and extent of the injury.

Sequelae. In general it progresses favourably and clears up within 24-48 hours without leaving scarring sequelae.

3.2.2 Solar retinopathy

Injury produced by sunlight in people who watch a solar eclipse. A small burn occurs on the retina that does not take up its entire surface and results in decreased vision. Household laser pointers could theoretically produce retinal photocoagulation (staring at a pointer for more than 10 seconds) but do not usually bring about impairment since exposure time is less than 1 second.

Symptoms and signs. Decreased visual acuity and central scotoma.

Examination. On examination of the fundus central retinal injury, rounded morphology with sharp edges and yellowish/whitish in colour is evident.

Complications. Not to be expected.

Treatment. Required. Visual acuity is fully recovered in the following months.

Healing. Healing time is different depending on the severity of injury and can range from hours to weeks.

Sequelae. Not to be expected.

3.2.3 Ionising or infrared lesions

They can be seen after irradiation of tumours in the face or nuclear accidents. The penetration of the eye depends on the type of radiation.

Symptoms and signs. The lesions always have a latency period. There can be loss of eyelashes, eyelid depigmentation accompanied by blepharitis and dry eye as a result of conjunctival injury with loss of goblet cells. After a period of higher latency ischemic retinopathy and loss of visual acuity may appear.

Examination. Clinical diagnosis based on history and complete eye examination.

Treatment. The best treatment is prevention, i.e., proper eye protection when facial tumour irradiation is performed. If the lesion has occurred it can be treated with artificial tears, panretinal photocoagulation with argon laser and cataract surgery.

Healing. Depending on the involvement it will range from 15 to 45-60 days.

Sequelae. Retinopathy may occur months after irradiation as ischemic retinopathy with haemorrhaging, cottony exudations, vascular occlusion and retinal neovascularisation. Years later a loss of visual acuity due to cataract development may be noticed.

3.3.4 Thermal burns

Heat can damage the eye directly or, more often, indirectly due to the large amount of heat released in the chemical reactions that follow eye contact with certain substances. The eyelid is the structure most involved followed by the cornea.

They can be produced by flames, hot steam, boiling water, splashing hot grease or an incandescent metal and explosions causing thermal coagulation of the corneal and conjunctival surface. They are injuries comparable to acid burns and produce surface coagulation. The eyelids are always involved due to the eyelid closure reflex.

The lesions produced depend on various factors: the temperature of the agent, the heat storing capacity of the material, duration and the contact area.

Symptoms and signs. Foreign body sensation or pain. Sometimes decreased visual acuity. Ciliary or conjunctival hyperaemia. If the cornea is affected, the epithelium will appear devitalised, oedematous and whitish.

Examination. Clinical diagnosis based on history and eye examination.

Treatment. Brief irrigation with saline or lactate solution or sterile water. After prior topical anaesthesia, check the fundus with a wet swab to remove foreign material. Occlusion with cycloplegic drops and antibiotic ointment. Check every 24 hours and replace the patch with cycloplegic drops and antibiotic ointment every day until the corneal injury has healed.

Healing. Mild burns heal without sequelae within a few days. More severe involvement will need a longer period (60 days).

Sequelae. Serious lesions which jeopardise functional integrity and eye appearance. Opacity of the lens and corneal scarring.

3.2.5 Electrical burns

They are injuries due to electrical shock when using electricity in the home and in industry. Cases have also been described in the literature in cardioversion and electroshock treatments and certain transurethral resection techniques.

Symptoms and signs. Virtually any part of the eyeball can be damaged by the passage of electric current. The most common eye injury after high-voltage electrical trauma is cataracts, which are most commonly in both eyes and late onset. There may be decreased visual acuity in the twelve months following the trauma. Secondary corneal perfora-

tion may occur in necrosis of the substantia propria due to electricity, albeit less frequently. Macular oedema may also appear.

Depending on the involved structures there may be:

- Eyelids: blepharospasm, burns, necrosis.
- Cornea: necrosis, perforation, leucoma.
- Iris: anterior uveitis, hyphema.
- Lens: anterior or posterior subcapsular vacuoles, anterior or posterior subcapsular cataract.
- Retina: macular oedema, macular pseudocysts, macular holes and pigmentary degeneration.
- Optic nerve: thermal optic neuritis.
- Orbit: orbit roof fracture, exophthalmos, paralysis of extraocular muscles.

Examination. ophthalmological examination assessing visual acuity, slit lamp examination and of the fundus under dilation.

Treatment. Lesions produced by electric current need medical and surgical treatment depending on the involved area. Cataracts require surgery for removal of the part of the lens that is cloudy and replacing it with an intraocular lens.

Healing. It will depend on the involved structures.

Sequelae. Cataracts, thermal optic neuritis, exophthalmos and paralysis of extraocular muscles may remain.

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Chapter XIII

Perforated Eardrum: basic medical attention/medical treatment

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1. Introduction

The perforation of the tympanic membrane entails a loss of its mechanical properties and consequently an alteration of its function not only as a transmitter of sound waves but also as a protective barrier for the middle ear. The proper functioning of the eardrum also depends on the structures that make up the middle ear, especially the Eustachian tube: tubal inflammation may be the cause of persistent perforation. In the medico-legal assessment of traumatic perforations of the tympanic membrane, we are often faced with the issue of whether this is a purely traumatic injury or a previous pathological condition is involved. Determining whether otitis is the cause of perforation or, on the contrary, a secondary complication is not always easy. When assessing bodily injury we have to establish a causal link between the traumatic antecedent and the injury and this is a complex task since in many cases the medical information we have in our possession is sparse and examination of the patient in the forensic medicine clinic is delayed. Our challenge as medical forensic experts is to interpret existing medical reports with the utmost rigour, correlate them with objectified clinical data at the time of the forensic medical visit and establish their causal relationship with the traumatic antecedent. The aim of this paper is to provide a broad and comprehensive overview of the causes, contributing causes and consequences of a perforated eardrum in the belief that we can only achieve a proper medico-legal assessment when we include all possible aetiologies in the range of differential diagnoses.

* Group Leader

2. Anatomy of the ear

The ear is a highly complex sensory organ that responds to sound and position changes. Its functions are hearing and maintaining balance. Anatomically it is divided into three parts: the outer ear, the middle ear and the inner ear.

2.1 Outer ear

Formed by the pinna, the external auditory canal (EAC) and the tympanic membrane, which is a thin layer that separates the outer from the middle ear. The function of the EAC is to conduct the vibration caused by variation of air pressure from the pinna to the eardrum.

2.2 Tympanic membrane

9 mm in diameter, elliptical and umbilicated in the centre where the handle and short process of the hammer are inserted. Insertion into the temporal bone is by means of a fibrous ring. A normal tympanic membrane is semitransparent and glossy grey in colour.

2.3 Middle ear

Located in the temporal bone with the chain of bones (hammer, anvil and stirrup) whose function is the connection and conduction of vibrations between the eardrum and the inner ear. Its outer side is formed by the tympanic membrane. The inner side communicates with the inner ear through the oval and round windows. The anterior side communicates with the nasopharynx via the Eustachian tube. The anterior wall communicates with the mastoid air cells. The upper part is formed by a thin bone layer separating the middle ear from the middle cranial fossa.

2.4 Inner ear

Located inside the petrous part of the temporal bone, it includes the essential peripheral organs of hearing and balance.

3. Physiology of hearing

The human ear is the organ responsible for the collection, transmission, transduction and coding of sound in bioelectric impulses and conducting them to the cerebral cortex. The process begins with the capture of sound in the pinna, the sound energy is amplified in the EAC, hits the eardrum and moves the chain of bones that transmit the acoustic energy to the perilymph through the oval window. The Eustachian tube plays a fundamental role since it enables ventilation of the tympanic cavity.

