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ESSOMM European core curriculum and principles of manual medicine

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Table 1 Elements of the professional training module in manual medicine

1. Basic course
10 ECTS (postgraduate advanced education) = 75 teaching periods of 45–50 min + 10 min intermission and 25 h of self-study, in which the basic knowledge and the basic skills of manual medicine are taught
2. Advanced course
20 ECTS (postgraduate advanced education) = 150 teaching periods of 45–50 min + 10 min intermission and 50 h of self-study, in which the advanced competencies and skills of manual medicine are taught
In sum
30 ECTS (postgraduate advanced education) = 225 teaching periods of 45–50 min + 10 min intermission and 75 h of self-study equivalent to 300 h of training
ECTS European Credit Transfer System

Part I: Curriculum of manual medicine

Introduction

Subject of manual medicine

Manual medicine is a medical discipline whose diagnostic and therapeutic procedures are based on contemporary scientific neurophysiological and biomechanical principles. By using enhanced manual skills, it firstly provides diagnostic examination of the locomotor system, head and connective structures and secondly, by using manual techniques, treats reversible functional disorders with the intention of curing, preventing and rehabilitating these conditions.

Manual medicine in Europe is an interdisciplinary additional competence for European medical specialists. Within the framework of a multimodal therapeutic concept, manual medicine encompasses the interdisciplinary application of diagnostic and therapeutic techniques for the management of reversible dysfunctions of the locomotor system and its associated reflexively interlinked conditions (vertebrovisceral, viscerovertebral and viscerocutaneous), whilst additionally considering psychosomatic influences.

Prerequisites for learning and practicing manual medicine

Preconditions for acquiring the post-specialist qualification "Manual Medicine" are a licence to practise medicine (physician, medical doctor) and being at least on the path to becoming a European specialist. The primary goal of this training is the acquisition of additional competence and skills in manual medicine by way of completing the professional training module.

The professional training courses for manual medicine should be designed to provide doctors in private practice or hospitals who are concerned with the diagnosis and treatment of reversible dysfunctions of the locomotor system with the best tools to enhance their manual diagnostic and therapeutic skills.

Manual medicine and its relationship to osteopathy and chiropractic

The European Scientific Society of Manual Medicine (ESSOMM) considers recognised osteopathic and chiropractic techniques as elements that contribute to manual medicine. Manual medicine, as used in Europe since the second half of the 20th century, is based on ancient roots as well as on osteopathy and chiropractic.

The aims of a scientific manual physician, an osteopathic physician and a non-medical chiropractor are identical. All three tend to use their hands to diminish pain, to improve function and to ultimately contribute to healing the patient's body.

The origins of manual medicine can be traced back to over 5000 years ago, while osteopaths and chiropractors contributed to the important anatomical and systemic understanding of functional disorders of the body in the 19th and 20th centuries. The founder of osteopathy was A.T. Still (1874) and of chiropractic treatment D.D. Palmer (1897), both practising in the USA.

In the course of time, various models and techniques were included, and different "schools" developed outside the US, e.g., in France, Great Britain, Belgium, Netherlands, Switzerland, Austria, Germany, Scandinavia, Czech Republic or Australia. Osteopathic or chiropractic techniques can be applied effectively by physicians and are based on theoretical models derived from anatomy, biomechanics and neurophysiology. Many of these empirically founded and safe techniques have been widely incorporated into the European core curriculum of manual medicine.

Principle structure of the professional postgraduate apprenticeship in manual medicine

The practice of manual medicine requires theoretical knowledge, competencies and enhanced manual skills, which are taught in structured courses by specially qualified teachers. A confirmation of the recognition/acceptance of the course as well as its teacher is to be obtained from the responsible national authority of physicians prior to taking the course. The course sequence should be obligatory.

Following the Bologna process in Europe, this higher medical training in manual medicine requires a total volume of 30 ECTS (European Credit Transfer System; applied to postgraduate advanced education). The professional training module should be therefore divided into the elements outlined in **Table 1**.

Implementation of the courses

The professional training facilities for this course must provide appropriate rooms for the theoretical excurse as well as exercise rooms with height-adjustable treat-

Table 2 Breakdown of the course structure
1. Basic course 10 ECTS or 100 h of the basic course should be organised in: – 30 h theory – 70 h practical experience
2. Advanced course 20 ECTS or 200 h of the advanced course are organised in: – 40 h theory – 160 h practical experience
ECTS European Credit Transfer System

Table 4 Practical experience	
Examination in manual medicine – of the peripheral joints – scanning examination of the spine – of the articular connections of the head – of the muscles of the extremities, the torso, the spine and the head – of the connective tissue	30 h
Evaluation of the results of examination	10 h
Basic mobilising, soft tissue and neuromuscular techniques in manual medicine for the treatment of dysfunctions of the joints, muscles and other tissues – of the spine – of the head – of the extremities – of the connective tissue	30 h

Table 3 Acquisition of basic knowledge and basic skills	
Theoretical principles of – functionality, neuronal control and functional pathology of the locomotor system – vertebrovisceral interactions – nociception, pain and nocireaction – biomechanical principles of the locomotor system as well as of dysfunctions of the locomotor system – general effects of the different manual medicine techniques, including vertebrovisceral and viscerovertebral interactions and functional chain reactions	7 h
Functional anatomy of the peripheral joints, the spine and the joints of the head	5 h
Structure of fascia, physiological and neurophysiological features of the connective tissue	5 h
Fundamental knowledge of imaging diagnostics and laboratory findings with respect to manual medicine	5 h
Pain in the locomotor system	2 h
Psyche and the locomotor system	1 h
Phenomenology of muscle tension and its significance in manual medicine	1 h
Specific manual medicine history taking	1 h
Clinical signs that can be influenced by manual medicine	1 h
Indications and contraindications for manual medicine treatment	1 h
Guidelines for documentation and patient information	1 h

Table 5 Acquisition of specific competencies and skills—theory	
Differential diagnosis of:	20 h
– dysfunctions and diseases (locomotor system/internal disease)	4 h
– radicular and pseudoradicular pain syndromes	4 h
– lumbar and gluteal pain	4 h
– cervicocranial and cervicobrachial pain, headache included	4 h
– balance dysfunctions and vertigo	4 h
Interpretation of medical imaging, especially functional radiology	4 h
Functional control of the locomotor system: motor patterns, their composition and plasticity	6 h
Interlinked dysfunctions (chain-reactions) in the locomotor system	10 h

ment tables. A maximum of three students should be planned per treatment table (European Union of Medical Specialists, UEMS; documents 2015).

The instruction consists of:

- theoretical lectures
- practical demonstrations
- exercise sessions

Following the introduction of the session which includes theory, treatment indications and contraindications, there is revision of the practical instructions of the previously taught manual examination and treatment techniques. Before the students themselves practice these techniques, they are firstly demonstrated by the course director or teacher, who will then supervise them during the exercises.

No more than 15 course participants per teacher should be placed in a course, and, as a matter of principle, each course should be evaluated by its participants. The course director and the teacher must have advanced experience in manual medicine practices. They are obliged to regularly participate in especially designated continuing education courses for teachers and recommendations for the continuing medical education of physicians by the local or national authorities must be respected.

Structure of the courses

Both the basic and the advanced courses are administered in blocks. The blocks' contents and order are determined by the institution/society/association offering the training. The length of the individual blocks may be between 20 and 60 teaching periods. For didactic reasons, no more than 10 teaching units (of 45–50 min each) should be conducted per day.

The emphasis is on the teaching of practical competencies, skills and knowledge. The theoretical course units can be integrated into the practical instruction. The individual blocks should be scheduled at least 3 months apart so that the time between the blocks can be used to practice, perform self-studies, and solidify the learned competencies and skills (**Table 2**).

This professional training course is completed with a final examination by the providing medical association, certified by the national authority.

Content of the courses

The term "hour" describes a course unit of 45–50 min.

Basic course

10 ECTS, 100 h.

Table 6 Acquisition of specific competencies and skills—practical experience

Segmentally specific manipulation techniques of the spine and the joints of the extremities	45 h
Enhancement of mobilisation techniques in such as specific techniques for muscle inhibition or muscle relaxation (muscle energy techniques, techniques based on post-isometric relaxation and on reciprocal inhibition and positioning)	50 h
Fundamentals of myofascial techniques	30 h
Treatment strategies for interlinked functional (chain reaction) syndromes	10 h
Differential diagnosis and treatment of dysfunctions of motor pattern at different control levels	10 h
Indications for physiotherapy and training for rehabilitation	5 h
Integration of the manual medical treatment into a multimodal treatment concept	10 h

Table 8 Essential knowledge in anatomy, biomechanics, physiology and pathophysiology

Functional anatomy and biomechanics of the locomotor system	K	3	bc ac
Physiology and pathophysiology of the locomotor system	K	2	bc ac
Functional analysis of the locomotor system	K	3	bc
Principles of manual medicine and postulated mechanisms of action	K	3	bc
Anatomy, physiology and pathophysiology of the nervous system in relation to pain and dysfunction	K	2	bc
Function and interlinked function (chain reactions) as well as the dysfunction within and between the organs of the locomotor system (spine, extremity joints, muscles, ligaments, fascia)	K	2	ac
Primary and secondary somatic dysfunctions, simple and complex dysfunctions in the locomotor system	K	3	ac
Specific postulated mechanisms of diagnostic and therapeutic techniques	K	3	bc
Clinical syndromes and differential diagnostics of the locomotor system	K	2	bc
Relevant ancillary diagnostics (e.g., laboratory, imaging, electro-diagnostics) to manual medicine	K	2	bc
Risks and benefits of other relevant therapeutic modalities compared to or in conjunction with manual medicine	K	3	ac
Indications and contraindications for different therapeutic options	K	3	ac

See **Table 7** for level numbers
K knowledge, *bc* basic course, *ac* advanced course

Table 9 Essential skills related to anatomy, biomechanics, physiology and pathophysiology

Exchanging relevant information specific to the individual patient's condition, within a meaningful dialogue, in order to obtain informed consent	A	2	bc
Effectively inform the patient about anticipated benefits and outcomes, potential risks and complications of manual medicine treatments	A	2	bc
To conduct effective history taking	A	2	bc
To conduct physical examination	S	3	bc
To perform effective, accurate palpatory diagnostics	S	3	bc
Competence to deliver safe, effective manual medicine treatment in a general population	S	2	bc ac
Competence to deliver safe, effective manual medicine treatment in complex morbidity or special musculoskeletal complaints	S	2	ac

See **Table 7** for level numbers
A attitude, *S* skills, *bc* basic course, *ac* advanced course

Table 7 Main focus of the courses

Levels of competence in Knowledge (cognition):	*K*
Basic knowledge	1
Reproducible knowledge	2
Applied knowledge in relation to manual medicine	3
Active teaching manual medicine knowledge	4
Levels of competence in Skills:	*S*
Functional tests, palpation	1
Applying manual medicine techniques under supervision	2
Applying manual medicine techniques without supervision	3
Active teaching manual medicine skill	4
Levels of competence in Attitude:	*A*
History taking	1
Inform about therapeutic options/ contraindications	2
Patient education	3

K knowledge, *S* skills, *A* attitude

Acquisition of basic knowledge and basic skills. 30 h (**Table 3**).