4. Aetiology of tympanic perforation

4.1 Barotrauma

Injury caused by sudden changes in pressure in the middle ear. Faced with sudden hyperpressure in the middle ear, the tube passively opens and lets the air through, but in the case of abrupt hypopressure the action of the peristaphyline muscles will be required to open the tube and let the air through from the nasopharynx to the ear. When atmospheric pressure is greater the nasopharynx mucosa is invaginated by the tubal ostium and acts as a valve, preventing ventilation. We can distinguish between different clinical forms based on their aetiology.

4.1.1 *The bends*

It is a professional and/or sports disease. Essentially it occurs during abrupt ascents without decompression. Nitrogen passes by diffusion into the blood, lungs and tissues as small air bubbles, causing clinical symptoms of pain, arthralgia, headache, paresthesia, pulmonary embolism, dyspnea and coronary ischemia. In quick dives, the sharp increase in pressure (1 atmosphere every 10 metres) can cause a perforated eardrum which combined with the stimulatory effect of the cold water on the labyrinth can disorientate the diver with potentially serious consequences. The involvement of the ear may lead to progressive sensorineural hearing loss, dizziness, vomiting and tinnitus.

4.1.2 *Blast*

A pressure wave occurs which together with the acute trauma of the sound of the explosion determines the damage. The wave first generates hyperpressure which causes eardrum subsidence, compressing the

chain of bones and collapsing the tube. The pressure is then abruptly reversed with a suction effect on the structures which causes important injuries to the middle ear such as fractures or dislocations of the chain of bones, eardrum perforation, bleeding, etc. The inner ear may also be damaged with significant impact on subsequent hearing.

4.1.3 Aero-otitis

In sharp ascents to or descents from significant heights (aircraft).

4.1.4 Barotrauma due to a direct blow to the ear

A common clinical form in gender violence due to a slap on the ear. The sudden change in atmospheric pressure can lead to blockage of the tube, a perforated eardrum and other ear injuries.

4.2 Direct trauma

4.2.1 Fracture of the petrous part of the temporal bone

Ear involvement will be secondary given the severity of the trauma. It can lead to significant functional sequelae. Half of skull base fractures affect the petrous part of the temporal bone, of which between 15 and 30% leave ear sequelae. Longitudinal fractures due to a blow to the temporal or parietal region may affect its path to the EAC and middle ear. In 20% of cases it also may affect the facial nerve.

4.2.2 Instrumental injuries

Eardrum perforations produced directly by cotton buds, needles, tweezers or other objects inserted into the inside of the EAC.

4.3 Burn injuries

Due to hot metal welds, acids, incandescent particles. They can occur in the workplace.

4.4 Middle ear infections

The collection of secretions in the middle ear can cause spontaneous rupture or sprain of the tympanic membrane with drainage of serous, pus and/or hemorrhagic fluid.

4.4.1 Acute otitis media, secretory otitis media and serous otitis

After the acute phase, they usually progress to healing with scarring of the tympanic membrane. Evolution towards chronicity depends among other things on the normalisation of the function of the tube.

4.4.2 Benign chronic suppurative otitis media

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Chronic inflammation of the middle ear mucosa, with repeated episodes of infection, otorrhea and permanent perforation of the eardrum without bone involvement of the tympanic frame.

4.4.3 Chronic otitis media cholesteatoma

Ear cholesteatoma can be defined as the growth of epithelial tissue in an abnormal location behind the tympanic membrane. Its origin may be congenital (remnants of embryonic tissue in the ear) or acquired. Acquired cholesteatoma is secondary to retraction of the eardrum, perforations and/or recurrent infections. It grows slowly but expansively and takes the form of a pseudotumor invading the tympanic cavity and the mastoid process. It can also erode and destroy the chain of bones.

4.5 Iatrogenic perforations

4.5.1 Grommets

Surgical technique widely used in paediatrics and especially indicated in serous otitis or recurrent acute otitis media, chronic tympanic membrane retractions, tubal problems and in all cases causing conductive deafness and which have a negative impact on the early years of childhood. Grommet surgery requires a small incision in the tympanic membrane, known as myringotomy, with placement of drainage. Grommets help prevent complications of chronic otitis, such as tympanic membrane perforation, scarring of the eardrum or the chain of bones, adhesive otitis media and/or the development of tympanic retraction pockets. Grommets usually fall out on their own between 3 and 6 months following insertion. In 1% of cases late complications may occur with persistent perforation after the drainage has fallen out. This possibility is essential information before surgery.

5. Clinical signs of perforated eardrum

5.1 Immediate earache

Variable intensity and subsides on its own.

5.2 Serous or serosanguineous otorrhea

If purulent, prior infectious disease should immediately be considered. If purulent discharge occurs later, the possibility of an infection of the middle ear secondary to perforation should be considered.

5.3 Intermittent or permanent tinnitus

5.4 Hearing loss

Proportional to the severity of the perforation. Hearing loss can be severe in cases of severe trauma, fracture of the petrous part of the temporal bone, dislocation or fracture of the chain of bones or injury of the inner ear.

5.5 Dizziness

Dizziness may be due to injury of the inner ear.

6. Diagnosis of perforated eardrum

It will be based on medical history, clinical examination by direct otoscopy and additional examinations.

6.1 Medical history

The patient's history is essential and must include their pathological antecedents, symptoms, form of presentation, etc. A traumatic perforated eardrum will usually be acute and on one side.

6.2 Clinical examination

Physical and otoscopic examination to see the perforation. A central perforation does not involve the edge of the eardrum (the annulus)

whereas a marginal perforation does. This is important because central perforations heal better than marginal perforations. Acoumetry with a tuning fork is an excellent diagnostic test for hearing loss and can adequately distinguish between conductive hearing loss (negative Rinne and Weber to the affected side) and perceptive hearing loss (positive Rinne and Weber to the healthy side).

6.2.1 Rinne test

The tuning fork is struck with a sharp blow and placed about 2-3 cm away from the EAC with the tines parallel to the ear (airborne conduction). Quickly, and without diminishing the intensity, the base of the tuning fork is placed on the mastoid process (bone conduction). Normally air conduction will be heard best or Rinne +.

6.2.2 Weber test

The tuning fork is struck with a sharp blow and placed on the frontal bone of the patient. Under normal conditions the sound is heard centrally. In conductive hearing loss the sound paradoxically moves to the ill ear while in sensorineural hearing loss it moves towards the healthy ear. The Rinne and Weber tests enable us to determine whether it is a case of conductive or sensorineural hearing loss. Most eardrum perforations lead to conductive hearing loss. If the injury has not been great, perceptive or sensorineural deafness is unlikely to occur. The presence of perceptive hearing loss with low intensity trauma will suggest pre-existing hearing loss (occupational acoustic trauma, chronic or recurrent otitis media, etc.).

6.3 Tone audiometry

It consists of stimulation with sounds at different frequencies to determine the hearing threshold (minimum sound intensity level the ear can hear for each tone). It should include audiometry by air conduction (bass, mid and treble tones that the patient can hear at their lowest intensity with a headset on the ear) and bone conduction (bass, mid and treble tones that the patient can hear at their lowest intensity with a device placed on the mastoid). The results are shown in an audiometric curve consisting of 2 plots for each ear. One of them determines bone conduction hearing and exclusively assesses the cochlear function and nerve pathways. The other assesses air conduction, i.e. the function of all the anatomical elements involved in hearing (outer ear, eardrum, chain of bones, cochlea and central pathways).

6.3.1 Normal audiometry

Overlay of bone and air plots above 20-30 dB.

6.3.2 Conductive hearing loss

Normal bone conduction plotting with pathological air conduction. The separation between the two hearing curves is called a gap and is the result of damage to the outer or middle ear.

6.3.3 Perceptive hearing loss

The air and bone plots descend in parallel.

6.3.4 Mixed hearing loss

It is a combination of conductive and perceptive hearing loss.

6.4 Speech audiometry

It assesses the speech intelligibility or understanding threshold, especially in perceptive hearing loss. It is a specialised technique and rarely used in our field.

6.5 Impedance audiometry

It is extremely useful in children and makes it possible to study conductive hearing loss. Requires the integrity of the tympanic membrane. Infrequently used in our field.