Practical experience. 70 h (**Table 4**).

Advanced course

20 ECTS, 200 h.

Acquisition of specific competencies and skills. *Theory*.

40 h (**Table 5**).

Practical experience.

160 h (**Table 6**).

Main focus of the courses

Table 7.

Basic knowledge

Knowledge represents an ongoing process, requiring constant effort, vigilance and updating. Anatomy, biomechanics, physiology and pathophysiology, as basics in manual medicine, are informed by current developments in science which are gathered, analysed and shared, when appropriate, by the ESSOMM.

Essential knowledge. **Table 8**.

Essential skills. **Table 9**.

Table 10 General anatomy objectives			
To comprehend and to describe the normal functions of the musculoskeletal and the nervous system	K	2	bc
To understand the anatomical basis of techniques used to investigate and manage disorders of the locomotor system	K	3	ac
See ◘ **Table 7** for level numbers *K* knowledge, *bc* basic course, *ac* advanced course			

Table 11 Specific anatomy objectives			
To describe macrostructure, anatomical relations and surface anatomy of the elements of the locomotor system	K	2	bc
To describe the course and relation of the peripheral arteries (especially the vertebral arteries) and the effects on these vessels of movements of the associated skeletal structures	K	1	bc
To describe and demonstrate the course and distribution of the peripheral and autonomic nerves	K	2	bc
To explain the motor and sensory mechanisms involved in movements and musculoskeletal complaints	K	2	bc
To recognise anatomical variants in neural and musculoskeletal structures	K	1	bc
See ◘ **Table 7** for level numbers *K* knowledge, *bc* basic course, *ac* advanced course			

Table 12 General physiology objectives			
To understand the physiological basis of the functions and disorders of the locomotor system	K	1	bc
See ◘ **Table 7** for level numbers *K* knowledge, *bc* basic course			

Table 13 Specific physiology objectives			
To describe different types of muscle fibres	K	1	bc
To describe muscle adaptability	K	1	bc
To describe the effects of rest, exercise and ageing on skeletal muscle in terms of histochemistry and molecular structure	K	1	bc
To describe the neurophysiology, activity and function of reflexes involving the locomotor system including somatovisceral, viscerosomatic and somatosomatic relationships	K	1	bc
To describe the basic metabolic principles and physiology of bone, muscle, connective tissue and nerves pertaining to the locomotor system	K	1	bc
To describe the molecular and cellular processes implicated in mechanisms of muscle contraction	K	1	bc
To describe the molecular and cellular processes involved in the generation and propagation of action potentials in nerves, muscles and synapses	K	1	bc
To describe the effects of rest, exercise and ageing on fascia in terms of histochemistry and molecular structure	K	1	bc
To describe the motor and sensory neurophysiological mechanisms to explain the symptoms of disorders of the locomotor system	K	2	bc
See ◘ **Table 7** for level numbers *K* knowledge, *bc* basic course			

Table 14 General biomechanics objectives			
To understand certain concepts of biomechanics and apply them to the locomotor system	K	2	bc
To recognise and describe the aberrations of function of the locomotor system	K	2	bc
See ◘ **Table 7** for level numbers *K* knowledge, *bc* basic course			

Anatomy objectives

General anatomy objectives. ◘ Table 10.

Specific anatomy objectives. ◘ Table 11.

Physiology objectives

General physiology objectives. ◘ Table 12.

Specific physiology objectives. ◘ Table 13.

Biomechanics objectives

General biomechanics objectives. ◘ Table 14.

Specific biomechanics objectives. ◘ Table 15.

Pain objectives

General pain objectives. ◘ Table 16.

Specific pain objectives. ◘ Table 17.

Diagnostic examination

Conventional medical examination. ◘ Table 18.

Examination using manual medicine techniques. ◘ Table 19.

Recording diagnostic findings. ◘ Table 20.

Treatment modalities

General treatment. ◘ Table 21.

Disease prevention and health promotion. ◘ Table 22.

Clinical pictures

Clinical pictures in manual medicine. ◘ Table 23.

Diseases, disorders and conditions. ◘ Table 24.

Certification

ESSOMM accepts national certificates of manual medicine for accreditation of a European diploma. A prerequisite is that their structured curriculum of manual medicine is as described in the previous chapters. For this acceptance the executive board of the ESSOMM is authorised to ensure that

Table 15 Specific biomechanics objectives			
To define, in biomechanical terms, the following terms as they are applied to joints: hypomobility, hypermobility, and instability	K	3	bc
To describe biomechanical differences between somatic dysfunction and capsular patterns	K	3	bc
To demonstrate an ability to apply and interpret the following terms with respect to any of the tissues of the locomotor system: stress, strain, stiffness	K	3	bc
To describe the movement of any joint in terms of translation and rotation about biomechanical axes	K	3	bc
See **Table 7** for level numbers *K* knowledge, *bc* basic course			

Table 16 General pain objectives			
To understand the physiology and pathophysiology of pain with their bio-psychosocial implications	K	2	bc
To understand the somatic and visceral structures which contain receptors capable of reporting noxious stimuli that may elicit pain	K	3	bc
To understand the relationship between pain and function, i.e., pain as consequence and as cause of dysfunction	K	2	bc
See **Table 7** for level numbers *K* knowledge, *bc* basic course			

Table 17 Specific pain objectives			
To describe, at an appropriate level, the classification of pain	K	2	bc
To differentiate acute and chronic pain and their proposed mechanisms	K	2	bc
To describe the anatomy, physiology, pathophysiology and currently understood mechanisms of pain	K	2	bc
To describe the understood patterns of referred pain to and from the locomotor system	K	2	bc
To describe the relationship between psychosocial factors and chronic pain	K	2	bc
To describe the role of the autonomic nervous system in relation to pain	K	2	bc
See **Table 7** for level numbers *K* knowledge, *bc* basic course			

Table 18 Conventional medical examination			
To perform a conventional medical examination in order to understand the condition of the patient with respect to decision-making regarding the indications and contraindications of the therapeutic options	S	3	bc
To perform history taking and examination with emphasis on orthopaedic, neurological, occupational and bio-psychosocial factors	S	3	bc
To perform systemic and ancillary tests, where indicated	S	3	bc
To prioritise diagnostic tests based on sensitivity and specificity	S	3	bc
See **Table 7** for level numbers *S* skills, *bc* basic course			

national curricula conform to this standard and can, when appropriate, refuse the European certification (diploma).

Part II: Principles of manual medicine

Neurophysiological background of dysfunction

Introduction

The results from various fields of basic research (neurophysiology, neuroanatomy, neuropharmacology, myofascial pain research, anaesthesiology and pain management medicine) in terms of translational research have created new evidence in manual medicine worldwide [1, 2]. The following explanations and diagrams on important key pain medical and functional terms achieve "a degree of simplification that is at the limit of tolerance, but basically the statements remain very close to the scientific content and are reliable" (Walter Zieglgänsberger, Max Planck Institute for Neuropharmacology, Munich, Germany).

The author of the present article and numerous experienced manual physicians have internalised this content into daily diagnostic and therapeutic activities and thus achieved an advanced specification of the differential diagnoses and, above all, an improvement in differential detailed therapeutic planning. This also and above all includes finding the indication for manual medical interventions.

The role of nociafferents

The body reacts to nociceptive stimuli via metameric and central interconnections in the sense of nocireactive motor system activation (**Fig. 1**).

Clinically, there is a pain-related motor coordination disorder (e.g., abdominal muscular defence, protective extremity reflex, gait disorder with activated coxarthrosis, malposition with lumbar spine blockage, signs of muscular imbalance).

Sympathetic system activation

Axon collaterals of the posterior horn neurons also excite sympathetic-origin neurons in the thoracic lateral horn and generate vegetative efferents (**Fig. 2**).

Clinically, the following can occur: changes in skin blood flow, piloerection, increased sweat secretion, tachycardia, increase in blood pressure, etc. There are also routes of parasympathetic dysregulation via the vagus nerve and the pelvic splanchnic nerves. An extreme form of sympathetic system activation is the complex regional pain syndrome 1 (CRPS 1, formerly known as Sudeck dystrophy).

Convergence

At the multireceptive dorsal horn neuron (MRHHN), not only afferents from the respective vertebral joints converge, but also afferents from various regions that are each assigned to a segment, i.e., afferents from

Table 19 Examination using manual medicine techniques

To perform examination to identify normal locomotor functions and their disturbance	S	3	bc
To perform manual techniques for the diagnosis of the locomotor system and other tissues involved in the patient's pathology: – joint play examination – examination of muscular tension – evaluation of the connective tissue tension – evaluation of viscerovertebral chain reactions	S	3	ac
To follow a holistic approach in the framework of medical diagnostic methods	S	3	ac
To perform screening examination to identify if there is a problem in the locomotor system that deserves additional evaluation	S	3	bc
To perform a complete manual examination starting from the whole person and then moving regionally with the final focus being local and specialised	S	3	bc
To perform a scanning examination to identify which regions and tissues within the region are dysfunctional and of relevance at a level appropriate to the treatment skills	S	3	ac
To conduct regional palpatory examinations of the tissues of the locomotor system to identify dysfunctions	S	3	bc
To perform manual and functional diagnostics of the locomotor system with special consideration of pain reactive signs	S	3	bc ac
To conduct palpatory examinations of local tissues to determine the specific dysfunctions considered for manual medicine treatment and the characteristics that will be important when considering the indications and contraindications of a specific treatment modality	S	3	ac
To conduct different palpatory examinations in order to look at and record elements of pain provocation, sensory changes, tissue texture changes, examination of range of motion and characteristics of end-feel barrier	S	3	bc ac
To conduct re-evaluation of diagnostic findings	S	3	ac

See **Table 7** for level numbers
S skills, *bc* basic course, *ac* advanced course

Table 21 General treatment

To perform manual techniques for the treatment of the locomotor system and other tissues involved in the patient's pathology such as – positioning techniques – exercises for stabilisation, muscle strain and muscle training	S	3	bc
To perform mobilisation techniques including specific techniques for muscle inhibition or muscle relaxation (techniques based on post-isometric relaxation and on reciprocal inhibition, and positioning techniques)	S	3	bc
To perform segmental manipulation techniques of the spine and manipulation techniques of the peripheral joints	S	3	bc ac
To supervise physiotherapy of the locomotor system and training for rehabilitation	K	2	ac
To perform myofascial and related soft tissue techniques	S	3	ac
To perform trigger-point therapy	S	3	ac
To apply treatment strategies for interlinked functional (chain reaction) syndromes	S	3	ac
To integrate the principles of treatment of manual medicine into multimodal treatment concepts	K	3	ac

See **Table 7** for level numbers
K knowledge, *S* skills, *bc* basic course, *ac* advanced course

Table 20 Recording diagnostic findings

To record the patient evaluation and patient progress by using various methods of measurement, e.g., visual analogue scale (VAS), dolorimeter, impairment scales, general health scales	S	2	bc
To record relevant specific findings in terms of manual medicine	S	3	bc
To maintain quality management	K	2	bc

See **Table 7** for level numbers
bc basic course

skin, muscles, tendons and internal organs (**Fig. 3**).

According to this, nociafferents from nonvertebral structures in the common final path of motor system activation can also lead to vertebral dysfunctions, for which we know numerous clinical examples (lung cancer, thoracic spine blockage, adnexitis, lumbar spine obstruction, prostatitis, SIG [sacroiliac joint] blockage, etc.). Clinical caveat: the first symptom of pancreatic cancer can be a recurrent thoracic spine obstruction.

Peripheral sensitisation

If a nociceptor is permanently stimulated above the threshold by a sustained noxious stimulus (e.g., UV radiation, mechanical overload of a joint or vertebral joint, or compression of the nerve), it changes its biochemical behaviour (**Fig. 4**). It secretes neurokinins (substance P; calcitonin gene-related peptide, CGRP; etc.) into the extracellular space, which in turn trigger the so-called inflammatory cascade.

Phospholipase A2 (PLA2) dissolves arachidonic acid from the membranes, cyclooxygenase 2 (Cox 2) converts arachidonic acid into prostaglandin E2, which attaches to receptors of the same nociceptor and increases the sensitivity of the nerve there. Thereby, the threshold of the nociceptor is lowered at the skin contact and the joint movements are painful. The neurokinins also produce vasodilation (Rubor) and extravasation (Tumor) and thus, in addition to lowering the stimulus threshold (Dolor) and occasionally converting proprioceptive into nociceptive afferents (functio laesa), create the full picture of inflammation, namely neurogenic inflammation. In the

Table 22 Disease prevention and health promotion			
To use all treatment modalities to prevent recurrence of presenting problems	A	3	ac
To recommend exercise and sound ergonomic behaviour for rehabilitation and prevention	A	3	ac
To instruct in self exercises	A	3	ac
See ◘ Table 7 for level numbers *A* attitude, *ac* advanced course			

Table 23 Clinical pictures in manual medicine			
To know and identify disorders or dysfunctions of axial and appendicular structures: – cranium – craniocervical junction – cervical spine – cervicothoracic junction – thoracic spine – thoracolumbar junction – lumbar spine – lumbosacral junction – sacroiliac joints, pelvic girdle – peripheral joints	K	3	bc
To know and identify viscerosomatic, somatovisceral, psychosomatic and somatosomatic reflexes	K	3	ac
To incorporate manual medicine disorders or dysfunctions into rehabilitative concepts, including the ICF model	K	2	ac
To know and identify the disorders and dysfunctions with the appropriate ICD code	K	3	ac
See ◘ Table 7 for level numbers *K* knowledge, *bc* basic course, *ac* advanced course, *ICF* International Classification of Functioning, Disability and Health, *ICD* International Classification of Diseases			

Table 24 Diseases, disorders and conditions			
To understand the differential diagnosis, relevance and interrelationship with manual medicine of the following conditions: – general neurological disorders (signs and symptoms) – neurological disorders – non-cervicogenic headache – orthopaedic disorders – rheumatologic disorders – spinal affections – vascular abnormalities – paediatric disorders – trauma of the spine – tumours of the spine	K	3	bc
To understand special consideration with respect to gender, age and development (especially paediatrics and geriatrics)	K	3	ac
See ◘ Table 7 for level numbers *K* knowledge, *bc* basic course, *ac* advanced course			

case of neurogenic inflammation, manual medical interventions do not work; preparatory pharmacotherapy is required here. Clinical therapeutic side glance: PLA2 is inhibited by steroids, Cox 2 is inhibited by non-steroidal anti-inflammatory drugs (NSAIDs).

Central sensitisation

Long-term nociceptive influx also causes sensitisation processes in the spinal cord, which are essentially comparable to the processes at the nociceptor during peripheral sensitisation (◘ Fig. 5). The processes in the spinal cord can be regarded as much more complex and complicated, since microglia, mast cells, astrocytes and neurovascular complexes are also involved to a large extent. Here, too, the starting point of these processes is the secretory activities of the afferent fibres, and the central, noninducible Cox 2 also plays an essential role in the synthesis of the centrally active prostaglandin E2.

The contemporary terminology for sensitisation of the spinal cord is "neurogenic neuroinflammation" according to Sandkühler [3].

Clinical significance: nociafferent influx is intensified, pain-inhibiting mechanisms are weakened, nociceptive receptive fields increase (pseudoradicular radiations, Head's zones, referred pain), inhibitory receptive fields are reduced and the psychoaffective components of pain perception are intensified; fears and dysphoric states increase [3, 4].

In terms of therapy, the focus here is on centrally effective NSAIDs, acupuncture and suitable proprioception produced by manual medicine.

Inhibitory system

In addition to the opioidergic and serotoninergic descending inhibitory functional systems, the GABAergic inhibition system plays an important role in manual medicine (◘ Fig. 6). By generating proprioceptive afferents (touch, massage, physiotherapy, movement in a pain-free space, manipulation and mobilisation), pain-inhibiting action potentials are generated in GABAergic interneurons, which reduce the level of activity of the multireceptive dorsal horn neurons and thus weaken the conduction of nociceptive excitations.

In addition to the possibility of manually releasing mechanical deadlocks, this manual medical possibility of intervening in neurophysiological pain regulation is likely to play an even more important role in explaining manual medical effects. This does not only seem to have a segmental effect, since a corresponding study has shown an increase in the pressure pain threshold even in places far from the manipulation [5, 6]. The neurophysiology of pain inhibition in general has been known for a very long time [7], but has only recently found its way into differential treatment planning [8, 9]. Clinically, all functional methods also target the pain-relieving systems.

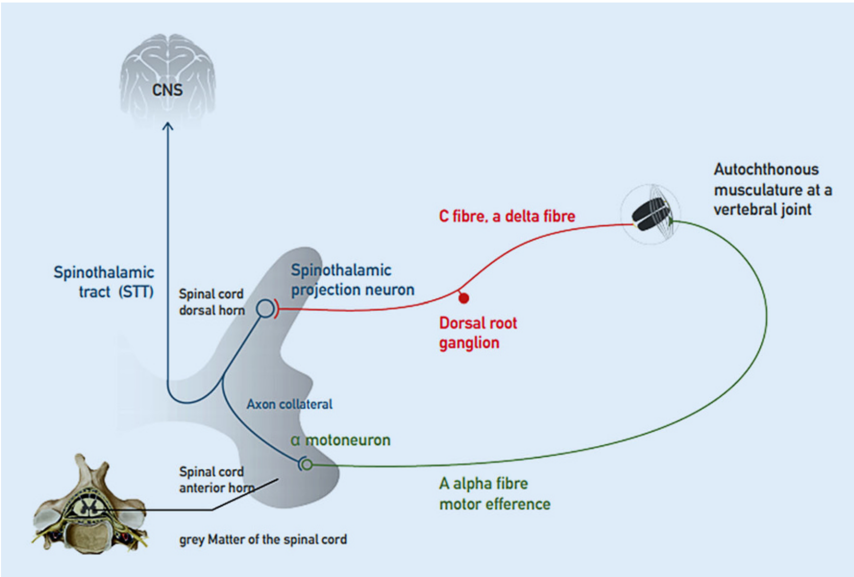

Fig. 1 ▲ Nociafferents arrive from the periphery to the multireceptive posterior horn neuron (MHN). The MHN generates reactive changes in motor coordination via axon collaterals targeting motor neurons in the anterior horn. (With kind permission from © Prof. H. Locher, didactic graphic D. Deltschew. All rights reserved)

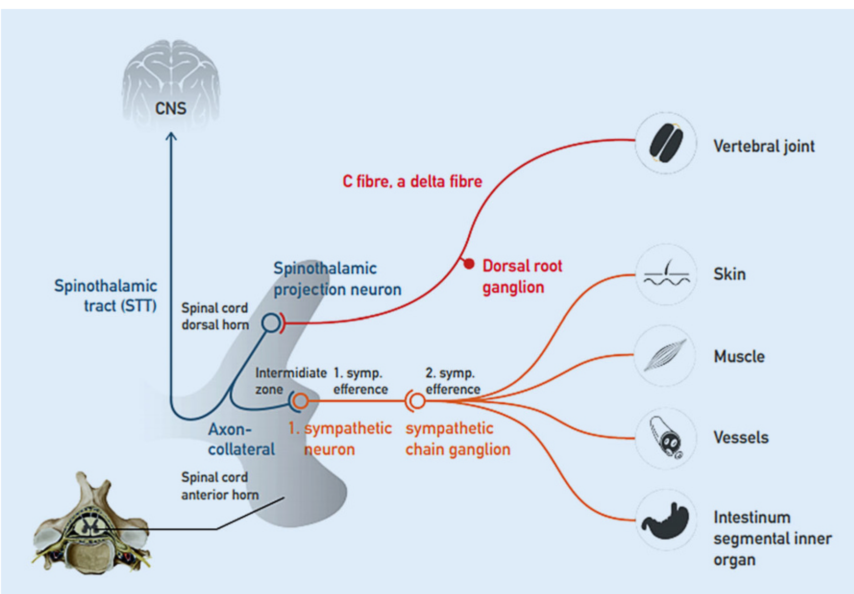

Fig. 2 ▲ Schematic drawing of the pathways of sympathetic system activation. *1. symp. efference* ramus communicans albus (white ramus communicans), *2. symp. efference* ramus communicans griseus (grey ramus communicans). (With kind permission from © Prof. H. Locher, didactic graphic D. Deltschew. All rights reserved)

Conclusion

Basically, manual therapy always has three components that cannot be separated from one another, since the subsystems are intensively networked with one another.