6.6 Otoacoustic emissions and evoked potentials

Useful in the assessment of hearing loss in young children, uncooperative adults and simulators.

7. Differential diagnosis of perforated eardrum

Otосcopy with seeing the eardrum will be essential for correct diagnosis and very useful for differential diagnosis. Faced with an acute perforation we will have to assess whether its cause is traumatic or non-

traumatic (acute or chronic otitis media, cholesteatoma, etc.). Acute traumatic perforation will have damaged margins with traces of blood with or without serous or serosanguineous secretion. If the perforation is due to chronic otitis media, the margins will be rounded with mucopurulent secretions and the eardrum will be dull and enlarged. If the examination takes place later on and the perforation has probably healed, we will see a scar on the eardrum. In these cases, we will need to assess the medical information and previous examinations. When the perforation is chronic, we will need to consider an underlying disease as a trigger and ask for information relating to the previous condition of the patient.

8. Treating a perforated eardrum

Simple, clean perforations which do not affect the fibrous ring will heal spontaneously during the first weeks. Large perforations with involvement of the fibrous ring and/or are complicated may evolve into chronic perforation. Treatment of simple perforations is to relieve pain and prevent infection. Surgery must be assessed in the case of complicated perforations or ones that do not heal spontaneously.

8.1 Physical and symptomatic measures

Heat application may relieve ear pain. Analgesics are to be administered.

8.2 Preventive treatment

It is important to keep the EAC clean and dry (prevent water getting in when showering, swimming, etc.). Antibiotic treatment may be indicated as prophylaxis against the possibility of contamination of the middle ear (septic water, introduction of contaminated objects, etc.).

8.3 Medical and/or surgical treatment

Antibiotics are to be administered for otitis secondary to perforation. When the eardrum does not heal spontaneously surgery should be evaluated.

8.3.1 Myringoplasty

Repair only of the tympanic membrane in inactive perforations with the chain of bones intact and the Eustachian tube functioning. Closing up the perforation with a graft improves hearing, reduces tinnitus and prevents the ingress of water into the middle ear, thereby avoiding the risk of infection and preventing the development of cholesteatoma. This surgical technique is indicated for chronic perforations lasting more than 2 months. The surgical approach may be endoauricular (via the EAC) or retroauricular (incision behind the ear). The temporalis muscle fascia is often used as a graft.

8.3.2. Tympanoplasty

Technique used when in addition to the perforation there are injuries to the chain of bones. Prostheses are designed to replace and reconstruct the chain of bones in whole or in part. Partial ones are used when the stapes is preserved and are placed between the stapes head and the tympanic membrane and/or the hammer. Total prostheses are indicated when only the stapes footplate has been conserved or has been replaced by a graft on the oval window. The prosthesis is positioned between the footplate or graft and the tympanic membrane or hammer. This surgical technique is only indicated when there is no infectious process in the ear, there is conductive hearing loss and the mobility of the stapes footplate will enable sound to reach the cochlea.

9. Perforated eardrum complications

9.1. Infections

Acute otitis media can present as a complication of perforation and the latter may be complicated by mastoiditis, brain abscess, adhesive otitis, scarring sequelae, etc.

9.2 Persistent symptoms

Some patients may have tinnitus after a perforated eardrum, sometimes persisting after healing.

9.3 Permanent hearing loss

A perforated eardrum is often accompanied by conductive hearing loss at less than 30 dB. Its severity is proportional to location and size (greater in tense parts than in flaccid ones). When healed recovery is complete and no residual hearing loss greater than 20 dB is observed. If the hearing loss persists at 4-6 weeks, injuries to the middle or inner ear will have to be ruled out. Persistent sensorineural hearing loss will suggest involvement of the inner ear.

10. Medico-legal aspects

10.1 Healing time

95% of traumatic perforations without complications or involvement of the middle or inner ear have a good prognosis with complete healing without surgery and without sequelae. Healing time varies depending on the author cited at between 30 and 60 days. The worst prognosis is when perforation is accompanied by tubal dysfunction or inflammatory and/or infectious pathology. It needs to be assessed whether the pathology is the result of the complication of the perforation or alternatively the perforation is a consequence of a previous pathology. In these cases treatment of complications can lengthen the healing period.

10.2 Lost and non-lost work days

Generally speaking mild perforated eardrum without involvement of the internal structures will not be an impediment to normal activities except in cases of people whose work exposes them to water such as swimmers, divers, etc.

10.3 Treatment

10.3.1 Basic medical attention

Treatment is symptomatic and prophylactic for simple, uncomplicated tympanic membrane perforations without damage to the structures of the middle and inner ear.

10.3.2 Medical and surgical treatment

When treatment for healing acute injuries and/or chronic is imperative. Antibiotic treatment of otitis media secondary to perforation. Surgical treatment of chronic perforations.

10.4.1 Sequelae

10.4.2 Chronic eardrum perforation

It can be considered chronic when it persists after 3 months or in recurrences after myringoplasty or tympanoplasty. It will lead to repeated infections, ear discharge, hearing loss and relative limitation on activities (swimming).

10.4.3 Chronic middle ear infection

A frequent sequela when there is pathology associated with the ventilation system of the middle ear (obstruction of the Eustachian tube).

10.4.4 Cholesteatoma

Marginal perforations may evolve to cholesteatoma more frequently.

10.4.5 Hearing loss

A distinction has to be drawn between perceptive hearing loss (due to injury to the cochlear, neuronal pathways or the central nervous system), conductive hearing loss (due to alterations of the outer or middle ear that prevent normal conduction of sound) and mixed forms.

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Chapter XIV

Assessment of Scaphoid Fractures: Old or Recent

Authors: Dr. Mercè Subirana Domènech* and Dr. Horacio Méndez López

1. Anatomical introduction

The carpal scaphoid is part of the first carpal row and articulates with the radius, lunate, trapezium and trapezoid. It is an indispensable element in the radiocarpal joint, carpometacarpal joint for the thumb and the intercarpal joints.

It participates in the abduction, rotation and opposition of the thumb. The first metacarpal transmits its motion to the trapezoid, which slides over the scaphoid.

The scaphoid has poor irrigation as 4/5 of it is covered with articular cartilage and it is the dorsal portion which receives irrigation between the middle and distal third (the dorsal parts of the radial artery). Hence tubercle or distal pole fractures heal quickly and do not usually lead to necrosis problems while the reverse is the case with proximal pole fractures.

2. Mechanism of production of fractures

They are usually found in young adults due to falling over with the wrist in hyperextension or forced dorsiflexion (at least 95° to produce the fracture). They may be associated with fractures of the distal radius epiphysis or ligament injuries.

Other mechanisms: violent movement by the hand in violent dorsiflexion not involving falling over.

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3. Classification of the fractures

- *By location*
 - Middle third (most common)
 - Proximal third
 - Distal third
 - Compromise of a marginal sector of the scaphoid tubercle (rare)
- *By the fracture line with the longitudinal axis of the scaphoid*
 - Horizontal oblique line (stable)
 - Transverse line (stable)
 - Vertical oblique line (unstable)
- *By displacement*
 - Not displaced (distance of less than 1 mm between the fragments)
 - Displaced: with separation, angulation or rotation of the fragments
- *By the compromise of the other carpal bones*
 - Isolated fracture: only affects the scaphoid
 - Associated fracture: with dislocations of other carpal bones

4. Clinical picture

Pain in the radial half of the wrist which intensifies with pressure on the anatomical snuffbox but nonspecific as it can also be seen in wrist sprains, Bennett fracture of the first metacarpal and radial styloid fracture.

Mild functional limitation: flexion-extension of the wrist.

Mild pain and disability for thumb and forefinger gripping and clamping movements.

Mild pain on axial pressure of the thumb in extension against the carpus.

Inflammation and oedema.

Watson's test: pain on the scapholunate interlining and a projection or clunk when compression is applied with the wrist tilted.