1. Mobilisations, manipulations, massages, etc., or physical stress induce an afferent pattern, which informs the pain-inhibiting systems and integrates them into a response.
2. Likewise, the blood circulation is promoted (massages, mobilisations, etc.), the milieu of the interstitial space is antinociceptively influenced by the blood circulation, thereby also changing the inflammatory situation, triggering immunological reactions, stimulating the signalling substance production of the fibrocytes, triggering the myocytes to release neurotransmitters. All of these active components were also "previously" causes of inflammation and obviously not adequately controlled by an intervention.
3. For example, high-velocity low-amplitude techniques or others eliminate functional disorders at least partially or, at least for a certain time, completely. That depends on whether the coordination is treated in parallel or there are already maladaptive changes in the joint and movement segment structures.

Every treatment technique triggers the activity of the pain-inhibiting systems and modulates the activity level of the peripheral nociceptive causes.

Any therapy that focuses on pain will and should have an intensity-dependent antinociceptive effect. High intensities are necessary for this. At the same time, it will and should reduce the causes of pain.

In the case of degenerative and or inflammatory diseases (arthrosis, rheumatic diseases), in metabolic diseases or after injuries, pain must be minimised directly or indirectly, but then in the long term for the peripheral and central reorganisation, active interventions need to be carried out. Maladaptation is not reversible, but represents the training status of the sensorimotor system; the muscles decide as an afferent pattern and as a biochemical signal generator about the pain situation.

Summary

All forms of manual therapy and many other functional therapy methods aim, among other things, at influencing pain-inhibiting systems [2].

Nociceptive afferent input into the central nervous system (CNS) needs to be reduced in order to facilitate the return of structural and functional musculoskeletal normality [10].

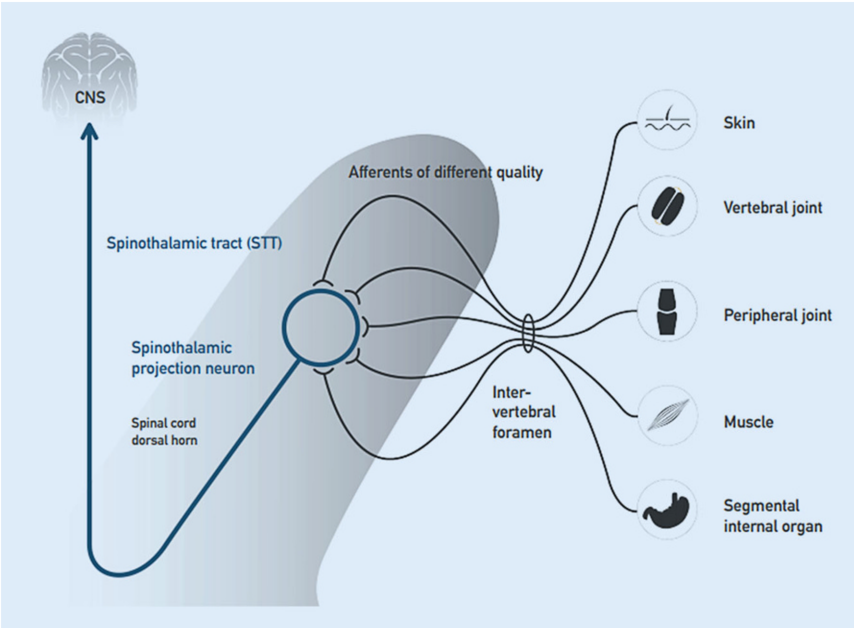

Fig. 3 ▲ Afferents from different tissues converge on a dorsal horn neuron, which why it is often called a wide dynamic range neuron (WDR). (With kind permission from © Prof. H. Locher, didactic graphic D. Deltschew. All rights reserved)

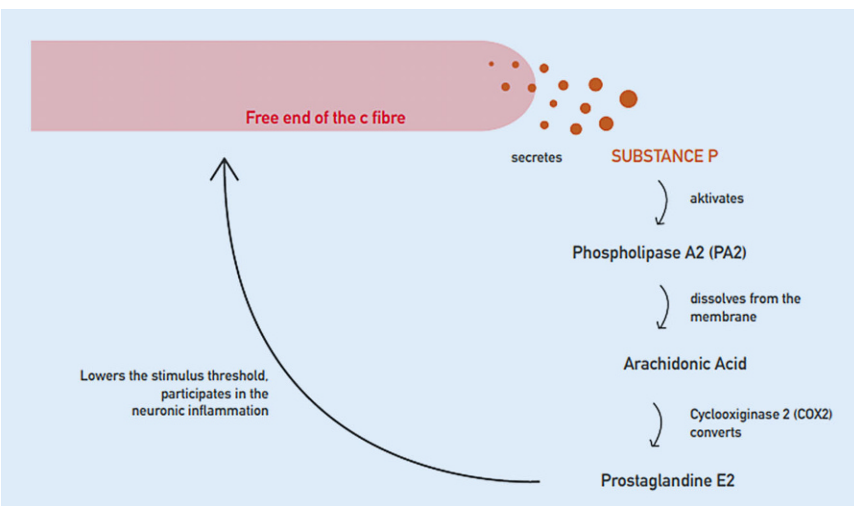

Fig. 4 ▲ Mechanisms of peripheral sensitisation. (With kind permission from © Prof. H. Locher, didactic graphic D. Deltschew. All rights reserved)

Principles of mobilising treatments of the spine

General considerations

1. The locomotor system and the autonomic nervous system respond to afferent input of any origin.
2. Segmental functional testing may increase or decrease nocireactive motor response in terms of changes in the tension of muscles and fascia.
3. Afferent input may also initiate reflex changes of tissues innervated by the autonomic nervous system.
4. Increased muscle tension results in reduction of passive range of motion of vertebral segments.
5. Segmental dysfunction can be identified by palpation.
6. Clinical functional examinations attempt to identify altered movement patterns that represent both restriction and free direction of motion.
7. Vertebral dysfunction may be caused by pathologies outside the locomotor system.

Manual medicine (provided by physicians) uses all the most up-to-date medical skills and knowledge such as anatomy, biomechanics, physiology, biochemistry and imaging.

The unique property of manual medicine amongst other medical specialties is the possibility of entering into the system of diagnosis by identifying the motor and other reactions by palpation (i.e., by the reproducible finding of functionally induced tissue changes; ◘ **Figs. 7 and 8**).

Principles of manual diagnostics

Prior to manual examination, a standard orthopaedical and neurological examination is always performed.

1. Global range of motion (ROM)—asymmetry?
2. Segmental or regional range of motion (mobility = **M**)
3. Segmental irritation = **I** (activation of afferent neurons followed by nocireaction)
4. Provocation to identify painful direction(s) = **P**
5. **MIP** diagnostic system is essential to identify the reversibility of any dysfunction of the spine
6. To plan any manual treatment, the **MIP** test must reveal at least one pain-free or unrestricted direction per plane

Principles of manual therapy

1. Basically, there are four possibilities of manual treatments:
   - Manual mobilisation—without thrust
   - Manual manipulation—with thrust
   - Neuromuscular techniques
   - Soft tissue techniques
2. Before the decision about the therapeutic manual approach, be aware of possible contraindications
3. No increase in nociception, no increase in pain and nocireaction during therapy
4. Achieving long-term decrease of nociceptive activity of multireceptive dorsal horn neurons

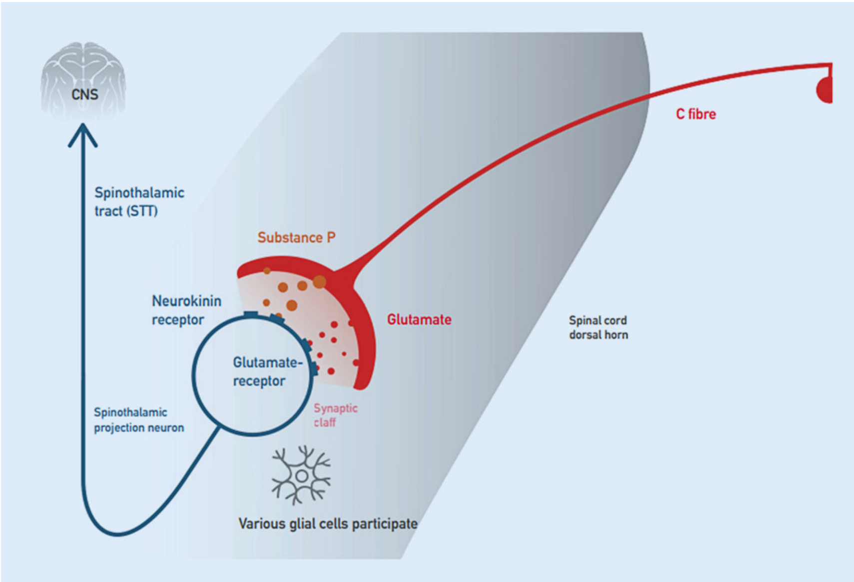

Fig. 5 ▲ Mechanisms of central sensitisation. (With kind permission from)

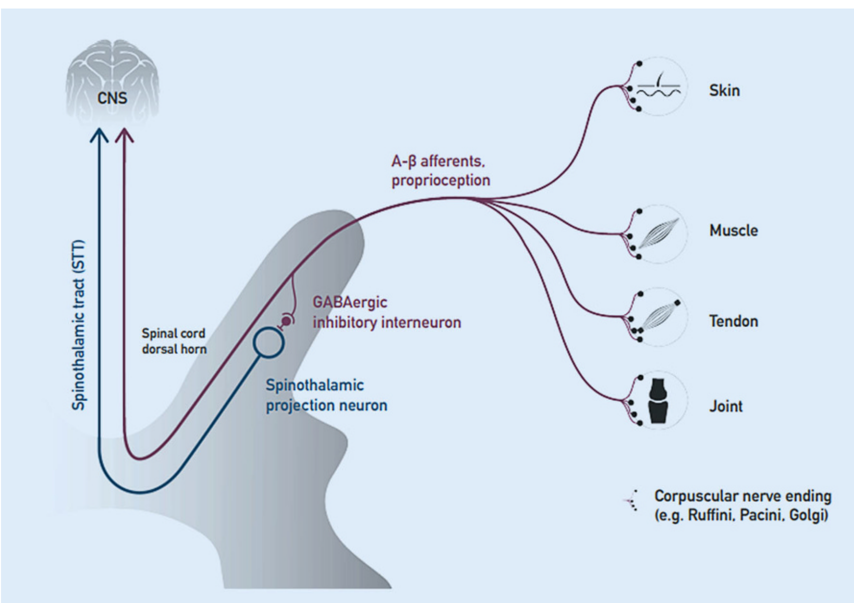

Fig. 6 ▲ GABAergic interneurons as mediators of inhibitory activity through proprioceptive influx. (With kind permission from)

Cervical spine

Diagnostic procedures of spinal dysfunction—cervical spine.