It may be present in many patients in a mild, asymptomatic form (when there is fibrous union) and go unnoticed by the patient or doctor.

When there is a history of trauma of falling with the wrist in hyperextension or forced dorsiflexion (minimum 95° to produce the fracture) or violent movement with violent dorsiflexion without falling and with mild or moderate symptoms, radiological study is needed to rule out scaphoid fracture. Diagnostic suspicion should be maintained until proven otherwise.

5. Radiology

5.1 Plain x-rays

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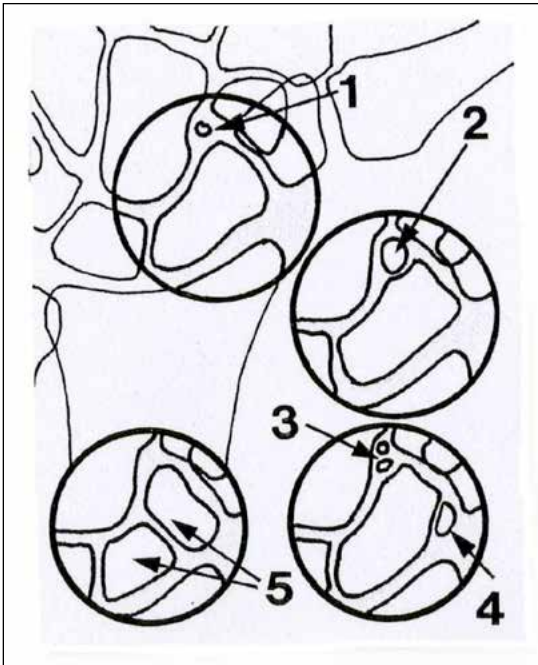
A scaphoid fracture is very often an *occult fracture* in 2-5% of cases and is detected by repeating radiology about 10 days after immobilisation, when the fracture edges have decalcified and there is a more visible radiological image. Radiology includes PA and LATERAL projections of the wrist. Furthermore, in one of them there will be PA projection with ulnar drift of the hand. In the other the hand is lifted by 40-45° supporting the ulnar side of the hand, with the fingers together and the thumb in front of them.



Special projections to be used in case of doubt are the Schneck projection, in which the tip is pulled with ulnar drift. Most often there is minimal displacement of the fracture (20% upper pole, 70% neck and 30% base)

There are 20% false positives in scaphoid X-rays and clinical diagnostic confirmation is required. Several ossification anomalies can be confused with fractures. The *central bone* may be small, large or double (figure). Three radiological patterns of presentation of the central carpal

bone have been described: firstly, as an independent and well-defined, single or double ossicle, the second as a formation with partial separation, and the third as an empty space(5) [1,2]. The *external radial bone* may be in the region of the tubercle of the scaphoid, although in some cases it may simulate an old unconsolidated fracture. It is thought that a “bipartite scaphoid” may result (rounded edges differentiate it from a fracture) and which may be bilateral [3,4,5].



1. Small central bone. 2. Large central bone. 3. Double central bone
4. External radial bone that may be in the region of the scaphoid tubercle and in some cases may appear to be an old unconsolidated fracture: it is now believed that the *bipartite scaphoid* (5) may result from this anomaly. The rounded and regular edges will differentiate it from a fracture.

- There are a number of signs that might lead us to think of an old fracture when assessing the initial X-ray:
- The space between the fragments is similar in appearance and distance to that of the other carpal spaces.
- Similar or even more marked sclerosis than the subchondral sclerosis in the rest of the carpal.
- Degenerative changes around the fracture.
- Variation in the space between the two fragments in a 4-position study.
- Large cystic cavity [6].

Consolidation is checked with more X-rays after 6 months.

Changes characteristic of delayed consolidation: Simonian and Markiewitz argue that there is delayed consolidation after four to six months have elapsed and non-union when evolution is more than 6 months [7,8].

There will be a series of radiological changes characteristic of non-union (pseudoarthrosis), such as:

1. Bone sclerosis
2. Cystic formation
3. Widening of the scapholunate space
4. Bone resorption
5. Secondary osteoarthritis

5.2 Computed tomography

Computed tomography (CT) is useful in the early detection of different varieties for proper treatment planning. Demonstration of sclerosis, suggestive of avascular necrosis, is similar in radiology and CT, although CT provides higher spatial resolution.

5.3 Magnetic resonance imaging

Magnetic resonance imaging (MRI) confirms or rules out fracture and makes it possible to assess the status of other carpal bones and capsuloligamentous structures. It detects *occult* injuries in the initial phase of injury and this makes it possible to specify the cases where there actually is a fracture which will alter treatment [9]. When non-unions are established it displays cortical interruption areas worse but better identifies trabecular bone injury and identifies fibrosis and cartilage matrix areas characteristic of the reparative phase and sometimes interposed synovial fluid (a sign of lack of consolidation). It has greater sensitivity and specificity than scintigraphy, greater anatomical resolution and definition of the degree of bone involvement: oedema, trabecular fracture, presence of bone necrosis. Bone sclerosis in RX/CT does not entail necrosis: enhancement after contrast administration rules it out.

5.4 Scintigraphy

It is a very sensitive method to detect bone or ligamentous pathology in cases with significant clinical signs and standardised normal radiographs. Its main disadvantage is a lack of specificity which requires further studies to provide a diagnosis. As in all fractures with

periarticular involvement, there will be high radionuclide uptake at 24 hours after injury. Uptake will be maximal at 2-5 weeks. Nevertheless, in cases of reflex sympathetic dystrophy, specificity and sensitivity can be up to 95%.

6. Treatment

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Initially a forearm metacarpal cast including the column of the thumb and which leaves the interphalangeal joint free, with the wrist in a neutral or slightly extended position.

Immobilisation varies between 6 weeks for scaphoid tubercle fractures, 8 weeks for distal pole fractures and 12 weeks for proximal pole fractures.

Tubercle fractures heal quickly in about a month. The rest may require 2 or 3 months of immobilisation.

Distal end and tubercle fractures have a good prognosis, consolidation in middle third fractures may sometimes be delayed and they may evolve to non-union, while proximal pole fractures heal most slowly, and sometimes a small free fragment may be left in those called *cap* or *subchondral*.

Evolution without treatment leads to DISI (dissociative scapholunate instability) with carpal collapse in 10 years.

6.1 Absolute surgical indications:

Old or recent fractures displaced more than 1 mm, associated or not with carpal dislocations. Proximal fractures seen late.

6.2 Relative surgical indications:

Non-displaced fractures in patients requiring rapid mobilisation of the extremity due to other causes (work, sports, etc.).

If surgery is needed volar access is recommended since there is less risk of injury to the dorsal vascular contribution. The fracture is reduced and fixed with a compression screw (Herbert) and repair of ligamentous structures in the case of associated carpal dislocations, sometimes requiring double access (dorsal and ventral). If there is comminution of the fracture site then bone grafts (iliac crest or metaphyseal area of the radius) are justified. Consolidation is between 5 and 12 weeks with an average of 9 [10].

7. Complications

7.1 Delayed consolidation and non-union:

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In cases of inadequate treatment or delayed diagnosis. This causes a focus resorption, decreased carpus height and alteration of the intercarpal axis which inevitably leads to secondary radiocarpal osteoarthritis. In scaphoid non-union, conventional MRI sequences cannot determine the vascular status of the proximal fragment. MRI with gadolinium (fat-suppressed T1-weighted sequences in the coronal and oblique sagittal planes following the long axis of the scaphoid) makes it possible to accurately determine the vascular status of the proximal fragment, the main prognostic factor in the treatment of these patients. Their treatment will vary according to the stage they are at.

7.2 Avascular necrosis (AVN)

Sclerosis on conventional radiography and CT is not a reliable sign of AVN. Scintigraphy is nonspecific and MRI contrast is the method of choice in the diagnosis of this entity.

7.3 Densification of the proximal fragment

This is not to be confused with necrosis but rather it is delayed revascularisation of the fragment confirmed by scintigraphy. This can cause a weakening of the cartilage and secondary osteoarthritis. It has to be monitored to make sure it does not evolve into necrosis around the sixth month.

7.4 Complex regional pain syndrome

Rare in these fractures.

7.5 Malunion

In most cases it is due to displaced fractures which were not reduced or incompletely reduced. Long-term evolution is the same as non-union [11].

8. Sequelae

The range of sequelae resulting from scaphoid injury is very wide since it is a bone that affects not only the distal part of the carpus but also significantly compromises the proximal part whether with associated injuries or not.

Moreover, various complications can lead to the same sequela and complication in generate more than one type of sequela.

There are also syndromes described as a sequel which are a complication in themselves. Examples are complex regional pain syndrome and Südeck's atrophy (comparable to residual or reflex sympathetic dystrophy).

Sequelae should be quantified using the scale established in Act 34/2003 and their definitions applicable to the following complications:

- Delayed consolidation and non-union.
- Densification of the proximal fragment.
- Avascular necrosis.
- Malunion.

All may be accompanied by posttraumatic arthritis.

- Complex regional pain syndrome.
- Südeck (as a complication associated with the evolution of the trauma).

Quantification of the sequelae:

A) In the *wrist* the following will be assessed

- A.1) The limitation to mobility specifying the types of movement and degree of reduction in each case.
- A.2) Forearm / wrist pain (1-5 points)
- A.3) Whether there are other carpal fractures and associated capsuloligamentous injuries.

B) With reference to the *hand*

- B.1) Whether there is inoperable scaphoid non-union (6 points)
- B.2) It will also be assessed whether there is difficulty in moving the index finger: ankylosis / arthrodesis of the index finger (including in all joints) in the functional position (7-10 points) and the non-functional position (10-15 points)
- B.3) Posttraumatic arthritis and hand pain (1-3 points), different from A.2
- B.4) The existence of osteosynthesis material (1-3 points)

- C) Consideration also has to be given to
- C.1) Complex regional pain syndrome, comparable to painful wrist (1-5 points) and / or hand pain (1-3 points), as both can be present at once.
 - C.2) Südeck syndrome or residual posttraumatic algodystrophy of the hand (1-5 points).
- We have already mentioned that they are complications in themselves or associated ones in the case of Südeck syndrome.
- D) An equally important aspect is disfigurement caused by scars and defects in the position of the affected joints.
- E) Whether these permanent injuries constitute incapacity for the injured person's work or activity has to be assessed.

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Chapter XV

Table IV of Act 34/2003

(Correction Factors for Basic Compensation for Permanent Injuries): PPD, TPD, APD and Severe Disablement

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In cases of injuries where the scale in Act 34/2003, of 4 November, is used once sequelae have been determined based on Table VI, it has to be assessed whether the permanent and irreversible sequelae qualify as permanent disability for the person's occupation or customary activity as partial, total or absolute permanent disability or severe disablement. Hence and only for those sequelae where it has been possible, we have attempted to establish unifying criteria to make it easier to determine the type of disability. However, each case needs to be studied individually and this study is only a guideline. At no time do we seek to provide a one size fits all solution to a problem that requires in-depth analysis on a case-by-case basis.

1. General grounds for using Table IV

Sometimes the issue has been raised whether a medical forensic expert should make an official statement about the existence of a particular degree of permanent disability when drawing up a health report about injuries.

This question can be answered without any hesitation, and that answer is *yes, always*.

When drawing up a forensic health report for insurance-related injuries, and in general by extension for any type of injury, we have to use the scale set out in Act 34/2003, and if we make statements about issues such as healthcare days (hospitalisation, lost work days and non-lost work days) which are in Table V or the sequelae that are in Table VI, it would be inconsistent if we then said nothing about permanent

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disability as it is in Table IV in the same scale.

If we fail to make a statement when there is a degree of permanent disability, we would not be fully assessing the impact of the injuries on a person and the court will have insufficient information to determine fair compensation.

2. Means of use

It should be done only under the parameters listed in Table IV which are:

- Partial Permanent Disability: permanent sequelae that partially restrict the person's occupation or usual activities without stopping them carrying out basic tasks in them.
- Total Permanent Disability: permanent sequelae that completely prevent the disabled person from performing their occupation or usual activities.
- Absolute Permanent Disability: sequelae that prevent the disabled person from doing any occupation or activity.
- Severe Disablement: permanent sequelae that require the help of others to perform essential activities of daily living such as dressing, moving, eating or similar.

Each of these degrees has its rightful compensation, but bearing in mind that it is not a specific amount as is the case with the points for sequelae in Table VI but rather a margin, it is important that when establishing the existence of a degree of permanent disability as full a description as possible of its impact on the person's activities is provided so that the court has the maximum possible information with which to award a sum within the margins provided by the scale.

Another issue to keep in mind is that we should not consider the possibility of not assessing permanent disability because it has not been recognised by the INSS (National Social Insurance Institute) if they have assessed it before us since the concepts are different. The INSS makes administrative decisions that not only take into account the sequelae but also assess other things, such as how long the person has been paying social insurance, waiting periods, contract conditions, etc., which have no influence on our expert report.

In addition, the definition of permanent disability and its degrees are not exactly the same in Table IV of the scale as in the Social Insurance Act. Thus the INSS only recognises paid jobs whereas Table IV talks about the "occupation or usual activity". Furthermore, for example, the definition of partial permanent disability is very different in the

Social Insurance Act where specific percentages for limitation have to be exceeded, while Table IV only says “that partially restrict” without requiring it to be to a certain degree or even significant.

Hence it may well be the case that a person who presents certain sequelae will have a certain degree of permanent disability recognised under Table IV while the INSS does not recognise it. This is a situation that can be compared with the medico-legal evaluation of healthcare days, which will not necessarily coincide with the days off work stated in the reports issued by the GP.

3. Suggested method for applying Table IV

Permanent disabilities are defined in Table IV of the scale as those affecting the “occupation or usual activity” of the injured person.

Occupation or activity does not only mean the person’s job or occupation but rather any activity they perform regularly such as:

- studying
- domestic chores
- regular sports activity
- regular leisure activity

When providing a medical evaluation of an injury, it is not common to have reliable information about what the person’s occupation or usual activities are and the fact that they have verbally told us cannot be considered reliable.

Hence it might be awkward and lead to a serious error if the medical forensic expert were to make a categorical statement about the existence of a degree of permanent disability without having reliable information about the injured person’s occupation or normal activities.

A cautious way of describing a degree of permanent disability might be as follows:

“The injured person states that their occupation or normal activity is.... If so, existing sequelae are compatible with the medico-legal concept of Permanent Disability.... [degree] as described in Table IV of the scale. This assessment is only valid for the purposes of Act 34/2003.”

It will be up to the injured person to prove to the court that they actually have the usual occupation they have reported to us so that the court can then award the appropriate compensation.

Below, and in the same order as in the scale, are a number of indicative criteria on the impact that sequelae may have on permanent disability.

4. Central neurological syndromes

4.1 Aphasia:

It has to be considered APD due to the sensory and combined way in which the comprehension is altered. In motor aphasia comprehension is normal but speech is not fluent, repetition, reading and writing are altered and naming is complicated, so it is most likely that it will also be assessed as APD.

4.2 Epilepsies

In general those forms that respond well to treatment do not cause permanent disability. In cases where despite treatment the person has sporadic seizures, it will be rated as TPD for working at height, drivers and all occupations that involve a risk to the person and others. In the event of poor response to treatment presenting frequent seizures in the course of a year, we can then consider TPD/APD based on the degree of severity of the disease.

4.3 Impairment of integrated higher brain functions accredited by specific tests (Glasgow Scale Outcome)

- Mild: there will always be PPD and in some cases TPD
- Moderate: as it requires supervision of activities of daily living (TPD or APD)
- Severe: it requires continuous supervision with limitation to the home or a centre (APD)
- Very severe: due to absolute dependence on another person (Severe Disablement)

4.4 Extrapyramidal syndromes

Given the rigidity, dystonia and tremor it can be considered TPD/APD based on its severity. In most cases the determination of permanent disability will depend on serious brain injuries.