1. Global range of motion (ROM)
2. Segmental ROM (end feeling) **M**
3. Segmental irritation **I**
4. Combined functional testing
5. Provocation: for the preparation of a thrust treatment, it is necessary to distinguish the directions of increase or decrease of nociception **P**
   MIP **M**obility—**I**rritation—**P**rovocation

Therapeutic techniques—cervical spine.

1. Global
   - neuroinhibitory techniques
   - segmental soft tissue techniques
2. Regional or segmental
   - neuromuscular techniques
   - segmental soft tissue techniques
   - segmental mobilisation (direct/indirect) in addition with facilitation using respiration and eye movements
3. Before the decision about the therapeutic manual approach, be aware of possible contraindications
4. No increase of nociception, no increase of pain and nocireaction during therapy
5. Achieving long-term decrease of nociceptive activity of multireceptive dorsal horn neurons

Thoracic spine

Diagnostics of spinal dysfunction—thoracic spine.

1. Posture
2. Global mobility
   - bending forward—bending backward—side bending
   - rotation in sitting position
3. Information about segmental **i**rritation **I**
   - segmental **m**obility **M**
   - muscular hypertonicity, nocireactive motor patterns
   - symptoms of autonomous regulation (skin rolling test, dermographism, skin temperature)
4. Segmental **p**rovocation by functional movements searching for functional asymmetries **P**
   MIP **M**obility—**I**rritation—**P**rovocation

Therapeutic techniques—thoracic spine.

1. Global
   - soft tissue techniques
   - axial traction techniques in upright position
   - tangential push-traction
2. Regional or segmental
   - crossed-hand technique in prone position
   - supine thenar technique
   - technique on tender points
   - neuromuscular inhibition techniques

Rib dysfunction

Diagnostics—rib. Rib

- **M**obility: costal or intercostal motion during respiration **M**
- **I**rritation: area insertion of the levator costae muscle **I**

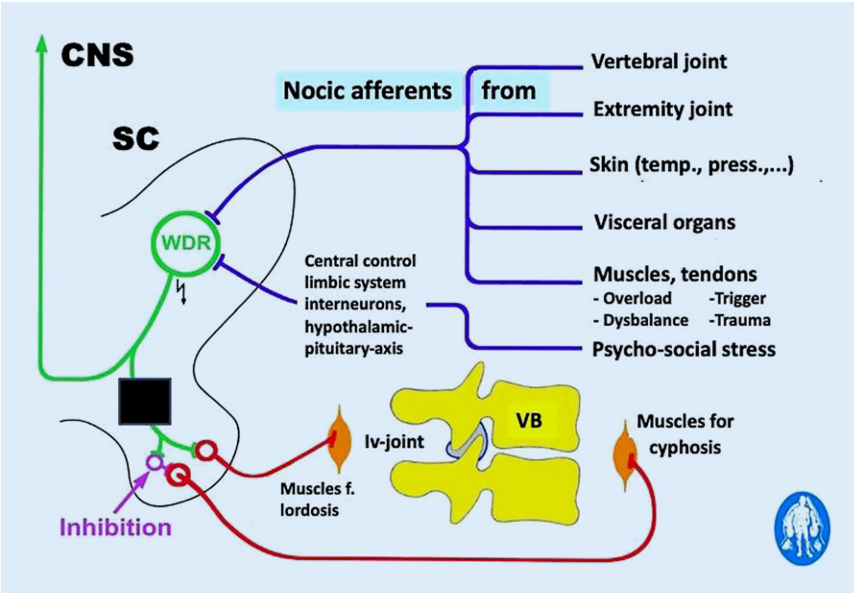

Fig. 7 ▲ Model of the neurophysiology of segmental dysfunction. The terms "muscle for lordosis" and "muscle for kyphosis" mentioned in the figure are used as a highly simplified model of three-dimensional complex innervation patterns of spinal muscles as a neurophysiological reaction of body protection. *VB* vertebral bone, *Iv* intervertebral joint, *SC* spinal cord, *WDR* wide dynamic range neuron. (With kind permission from © Prof. H. Locher, MWE)

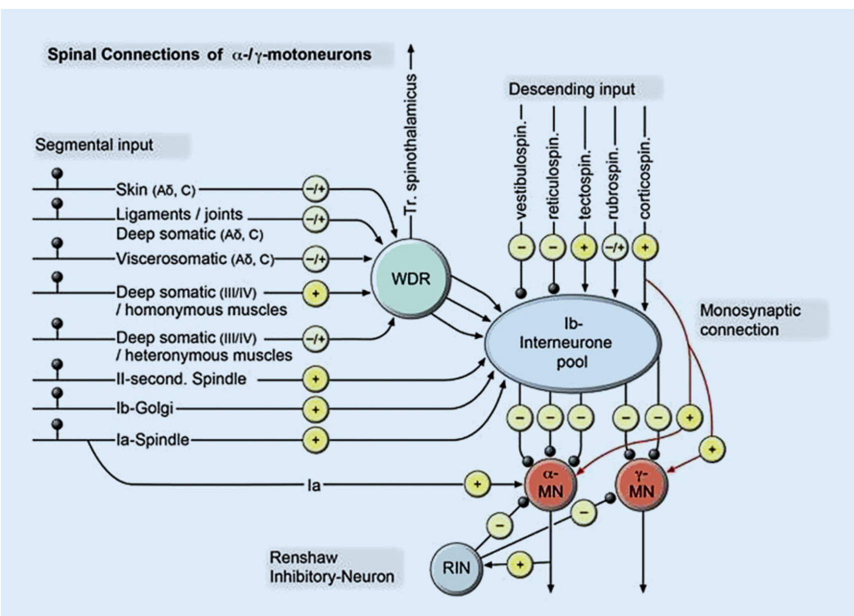

Fig. 8 ▲ Some known contents in the neural connections of the spinal cord shown in **Figs. 1–7**. *WDR* wide dynamic range neuron, *RIN* Renshaw inhibitory neuron, *MN* motoneuron. (With kind permission from © Böhni, Lauper, Locher; 2014)

- Provocation: checking irritation under inspiration and expiration **P**
  MIP Mobility—Irritation—Provocation

Therapeutic techniques—rib. Rib
- Mobilisation in prone position
- Crossed-hand technique in prone position
- Mobilisation in lateral position
- Supine thenar technique

Lumbar spine

Diagnostics of spinal dysfunction—lumbar spine.
1. Posture
2. Testing of regional or segmental Mobility **M**
   - side bending, flexion extension
   - rotation in sitting position
3. Irritation: paraspinal segmental muscles **I**
4. Provocation: check for painful and pain-free motion directions **P**
   MIP Mobility—Irritation—Provocation

Therapeutic techniques—lumbar spine.

Lumbar spine
- Soft tissue techniques
- Neuromuscular techniques
- Regional or segmental mobilisation (e.g., rotation traction in lateral recumbent or prone position)

Sacroiliac joint (SIJ) and pelvic girdle

Preliminary remarks. All movements of SIJ components are defined by the anatomic form of the joint surfaces of the ilium and the sacrum and are physiologically possible only in a minimal range of a few degrees (2–4°). In contrast to all other joints according to the definition of true diarthrosis, actively intentioned movements within the SIJ in a functional direction are not possible. Therefore, movements of the SIJ are not comparable with the functional pattern of other joints.

Didactically motivated orientation on functional three-dimensional axes has no basis in functional biomechanical evidence and should not be considered further. In the basic course, we teach only techniques that involve unspecific forces to the SIJ components. The selection of techniques depends on the results of the functional examination and the pain-provocation tests. The aim is to influence the dysfunction based on reactive processes in order to induce a reduction of tension and pain.

There is wide variation in the accepted practice of SIJ testing. A general consensus has not been achieved. Regarding forward bending or so-called spine tests, even though clinical mobility exists, diagnostical conclusions are uncertain. At this stage we note that mobility tests have not been proven sufficiently reliable. Instead

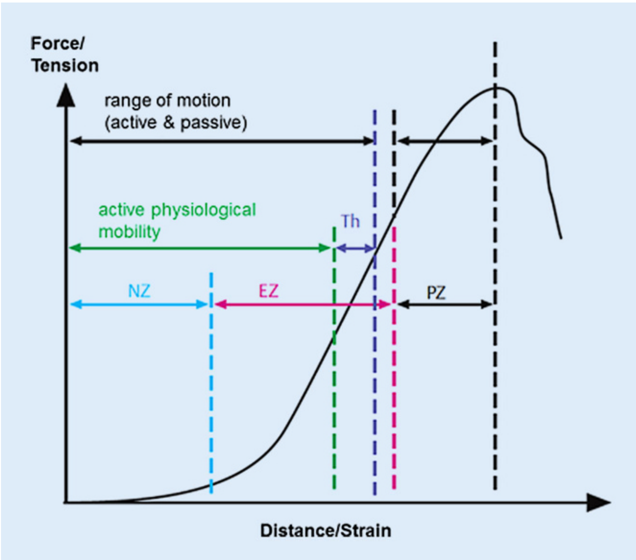

Fig. 9 ▲ Diagram on distance/force adapted to movements of joints (and segments) concerning the degree of mobility (joint play). *NZ* neutral zone, *EZ* elastic zone, *PZ* plastic zone, *Th* therapeutic range. (With kind permission from © Böhni, Lauper, Locher, manual medicine 1, 2014, Thieme)

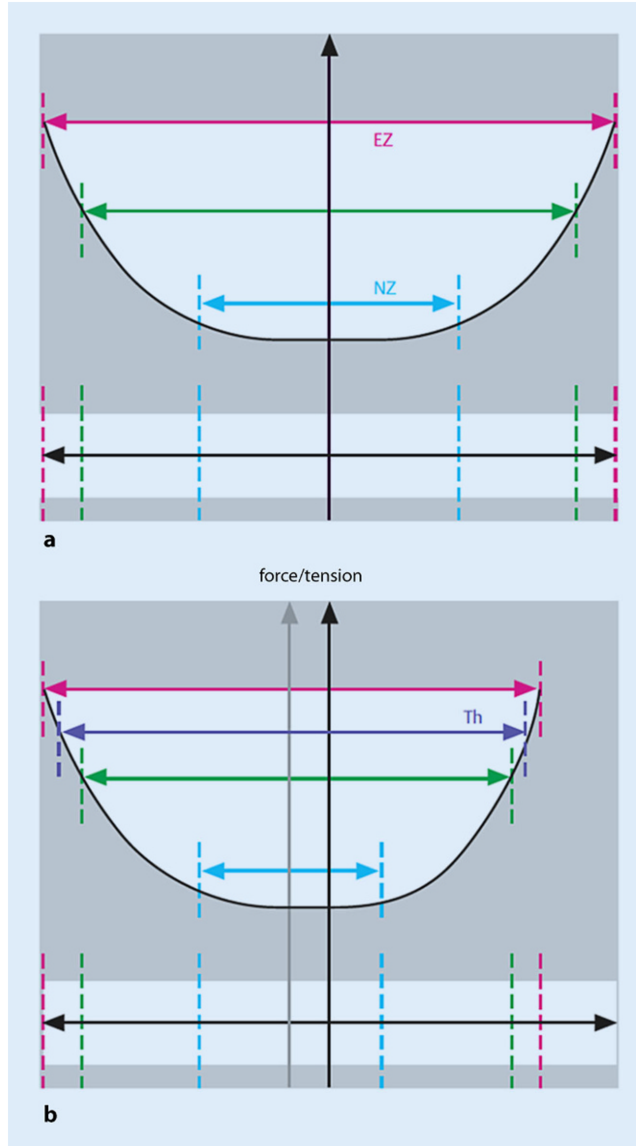

Fig. 10 ▲ Diagram on distance–force (tension) of one degree of freedom of mobility (e.g., flexion/extension or gliding anteroposterior/posteroanterior). **a** Normal findings. **b** Restriction to the right side, actual neutral position moved to left side (*grey ordinate*). *EZ* elastic zone, *NZ* neutral zone, *Th* therapeutic range. (With kind permission from © Böhni, Lauper, Locher, MM1, 2014, Thieme)

of using mobility tests, it seems therefore advisable to use pain-provocation tests.