4.6 Persistent vegetative state

It will always be Severe Disablement.

4.6 Cerebellar syndrome

If it is unilateral, the intentional tremor, ataxia (hypermetria, asynergy, dyschronometria, dysdiadochokinesia) with involvement of balance and gait and hypotonia will give rise to APD. If it is bilateral, Severe Disablement.

4.7 Dysarthria

Imperfect speech articulation due to the impact on the motor processes involved in it entails difficulty in language expression and therefore may be TPD for occupations that require correct verbal communication, as in the case of teachers, lecturers, presenters, etc., and in all other occupations PPD in moderate and severe cases. It is usually accompanied by other serious neurological damage which will determine the type of permanent disability.

4.8 Ataxia

It is a motor incoordination with gait disorder which can be very disabling and appears associated with severe TBI due to cerebellar injury or diffuse motor and axonal injury which will determine the type of permanent disability.

4.9 Apraxia

It involves an impossibility to perform a previously learned motor activity with involvement of instrumental and handling activities of daily living. This may mean APD since while motor, sensory and comprehension systems are complete, there are problems in limb coordination, sensory-guided movements, kinetics and hand-eye tasks in extrapersonal space.

5. Psychiatric syndromes

5.1 Post-concussion syndrome

In principle it is not considered to be permanently disabling as it requires treatment and can improve. However, in some cases it can be considered that there is a risk either to the person or others in relation to certain activities and then it should be assessed as TPD.

5.2 Organic personality disorder

It will be assessed according to the severity of the clinical picture.

- Mild: there will always be PPD and in some cases TPD
- Moderate: as it requires supervision of activities of daily living (TPD or APD)
- Severe: it requires continuous supervision with limitation to the home or a centre (APD)
- Very severe: due to absolute dependence on another person (Severe Disablement)

5.3 Reactive depressive disorder

It may be grounds for TD but never for permanent disability.

5.4 Posttraumatic stress disorder

It may be grounds for TD but never for permanent disability.

5.5 Anxiety disorder, panic disorder and generalised anxiety disorder

They may be grounds for TD but never for permanent disability.

5.6 Agoraphobia and specific phobias

In severe, properly documented cases that are persistent over time it may be grounds for permanent disability.

5.7 Somatoform disorders

Somatisation disorder, undifferentiated somatoform disorder, conversion disorder, pain disorder, hypochondria, body dysmorphic disorder and unspecified somatoform disorder: they are not grounds for permanent disability.

6. Eye system

Visual acuity (VA) allows us to objectively determine the functional impact of eye disease on the usual occupations of these patients. The recommendations for use of Table VI tell us that loss of visual acuity is

to be assessed in all cases without optical correction. When using Table IV loss of visual acuity is assessed with the optical correction required for better vision, except in the case of professions where regulations state that they cannot be carried out with optical correction. Bearing in mind the Institute of Legal Medicine of Catalonia protocol for the scale in Act 34/2003 “Eye System” (Dr. González García), we can distinguish between several visual acuity loss groups and establish visual capability or functional impact.

- Visual acuity from 10/10 to 8/10: it is considered that there is no significant loss and it is not assessed. It is not assessed in cases where the loss is 20% in each eye either since visual capacity is 100%.
- Visual acuity from 7/10 to 5/10: in practice there is 90% capacity vision. PPD has to be assessed for occupations that require 100% VA: precision eye work (watchmakers, jewellers, etc.).
- Visual acuity from 4/10 to 2/10: there is a decrease in vision. Vision of 2/10 in both eyes entails a loss of 40% of visual capacity. TPD has to be assessed for precision eye work occupations and occupations with risk for the person and others, and PPD for everyone else (non-precision work, students, homemakers, the elderly, etc.).
- Visual acuity of 1/10 or less is moving towards blindness with overall losses of over 65% in visual capacity. VA of 1/10 means it is possible with difficulty to move around, walk down the street and carry out the most basic activities and hence is to be assessed as APD. Instances of VA less than 1/10 are to be assessed as SEVERE DISABLEMENT in all cases.

It should be borne in mind that there are a number of occupations (taxi drivers, ambulance drivers, driving school instructor, law enforcement agency drivers, professional bus and truck drivers) which require VA of at least 0.8 and 0.5 in the better and worse eye respectively with or without optical correction and where monocular vision is not allowed.

7. Hearing system

7.1 Tinnitus

Depending on its intensity and frequency it can be incapacitating (IMLC Protocol Act 34/2003, Dr. Vilella and Dr. Martínez). However, it cannot be objectified clinically and any permanent disability will be determined by the underlying pathology.

7.2 Vertigo

Sporadic cases will be compatible with TPD for occupations involving risk for the person and others, such as working at height, drivers, dangerous machinery drivers, etc. Persistent cases and ones with an objectified underlying organic cause would result in TPD for all regular jobs and occupations

7.3 Hearing acuity deficit

100% of total hearing loss and hearing loss that does not allow a level of conversation should be assessed as APD, except for people who do jobs which do not require interaction with their surroundings and are carried out entirely on their own. When there is hearing loss but a level of conversation can be maintained, this can be considered TPD for occupations that require an optimal level of hearing and PPD for everyone else.

8. Respiratory system

Functional sequelae are determined by the effect on the ventilatory capacity of the lungs. This capacity may have been affected by rib injuries that prevent or hinder respiratory excursion, total or partial lung resection, permanent impairment to the lung parenchyma by gases or other elements, and also ventilatory involvement caused by phrenic nerve palsy.

Spirometry is the “king” of tests for assessment, although gaseous diffusion tests are also valuable. It should be remembered that:

- It is important for the assessment of spirometry that it has been carried out under optimal conditions and the patient has fully understood the instructions.
- It is hard to assess a single spirometry test (where you cannot see a trend in the spirometry data depending on treatment response or the evolution of the disease process).
- On other occasions they may be spirometry tests carried out for an acute respiratory process (infection) in which the figures have been severely altered but return to normal once the process has concluded.
- Initially assess FVC and FEV1 and take the lowest value as the reference.
- Figures between 100% and 75% can be considered as normal (not PD).

- Figures between 75% and 60% might determine PPD or TPD.
- The involvement is important between 60% and 40% (TPD, APD).
- Below 40% (APD, and even SD in cases of minimum exertion dyspnoea requiring continued oxygen).

Spirometry tests performed solely for the purpose of being presented in court proceedings for obtaining disability or other proceedings where they might be significant are of little value, with the exception of those specifically requested to clarify disparate spirometry tests after the healthcare professional who carries them out has been informed of their purpose.

Another factor to be taken into account when evaluating spirometric results is whether the figures might improve if bronchodilator treatment is carried out. If it is used the spirometry test following bronchodilator treatment will have to be assessed as this is the patient's *normal* condition; it would be like assessing a patient with correctable or improvable visual impairment with optical correction.

8.1 Medical forensic assessment

As a guideline it can be considered that:

- a) A spirometry test with FEV1 and FVC figures between 75 and 100% are normal and therefore do not lead to any functional limitations.
- b) Figures between 60 and 75% would indicate a mild ventilatory condition which would not have a significant functional impact in terms of assessment either, although it would suggest that a morbid process is being produced which coupled with another restrictive disease in the patient might incapacitate them for doing jobs involving large physical exertion (greater limitation the lower the figure). PPD or TPD for jobs that require great exertion.
- c) Spirometry results between 40 and 60% clearly restrict the ability to make physical efforts (60% would restrict the basic tasks of a labourer but not a supermarket sales assistant, while figures at the lower limit would restrict the latter in duties such as carrying weights) but allow light or sedentary work. TPD for occupations involving significant physical exertion such as a bricklayer.
- d) Figures below 40% typically bring about dyspnoea with small or minimal exertion incompatible with working and in very serious cases will restrict them in activities of daily living (figures 25%).

Another aspect to consider in relation to spirometric figures when assessing the work ability of a patient with respiratory disease are the environmental conditions of the workplace, whether it is outdoors, working in cold storage rooms or high humidity conditions, environmental pollution due to gases or others of similar intensity, which can

cause greater functional impairment of the patient in such places than if environmental conditions are appropriate, as would be the case with a room in a health centre, clinic or hospital. This factor would not influence instances of normal spirometry or ones which are slightly deficient, but would impact moderate conditions and obviously the most serious ones. APD.

9. Cardiac system

9.1 Heart failure

The scale includes four levels of functional impairment which match the NYHA classification. They are:

- Class I: dyspnoea with major exertion (ejection fraction between 60 and 50%)
- Class II: dyspnoea with moderate exertion (ejection fraction between 50 and 40%)
- Class II: dyspnoea with minor exertion (ejection fraction between 40 and 30%)
- Class IV: dyspnoea when at rest (ejection fraction < 30%)

The most appropriate functional test to assess this impact is an echocardiogram which gives the ejection fraction figure (normal EF 55%). The ejection fraction and clinical signs can be used as key factors to assess the physical capacity of a patient. Thus:

- a) Ejection fraction between 60 and 50% with dyspnoea with major exertion and minimal functional impairment: it would not be incapacitating or only for those activities that require intense physical exertion (PPD or TPD).
- b) Ejection fraction between 50 and 40% with dyspnoea with moderate exertion would be incapacitating for activities requiring medium or high intensity physical exertion (PPD or TPD).
- c) Ejection fraction between 40 and 30% with dyspnoea with minor exertion would be incapacitating for all activities that are not strictly sedentary (TPD).
- d) Ejection fraction below 30% with dyspnoea when at rest or minimum exertion would be highly incapacitating from the work standpoint (APD).

9.2 Valve prostheses

From the functional point of view we could establish a relationship similar to the one above. However, in this case whether valve replace-

ment will lead to improvement needs to be considered, and this is sometimes postponed until allowed by the clinical situation.

In these cases the usual compulsory use of anticoagulant therapy should be borne in mind when assessing occupational activities with high risk of bleeding, for example, cuts.

9.3 Sequelae after cardiac trauma

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Functional capacity will be determined by the clinical signs, frequency and severity of episodes, if evolution is in the form of crisis and even the treatment (use of anticoagulants with risk of bleeding).

10. Peripheral vascular disease

10.1 Operated traumatic aneurysm

Highly variable depending on the functional sequelae.

10.2 Posttraumatic venous disorders

Phlebitis or venous trauma in patients with previous venous disease:

- Mild (appreciation of varicose veins and pigmentation). No PD or PPD
- Moderate (oedema, eczema, pain and indurated cellulitis). PPD or TPD (prolonged standing activities)
- Severe (severe ulcers and trophic disorders). TPD and even APD depending on the severity and persistence of the ulcers.

10.3 Posttraumatic arterial disorders

- Intermittent claudication and coldness (based on functional impact). PPD or TPD depending on claudication distance.
- Intermittent claudication, coldness and trophic disorders (based on functional impact). PPD or TPD depending on claudication distance.

10.4 Posttraumatic arteriovenous fistula

- No regional or general impact. No PD.
- With regional impact (oedema, varicose veins, etc.). As in the vein disease section.

- With general impact (assess by heart failure). As in the heart disease section.

10.5 Lymphoedema

No PD, PPD or TPD, depending on the degree of involvement, the affected extremity and whether bilateral or not.

10.6 Substitute material and/or prosthesis

No PD or PPD (depending on whether it causes some type of functional impairment).

11. Upper limb sequelae

Anatomical and/or functional upper limb sequelae can often involve a degree of permanent disability. Generally it would be partial disability (PPD), less often total disability (TPD), and very rarely would be absolute disability (APD) as there are many jobs which even with amputations can be done with severe sequelae at this level; the typical example of a job that could be done with important sequelae at this level would be a concierge.

Although it is not possible to correlate all upper limb sequelae and all occupations or activities, below are a number of general guidelines.

11.1 Shoulder

It is important to determine whether the sequelae affect the dominant limb and correlate them with the occupation or normal activity.

Shoulder limitations usually have a significant impact on the occupation or normal activity when they mean the person is unable to reach the horizontal (90°) since most jobs do not require more. However, even with sedentary occupations such as a clerk they should be assessed as PPD given that no matter how small the limitation they always “partially restrict”. In other occupations such as installer, furniture assembler, painter or plasterer, a limitation of approximately 25% would mean TPD.

Sequelae of this joint will never entail APD even in the case of amputation of an arm.

Painful sequelae of the shoulder may have an impact similar to functional limitations, especially for activities that require repetitive movements with the joint.

11.2 Elbow

It is important to determine whether the sequelae affect the dominant limb and correlate them with the occupation or normal activity.

Anchylosis and elbow limitations on the dominant limb almost always lead to PPD except for minimal limitations. When the limitation is important they will entail TPD for occupations or activities such as installer, assembler, painter or mechanic.

They will never lead to APD.

Painful sequelae of the elbow may have an impact similar to functional limitations, especially for activities that require repetitive movements with the joint.

Sequelae of this joint will never entail APD even in the case of unilateral amputation.

11.3 Wrist and hand

In this case it is not important to determine whether the sequelae affect the dominant limb since virtually all occupations or normal activities involve both hands.

Anchylosis and wrist or hand limitations almost always lead to PPD. When the limitation is important they will entail TPD for occupations or activities where good handling skills are essential, such as installer, plumber, carpenter, mechanic or watchmaker.

They will never lead to APD even in the case of unilateral amputation.

Painful sequelae of the wrist and hand may have an impact similar to functional limitations, especially for activities that require repetitive movements with the joint.

11.4 Fingers

Amputation of a finger will have a very different impact depending on whether it is the index or another one, since the index finger is essential for a pincer movement. This movement is essential in many occupations such as seamstress, plumber, installer, etc., and if it cannot be made then TPD should be reported.

PPD will almost always be indicated for the rest of individual amputations or limitations.

Sequelae of the fingers rarely lead to APD except in cases where functionality is lost in both hands, such as loss of both thumbs or amputation of multiple fingers on both hands.

12. Lower limb sequelae

Just as with the upper limbs, it is also common that lower limb sequelae can lead to permanent disability.

Here, and in addition to the anatomical and/or functional impact of the sequela on the occupation or normal activity, it also has to be borne in mind that there are many occupational, sports and leisure activities which require adequate ability to stand or walk. Furthermore, commuting or travelling to the activity site is an essential part of it and therefore any difficulty in making this trip would be PPD.

12.1 Limping in general

The existence of lameness will always entail permanent disability which will be at least partial (PPD). For ordinary occupations requiring prolonged walking, such as postman or woman or sports or leisure activities conducted on a regular basis, limping will be TPD.

Limping will never lead to APD.

12.2 Limb difference

If less than 3 cm and can be offset orthopedically, it is unlikely to lead to PPD.

If more than 3 cm and even though it can be offset with special footwear it leads to difficulty in walking and therefore will always be at least PPD. If the usual occupation requires prolonged walking such as postman or woman or sports or leisure activities conducted on a regular basis, significant limb difference will be TPD.

Limb difference will never lead to APD.

12.3. Hip, knee, ankle and foot

Anatomical and/or functional sequelae at these levels will never lead to APD even in the case of amputation of a leg.

Amputation or significant functional limitation does produce TPD for occupations and/or activities which require prolonged standing or walking. Mild functional limitations will always lead to PPD.

Hip or knee replacements have to be assessed by outcomes (pain, limitations) as do anatomical and functional constraints. However, even with a good prosthesis result they will find it hard to perform jobs where constant walking is required.

Painful sequelae of these joints have an effect on walking similar to functional limitations.

12.4 Toes

Anatomical and/or functional sequelae at these levels will never lead to APD even in the case of unilateral amputation.