Signs of irritation in SIJ-related tissues may give information on SIJ dysfunction, acknowledging the fact that projections on S1 and S2 also derive from different lumbar segments.

No other dysfunction deserves more consideration of differential diagnoses than the SIJ. The initial diagnostic procedure should exclude other (such as lumbar or higher convergence) dysfunctions before identifying SIJ dysfunction.

Depending on the results of functional or pain-provocation tests as well as other clinical findings concerning the SIJ, the decision about appropriate therapeutic techniques can be made.

Diagnostics of joint dysfunction—sacroiliac joint and pelvic girdle. Sacroiliac joint and pelvic girdle

- Compression test
- Distraction test
- Thigh thrust (**4 P** test = "**p**osterior **p**elvic **p**ain **p**rovocation")
- Sacral springing test
- Pelvic torsion test (Gaenslen test)
- **F**lexion–**ab**duction–**e**xternal **r**otation test (**FAbER** test, Patrick test, sign of 4)

Therapeutic techniques—sacroiliac joint and pelvic girdle. Sacroiliac joint and pelvic girdle

- Nonspecific mobilisation in nutation/counternutation direction
- Traction mobilisation by vibration
- Adduction mobilisation in prone position
- Ilium rotation to induce sacrum nutation without high-velocity thrust

Principles of mobilising treatments of peripheral joints

General considerations

In order to define the appropriate technique for manual treatment of peripheral joints, a concise basic examination of the respective joint is necessary. This examination comprises investigating the functional movement, the range of motion in all planes, the joint play and the palpation of the different tissue levels in order to differ-

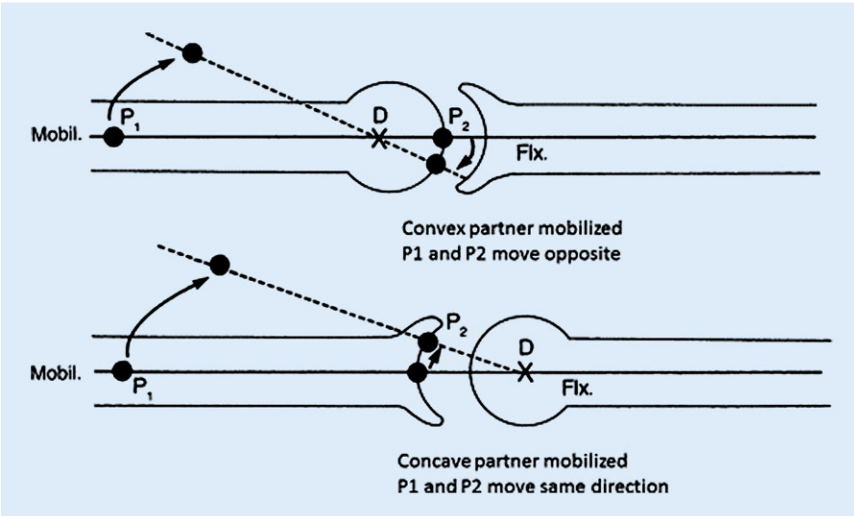

Fig. 11 ▲ Convex–concave rules. *P1* distant point to joint, *P2* near point to joint, *D* rotation point, *Mobil.* punctum mobile, *Flx.* punctum fixum. (With kind permission from © Bischoff & Moll, 6th ed., 2011, perimed)

entiate structural lesions from functional disorders. This is a precondition before starting with any manual treatment.

In general, manual techniques in peripheral joints are indicated in the following conditions:

1. Reduction of functional mobility and joint play
2. Painful functional disorders
3. Posttraumatic, postinflammatory, postoperative, postimmobilisation and degenerative stiffness
4. Certain cases of neuropathology (e.g., spastic paralytic contractions)
5. Post-CRPS stiffness or contraction

In terms of peripheral joints, we talk specifically about joint play (◘ **Fig. 9**). These movements are related to some considerations involving general biomechanical rules of joint mobility.

In this sense, we follow the model of dysfunction as a reduction in the total joint play (◘ **Fig. 10**).

The following movements in a joint can be differentiated and should be taken into consideration before planning any kind of diagnostic or therapeutic approach:

1. Rolling = wheel runs on a road
2. Gliding = wheel turns on the spot
3. Roll-gliding = wheel runs and glides at same time
4. Translation = wheel glides without rotation
5. Axial forces: compression (coaptation) and separation (decoaptation)

Although the exact role of compression to a joint during a manual procedure has not been completely defined, it has been shown that the improvement of joint play movements will have a positive influence on the nutrition and regeneration of joint cartilage. The repetitive change of compression and separation acts as a pump of synovial fluid in and out of the cartilage. This way compression may have a positive therapeutic influence on joint cartilage; it may also improve proprioception and reduce nociception as well as having positive effects on the capsule.

In order to improve any functional movement of a peripheral joint, the reduced joint play capacity of the respective joint must be increased.

In terms of the peripheral joints, the convex–concave rules have to be considered:

1. In case of mobilisation of the (peripheral) convex partner:
   - P1 and P2 are moving in the opposite direction
2. In case of mobilisation of the (peripheral) concave partner:
   - P1 and P2 are moving in the same direction (◘ **Fig. 11**)

This means, for example, that in order to improve abduction in a shoulder, the humerus head has to be mobilised in a caudal direction, or to improve flexion of the knee, the tibial head must be mobilised in dorsal direction.

Therapeutic procedures on limbs are possible applying traction or light compression.

The capsular pattern indicates specific movement restrictions. Each joint has its typical capsular pattern. In a hip joint this is the internal rotation (and extension).

For functional examination, follow the algorithm **MANSC-VV**:

- **M**yofascial
- **A**rticular
- **N**euromeningeal
- **S**tabilisation tests
- **C**entral disorders
- **V**isceral and **v**ascular

Upper limb

Finger joints. Therapeutic procedures:

- Dorsal and palmar mobilisation
- Lateral mobilisation to both sides
- Rotational mobilisation
- Three-dimensional mobilisation

Thumb. Saddle join: convex–concave in one plane, concave–convex in another plane.

Therapeutic procedures:

- Mobilisation of the saddle joint

Wrist.

1. Diagnostic procedures:
   - check for function (extension, flexion, abduction, adduction)
   - check the mobility of every carpal bone—in two rows (respect the convex–concave rule)
2. Therapeutic procedures:
   - traction
   - translation of the first or second row in all possible directions
   - mobilisation of each carpal bone individually

Elbow.

1. Diagnostic procedures:
   - check function humeroulnar, humeroradial and radioulnar joint (range of motion)
   - palpation: muscles, ligaments, insertions and nerve passages

Table 25 Diagnostic procedures for the foot

	Joint line	Diagnostic procedure
1	Upper ankle (tibiotalar)	Flexion–extension
2	Lower ankle (talocalcanear)	Extension–flexion
3	Chopart and Lisfranc lines–middle foot	Supination–pronation
4	Metatarsophalangeal joints (I–V)	Inversion–eversion

2. Therapeutic procedures:
   - traction
   - soft tissue techniques to the elbow
   - mobilisation of the elbow

Shoulder girdle. The shoulder has five different areas of mobility, which all have to function correctly. All areas and related muscles must be tested.

- Acromioclavicular joint
- Sternoclavicular joint
- Glenohumeral joint
- Scapular-thoracic gliding area
- Subacromial gliding space

1. Diagnostic procedures:
   - examination directions:
   - abduction and elevation
   - internal and external rotation
   - movements to back and neck
   - articular mobility anteriorly and medial clavicular ligaments
2. Therapeutic procedures:
   - soft tissue and muscle techniques
   - mobilisation techniques:
     scapular-thoracic gliding area
     subacromial space
     acromioclavicular joint
     sternoclavicular joint
     glenohumeral joint

Lower limb

Foot. Table 25.

Toes, metatarsal connections, tarsometatarsal, ankle and subtalar joints, distal tibiofibular connection:

Therapeutic procedures:

- Soft tissue and muscle techniques
- Mobilisation with respect to the individual joint play

Knee. Tibiofibular, femorotibial, patellofemoral joint.

1. Diagnostic procedures:
   - functional mobility and joint play
2. Therapeutic procedures:
   - soft tissue and muscle techniques
   - mobilisation

Hip.

1. Diagnostic procedures:
   - examination of mobility and joint play
2. Therapeutic procedures:
   - soft tissue and muscle techniques
   - mobilisation

The nature of segmental dysfunction

Introduction

Using the biomedical model rooted in Aristotelean logic, neuromusculoskeletal disorders have been thought to be caused by structural change or injury to tissues, which in turn drives pathology and its symptomatology [13]. On the other hand, manual medicine practise rests on different principles, where it is the ability of the human body to self-regulate, self-heal and maintain its health that is the focus, with the practitioner working to restore the interrelationship between structure and function [14]. Therefore, segmental dysfunction (SD) encompasses a more holistic and multifactorial approach, where the disruption of normal physiological function of the neuromusculoskeletal system is determined by changes in neurophysiology, sensorimotor functions and the autonomic nervous system, as well as by motivational affective cognition and other psychological factors [13]. This makes SD a complex entity to explore, understand and treat.

Physiological changes

By different authors, SD was shown to be associated with segmental neurophysiological facilitation at the spinal cord level, which in turn effects changes in peripheral tissues and viscera.

Further elaborations of Korr's model developed to accept that SD originates from a nociceptive stimulus within a body structure, with or without actual structural damage or injury [15]. In this "pain model", nociceptive neurons activated peripherally within viscera or somatosensory structures (e.g., skin or musculoskeletal components, including connective tissue) activate nociceptive responses in adjacent peripheral tissues as well as interneurons with which they synapse in the dorsal column at a specific nerve root segment in the spinal cord. This afferent signal processed by interneurons has ramifications in other parts of the nervous system, for instance ventral horn motor neurones, autonomic nervous system ganglia, and via the spinothalamic tract to central nervous system pain-processing centres such as the insula, anterior cingulated cortex, prefrontal cortex and somatosensory cortex [14–16]. The orchestrated activation and interplay of the different branches of the nervous system (peripheral, central and autonomic) interact to maintain and modulate the nociceptive drivers, fostering the malfunctioning of musculoskeletal and/or visceral structures. As there is no overarching model that helps determine the balance and interactions between all the pathophysiological pathways that drive pain and the development of SD, it is helpful to review each process individually, as they manifest in each patient in different proportions and context.

Peripheral inflammation. It is well established that direct or indirect injuries, as well as functional limitations (i.e., without structural damage) can be characterised by inflammatory responses in the affected tissues [17]. In musculoskeletal SD, the pain generators include muscle, tendon, ligament, fascia, intervertebral disc, articular cartilage, joint capsule, bone (periosteum) and vascular stroma [18]. The activation of nociceptor free endings within the connective tissue of visceral, dermal or musculoskeletal structures will cause the local release of peptide transmitters, such as somatostatin and substance P, as well as other inflammatory mediators such as histamine, serotonin and immune complements [15]. This leads to local tissue vasodilation, immune cell activation (e.g., macrophages and lymphocytes) and oedema due to increased leaky capillaries. The modulation of the nociceptive neuron ensures this response spreads from the site of the original nociceptive stimulus to the surrounding area, recruiting other nociceptors and lowering their threshold to respond to further stimulus, creating a cascade response that further assists other branches of the nervous system, such as autonomic response and nociceptive reflex.

Nociceptive reflex. The activation of nociceptors has a modulatory effect, via their cell bodies in the dorsal root ganglia, at the corresponding segmental spinal level, where they synapse directly, or indirectly via interneurons, in the dorsal horn. Through a series of interneuronal connections within the spinal laminae, a signal is transmitted to the segmental motor neurones in the anterior horn, which in turn exerts a musculoskeletal reflex in the muscle fibres affected by the nociceptive stimulus, leading to muscle spasm or hypertonicity. However, the web of interneurons amplifies the nociceptive signal beyond the affected segment, thus orchestrating a variety of coordinated sensory, musculoskeletal (somatic) and autonomic (visceral) responses, which affect the functioning of multilevel dermatomes, myotomes and viscerotomes [15]. It is therefore possible to infer that a nociceptive reflex can cause a combination of somatosomatic, somatovisceral, viscerosomatic and viscerovisceral responses.

Autonomic response. Theories that the autonomic nervous system modulates peripheral and visceral responses following a peripheral painful or nonpainful stimulus have been proposed since the 1950s [19]. Furthermore, Korr demonstrated that sympathetic pathways at segmental levels correlating to the SD showed increased activity [20], which is thought to explain why pain can persist once the nociceptive stimulus appears to have been removed, with sympathetic activation facilitating the sensitisation of local nociceptors, giving way to clinical signs such as hyperalgesia and allodynia [21]. From a neurophysiological viewpoint, the precursor of the recruitment of the sympathetic system in the development of SD is thought to be the nociceptive reflex, where segmental activation of the sympathetic root ganglia by nociceptors or interneurons causes a *nociautonomic reflex* commensurate to the autonomic function of the segmental level affected, e.g., increased heart rate, vasoconstriction in viscera and skin, vasodilation in muscles, raised blood pressure, increased gastrointestinal stasis or bronchodilatation [15]. These are the basic mechanisms supposed to be responsible for development of CRPS 1 and 2. Furthermore, an immune component with autonomic modulation cannot be excluded, as the thymus, spleen and lymph nodes receive sympathetic innervations that could modulate T and B cell function [15]. Overall, autonomically driven changes can explain the changes observed in the tissues affected by SD, such as the tissue fullness and movement restriction due to muscular oedema and swelling.

Proprioception. The characteristics noted in an SD, such as tissue changes and tenderness, are thought not to merely limit musculoskeletal movement range and function, but also its spatial sense of position, otherwise known as proprioception [22, 23]. In fact, the changes exerted onto an area of SD via somatic and autonomic pathways affect the position in which a body part is held [15]. For example, the muscle contracture due to a somatic nociceptive reflex or the muscle engorgement due to a sympathetic nociautonomic reflex moves the joint correspondent to the area of SD, away from its neutral position. Not only would this affect the available range of motion of that joint, but recalibrate the afferent sensory input, thus changing its neutral position and proprioceptive balance. Furthermore, the apparent inhibition of deep segmental muscles and increased activation of superficial musculature caused by nociceptive drive may also impair proprioception, further exposing an area of SD to further injury due to poorer motor control [24].

Central sensitisation. Pain is not merely a passive relay of neuronal action potentials dependent on the location and intensity of the peripheral noxious stimulus, but rather, as Melzak & Wall showed in 1965, pain perception depends on a complex endogenous pain modulation and inhibition system within the CNS [25]. It has become well established that the majority of chronic musculoskeletal pain and SD are characterised by dysfunction of CNS processing, where the balance between descending anti-nociceptive (inhibitory) mechanisms and ascending nociceptive facilitation is disrupted, causing an amplification of pain transmission [16]. The increased sensitisation and excitability of neurons in the central nociceptive pathways, such as dorsal column and laminar interneurons, represents an increase in presynaptic signal strength or postsynaptic response, as well as a reduction in descending inhibition [25]. It therefore gives a neurobiological basis to explain phenomena such as *hyperalgesia*, where there is an increased and prolonged response to a noxious stimulus, and *allodynia*, where the pain threshold is reduced [24]. The pain hypersensitivity observed in central sensitisation thus represents an amplification of central nervous signalling [25].

Fascial modulation. Recently, the role of the fascia has been revisited to give a different dimension to the nociceptive and central sensitisation models used to describe SD. The mechanical importance of the fascia stems from the recognition that normal musculoskeletal movement does not rely solely on each individual structure (tendon, ligament, muscle, fascia, fat pad and bursae), but on their interrelation as a "supertendon", where the function of the whole is greater than that of its individual parts [26]. Therefore, the idea of fascia as an encompassing organ supporting muscles and viscera organ developed. In this context, a specific insult to the fascia, such as a strain, mechanical overload or painful stimulus, triggers the sensory system and activates a chain reaction that leads to the formation of an SD.

Diagnostic applications

The clinical manifestation of SD can be thought of as the cumulative result of the pathophysiological changes underpinning it. However, the significant heterogeneity of clinical features in each patient requires a practitioner to harness diagnostic skills capable of recognising and correlating the clinical features of SD with the patient's history [27]. This reliance on clinical acumen therefore has a tendency toward poor diagnostic reliability [24], as the ability to characterise each SD is operator dependent. Nonetheless, our understanding of the pathophysiological process can help to single out recurrent clinical features of SD and develop a structured framework for diagnostic purposes, which then facilitates the placing of an SD in a clinical context relevant to the patient's ailment and condition.

Sensory changes. During the clinical examination, the most common sensory changes noted in SD include tenderness, paraesthesia and numbness, as well as alterations in temperature perception and proprioception. Tenderness can arise directly from a physical structure, such as joint capsule or trigger point, thus indicating a specific source of SD, or as formulated in the central sensitisation model, it can manifest as hyperalgesia or allodynia within any tissue or segmental region of the neuromusculoskeletal system. Therefore, pain history and other sensory changes are an important part of the differential diagnostic process, as they provide an insight into the particular anatomical distribution of a SD. In fact, in a cross-sectional study, 455 patients with chronic low back pain were reviewed and a good correlation was found between pain measured using a 0–10 visual analogue scale (VAS) and the location of the tender somatic dysfunction, with the lumbosacral region as the most common site of the SD [28, 29]. Similar findings were noted in an earlier study of 216 patients with chronic musculoskeletal pain, where a good statistically significant link was found between SD and pain distribution [30]. Although the pathophysiology of neuromusculoskeletal pain shows that its location does not always correlate with the underlying aetiological site, these studies lend support to using tenderness and sensory changes as a guide to locate primary or secondary SDs and start "mapping" a diagnostic formulation.

Tissue texture changes. Alongside the sensory dysfunctions noticeable with an SD, changes to the palpatory qualities of a tissue are another integral component of the clinical diagnostic process. In line with the pathophysiological changes expected with SD, changes to tissue texture can be driven by local inflammation, fascial disturbances, and nociautonomic and nociceptive reflexes [15, 21, 25, 31, 32], which in turn can lead to physical tissue changes such as increased skin drag, tissue oedema, temperature changes, increased perspiration, muscle spasm and fascia contracture [14]. These tissue features are the somatosomatic and viscerosomatic representation of SD and can be traced back to a specific structure (e.g., muscle, ligament, tendon, articular joint), as well as a single spinal segment or a group of them [33], thus facilitating the location of the primary and secondary SDs. Overall, despite the variability introduced by the subjective palpatory skills of clinicians, the tissue changes observed define the clinical picture that helps to determine possible differential diagnoses.

Asymmetry of anatomical landmarks. In addition to sensory and tissue changes, other clinical features associated with SD include anatomical asymmetry [27], especially affecting the vertebral column and pelvis, although any musculoskeletal structure can show visible anatomical differences with its corresponding opposite. Recognition of anatomical asymmetry rests on the clinical skills of the clinicians, from observation (e.g., for scoliosis, posture or scapular protraction) to palpation, where a sacral torsion or anterior/posterior innominate is confirmed after tactile assessment.

Restriction of joint movement. Another important clinical feature that defines SD clinically is motion restriction of an articular joint, typically affecting a zygapophyseal joint of a spinal segment, although any articulation of the musculoskeletal system can be involved [14]. Pathophysiological factors such as joint inflammation or proprioceptive deficits may theoretically create a motion restriction in isolation with no tissue, sensory or visible anatomical changes. However, in reality, motion restriction is a functional impediment due to a combination of elements, from muscle spasm and tissue oedema to pain-induced muscular inhibition and postural adaptations [14, 27]. In fact, it is a recognised practice among manual medicine clinicians to monitor tissue tension and changes to better understand the motion restriction at a specific joint or spinal level [33], thus highlighting the importance of monitoring the overall behaviour of an area to the changes imposed by palpation and motion.