The importance in walking of the big toe has to be borne in mind as it is crucial for stability and momentum. Hence complete or partial big toe amputations will always lead to at least PPD, and if the person's occupation or habitual activity requires prolonged walking, such as postman or woman, it will mean TPD.

Total amputation of the other toes affecting 1 or 2 does not necessarily have a functional impact on walking just like partial amputations. However, multiple total or partial amputations of toes may have an impact on walking and should therefore be assessed as at least PPD or TPD if the person's occupation or habitual activity requires prolonged walking.

13. Spinal cord

Table IV shows that Severe Disablement is recognised for people suffering from permanent sequelae that require the assistance of others to perform the essential activities of daily living such as dressing, moving, eating or similar. This is the case of quadriplegia, paraplegia, chronic coma vigil or vegetative states, major neurological or neuropsychiatric sequelae, blindness, etc.

Severe disablement is also possible in severe quadriparesis, severe paraparesis, severe transverse spinal cord syndrome and severe spinal cord hemisection syndrome (Brown-Séquard syndrome). In case of doubt, the most advisable option due to its simplicity is to use the "Scale to determine the need for assistance from another person" in Annex 2 of Royal Decree 1971/1999, of 23 December, at the end of this chapter.

Mild forms of the above pathologies can be assessed as PPD or TPD as appropriate and in moderate cases as TPD/APD as appropriate.

14. Cranial nerves

14.1 III - Oculomotor nerve

Complete palsy (diplopia, paralytic mydriasis forcing occlusion, ptosis) functionally entails sight in terms of the visual acuity of the

healthy eye. In other words, the parameters set out in the section on visual acuity are to be used. *In paresis* clinical involvement is partial: it is to be assessed based on diplopia and ptosis, and in particular by the high positions of gaze by the involved eye, and the healthy eye will be used as a reference. However, diplopia is highly disabling and most likely by itself can require TPD. Diplopia will be considered a sequela at a year/year and half after injury.

14.2. IV - Internal oculomotor or pathetic nerve

Complete palsy (lower field diplopia) makes it impossible to look downwards, especially when looking at the inner side, so the injured person cannot see their contralateral shoulder and has difficulty tying shoes, going down stairs and reading. The chances are that if the good eye has optimal VA it can compensate for the functional impairment caused by palsy. If there is also visual impairment in the good eye, there will then be a limitation for all tasks requiring optical precision in driving and posing risk for the person and for others. *Paresis* involves difficulty in looking downwards, especially when looking at the inner side and the injured person has difficulty in seeing their contralateral shoulder and tying shoes, going down stairs and reading. However, this difficulty can be compensated globally by the unaffected other eye. However, diplopia is highly disabling and most likely by itself can require TPD. Diplopia will be considered a sequela at a year/year and half after injury.

14.3 V - Trigeminal nerve

Intermittent pain cannot be a permanent disability. *Continuous pain* without response to treatment is grounds for APD.

- Suborbital palsy: ophthalmic branch hypo/anaesthesia does not entail permanent disability.
- Inferior palsy: maxillary branch hypo/anaesthesia involves hypoesthesia, anaesthesia and paresthesia of the maxilla and maxilla palsy and in principle does not entail permanent disability.
- Lingual palsy: dentomandibular branch hypo/anaesthesia involves mandibular hypoesthesia, anaesthesia and paresthesia and paralysis of the chewing muscles. PPD can be assessed in anaesthesia, assessing difficulty in chewing food as a physiological activity which generates everyday pleasure domestically. Speech impairment may be considered TPD for tasks requiring this function to be complete.

14.4 VI - External oculomotor nerve

There is an inability to abduct the eye on the involved side beyond the midline. Functionally the injured person can correct this by turning their head to the side of the injury so that the involved eye remains fixed on the object of interest and the normal eye moves. However, in cases where there is diplopia this is very disabling and most likely by itself may be grounds for TPD. Diplopia will be considered a sequela at a year/year and half after injury.

14.5 VII - Facial nerve

Palsy may be grounds for TPD in occupations where facial appearance is essential (asymmetry and hemiface muscle atrophy, not able to purse lips, raise eyebrows, show teeth, deflect the labial commissure to the healthy side, tearing) and also for wine tasters, cooks, food tasters, etc. since the involvement of the sensory part of the nerve entails involvement of taste of the anterior two thirds of the tongue and the hard and soft palates. Moreover, in all cases of palsy PPD for regular activities should always be assessed as chewing and swallowing are involved, describing a metallic taste dysgeusia. This point is related to the fact that food can be considered as a physiological activity which brings pleasure.

In the case of *paresis* PPD may be assessed based on the severity of the paresis and the type of occupation.

14.6 IX - Glossopharyngeal nerve

Generally permanent disability is determined by the severe head trauma which injured this nerve and the palsy/paresis of the palate, changes in the voice and swallowing and taste alterations are secondary.

14.7 X - Pneumogastric or vagus nerve

The type of permanent disability is determined by the severity of the lesions originating the involvement of this nerve.

14.8 XI - Spinal accessory

Functionally there is difficulty in turning the head sideways toward the affected side and impossibility of the abduction of the upper limb. However, its involvement in isolation is very rare since the production

mechanism is direct cervical trauma with stabbing or sharp objects or traumatic manoeuvres with stretching of the nerve and where permanent disability will also be determined by the original injuries.

14.8 XII - Hypoglossal

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Involved in chewing, swallowing and articulation of words (most vowels and some consonants), resulting in PPD in activities of daily living and TPD for those occupations that require integrity of language (teachers, vendors, announcers, etc.). In other cases the type of permanent disability is determined by the severity of the occipital trauma.

15. Peripheral nervous system

The *functional impact* section of Chapter 7 of the IMLC Protocol for Act 34/2003 (Dr. Cabús, Dr. Fuertes, Dr. García García, Dr. Martí Amengual, Dr. Pérez Rico, Dr. Salort and Dr. Soler Murall) contains the functional deficit presented by the patient according to the affected nerve. In the case of involvement of the upper limb peripheral nervous system we have to assess whether the patient is right- or left-handed, what kind of movement is affected and whether this movement is necessary for their regular occupation. We also need to assess whether both upper extremities are necessary or not for the person's usual occupations. The same applies in the case of involvement of the lower limb peripheral nervous system, although in this case we can give a more global assessment of the impact on gait, prolonged standing, prolonged walking, going up and down stairs and whether the person can run or not.

If there are any doubts or queries and by analogy the reader is advised to see the section on upper and lower limbs.

16. Endocrine disorders

16.1 Anterior hypothalamic-pituitary hypofunction (TSH and ACTH deficiency):

Secondary adrenal insufficiency caused by ACTH deficiency usually responds well to glucocorticoid replacement therapy which means it would not cause permanent disability.

16.2 Neurohypophysis injuries (diabetes insipidus)

The signs and symptoms of uncomplicated central diabetes insipidus can be eliminated completely by treatment with DDAVP (desamino-8-D-arginine-vasopressin), a synthetic analogue of vasopressin, which means it would not cause permanent disability.

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17. Skin

Limitations that may be caused by skin lesions are:

- Disfigurement
- Functional
- Combined

The area involved is important from the aesthetic point of view so face and hands would lead to a greater degree of involvement.

Disfigurement sequelae can hinder work activities which include not only those which are primarily dependent on appearance (models) but also others where physical appearance is important due to their social relationship (serving the public, salespeople, teachers, etc.). TPD

In some cases in which involvement is particularly serious it may result in work disability. Recreational and routine activities should not be jeopardised except in very serious cases that could generate social rejection. APD

Functional impact is determined by scars (retractile, keloids, etc.) which could hinder mobility or cause pain (adhesions, neuromas, etc.). Thus an armpit scar that limits arm abduction would affect work such as spinning in the textile industry or leisure activities such as tennis (TPD).

It is of special interest when involvement occurs in hands.

Mixed effects would be determined by the concurrence of aesthetic and functional disorders (e.g., pathological gait, dynamic and functional aesthetic alteration of gait) and here the field of activity involved and/or degree of disability can be expanded.



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