Therapeutic applications

Once the SD has been diagnosed and defined by the clinical examination, a therapeutic approach can be planned using different manual medicine techniques in order to reduce pain and restore normal physiological parameters: tissue texture, symmetry, motion range and function. However, in manual medicine, a patient-centred holistic approach requires a variety of therapeutic modalities to address both physical and biopsychosocial drivers of SD [34]. Therefore, exercise prescription, postural changes, ergonomics and education are integral components of the treatment plan [35]. Still, the use of manual therapies remains the cornerstone of treatment to reverse neurophysiological dysfunctions. In practice this approach focuses on both on primary and secondary dysfunctions, in recognition of the interplay between different structures on the neuromusculoskeletal system and the inherent difficulty in differentiating between causal SDs and consequential or adaptive SDs [17].

Soft tissue techniques. The localised tissue changes driven by inflammation, autonomic and somatic responses can be improved and reversed by soft tissue techniques. The manual mobilisation of tissues facilitates motion, pliability and muscle relaxation [27], probably due to a combination of local effects, for instance mobilisation of oedema by *effleurage* [34] and segmental feedback via spinal cord autonomic and visceral reflexes. Specific soft tissue and fascial release techniques have been shown to reduce pain, possibly due to modulation of connective tissue viscoelasticity, piezoelectric properties and hydration [36], as well as reduced proinflammatory mediators and fibroblast proliferation [37]. The application of mechanical load appears to be the main modulator of soft tissue responses [31, 32], although this seems to relate to the type, duration and frequency of the kinetic load.

Joint mobilisation. In order to restore normal active and passive articular motion, the application of repetitive and oscillatory movements to engage with the restricted barrier can be effective and is termed *joint mobilisation*. The normal range of movement needs to be assessed and tested in all its planes, flexion/extension, rotation and side bending, but also in its accessory movements, from anteroposterior to mediolateral and cephalocaudal translation [33]. The joint mobilisation can be delivered passively by the clinician, or it can be facilitated by muscle energy techniques (MET) that relax the hypertonic muscle maintaining the movement restriction by using an isometric contraction to cause motoneuron inhibition via its corresponding somatosomatic reflex mediated by Golgi tendon organ and dorsal column interneurons [38, 39].

Manipulation and high-velocity low-amplitude (HVLA) thrust. HLVA thrusts are frequently considered the defining treatment of manual medicine, and significant research has focused on elucidating the therapeutic properties of these manipulations, although no current mechanistic model is able to fully explain their effect. However, there is supporting evidence showing that manipulations exert improvements in the parameters that define SD and restore homeostasis.

Pain physiology education. Chronic musculoskeletal pain and its associated SD are often characterised by features of central sensitisation, where addressing the biopsychosocial context is an essential part of the therapeutic approach [16, 25]. In fact, all the manual therapy techniques available are insufficient on their own to treat and benefit patients, as we now know that levels of vigilance, stress and anxiety modulate descending inhibitory nociceptive mechanisms and contribute to aggravate central sensitisation, where patients experience pain as more threatening and catastrophic, developing lower pain tolerance and poorer coping strategies. A promising therapeutic strategy comes not from new pharmacological agents, but from patient education. In fact, pain (neuro)physiology education to reduce the gap between the perception of the patient and their therapist about pain and its treatment was shown to increase patient motivation for rehabilitation [16]. The long-term effectiveness of this approach is currently awaiting validation, but it should already encourage its use in managing SD with patients affected by chronic pain.

Functional treatment. As there is growing appreciation of the importance of addressing perpetuating factors behind SD [34], it would appear logical to strengthen traditional therapeutic strategies based on manual therapies with functional treatment or rehabilitation. This recognises the interdependence of neuromusculoskeletal structures with their collective function and use, thus allowing clinicians to add an active, engaged and holistic treatment component where patients are not merely passive recipients of treatment. Therefore, alongside manual medicine and patient education, functional treatments such as prescription of physical activity; respiration, core stability and postural exercises; mindfulness training; and sleep and nutritional advice should form part of a patient-oriented approach to individualise and optimise care [31, 32, 34, 40]. This adds a new and complex dimension to our understanding of SD, but also provides a promising new paradigm.

Discussion

Our pathophysiological understanding of SD has significantly evolved as our scientific knowledge has progressed. From its initial concept of imbalanced bodily fluids and humors [41], through a reductionist phase where basic neurophysiology appeared to explain most nonstructural pain, we have recently started to embark on a novel trajectory where our basic paradigms of pain physiology and body functions are challenged by the complex interactions of different systems, where the somatic neuromusculoskeletal and autonomic nervous systems interplay with biomechanics, fascia structures, endocrine and immune responses as well as emotional cognitive functions [13, 15, 16, 25, 31, 32, 36, 42].

Nonetheless, from a clinical viewpoint, the core tenets of SD remain rooted in nonstructural pathology characterised by tissue changes with movement asymmetry and restriction [14], perhaps with the addition of functional movement and psychosocial factors to render a more holistic and unifying clinical approach. The recent addition of pain physiology education as one of the therapeutic strategies goes in the right direction [16], thus placing SD beyond the physical sphere and encouraging clinicians to implement a multifaceted and individualised approach to treatment. However, a core component of manual medicine is the use of manual "hands-on" techniques. Pain physiology education is a useful additional tool, but does not mark a true strategic change to current diagnostic and therapeutic paradigms.

The recent introduction of fascia dysfunction as an important pillar of SD is interesting and merits consideration [31, 32, 36], but perhaps the fascia simply represents a microcosm of the multisystemic forces exerted on all neuromusculoskeletal tissues, from joints and tendons, to muscles, ligaments and fat pads. Hence, our better understanding of the biochemical, endocrinological, neuromusculoskeletal processes underlying fascia dysfunction in the context of SD does little to change the current manual medicine armamentarium, although it does question whether a more integrated clinical analysis of SD is necessary. Such a unifying component that adds to our definition of SD and facilitates a more comprehensive and integrated approach could be represented by functional movement. In fact, assessing how well coordinated and orchestrated movement is could provide diagnostic information to better guide therapeutic approaches, where manual therapies can be complemented by individualised exercise programmes, encompassing normal movement restoration, activity-specific exercises (e.g., sports), postural changes, ergonomics, breathing techniques and sleep training.

Although none of the new theories of SD provide sufficient evidence to change the current manual medicine approach, perhaps their real contribution to paradigm change may be to shift our focus away from defining the characteristics, diagnostic features and therapeutic targets of SDs, thus preventing a simplistic management of "issues in the tissues"

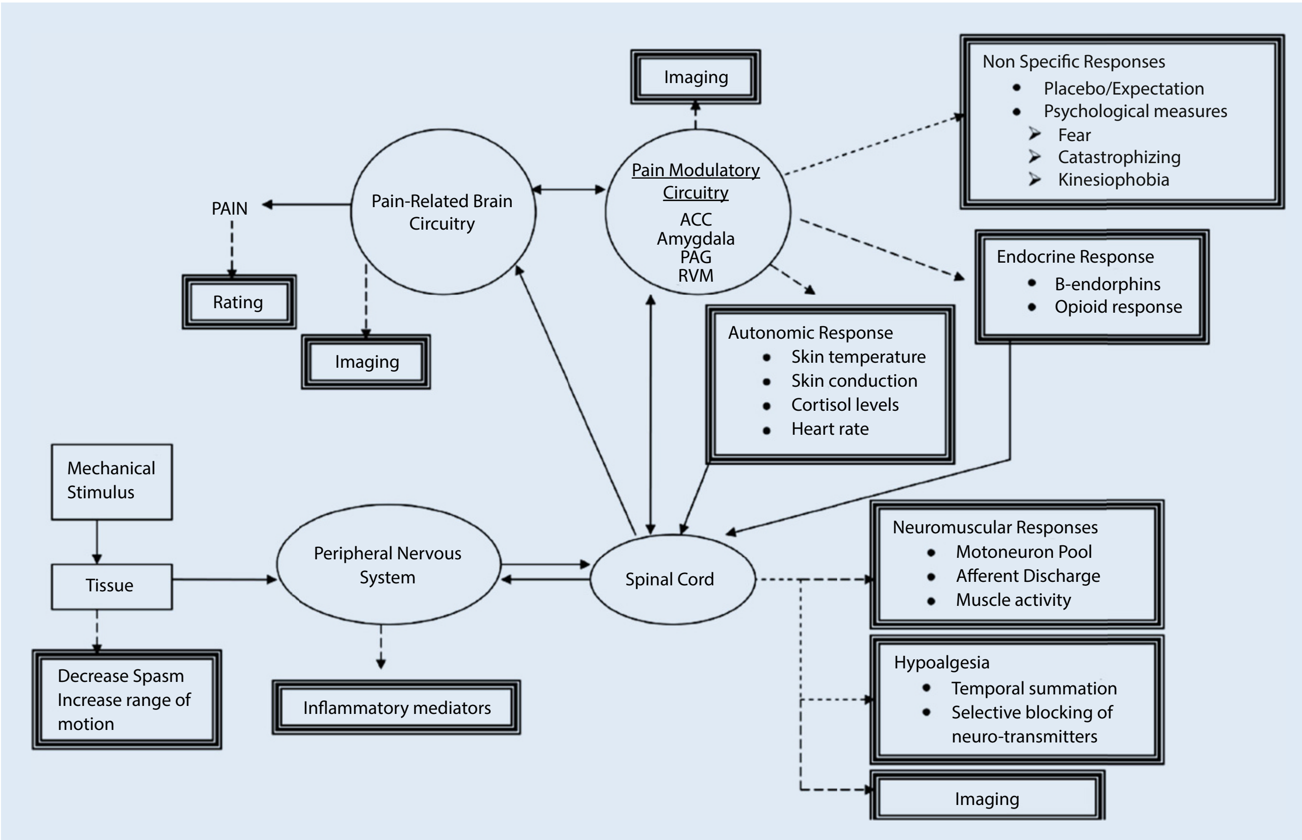

Fig. 12 ▲ Comprehensive model of the mechanisms of manual therapy. *ACC* anterior cingulate cortex, *PAG* periaqueductal gray, *RVM* rostral ventromedial medulla. (With kind permission from © Bialosky et al. 2009 [44])

and promoting treatment of the whole patient. Interestingly, progressing toward a holistic "whole-person" approach may place manual medicine on a converging path with the original Stillian tenets and conventional medicine, where holistic practices are increasingly encouraged to tackle the complex burden of chronic noncommunicable diseases.

Conclusion

Segmental dysfunction remains an integral component of manual medicine, guiding its concepts, diagnostic thinking and therapeutic approach. Scientific innovation and discovery have provided novel insights and a better understanding of the biological processes that define SD in the human body, often supporting the beneficial role of manual approaches. However, in order for manual treatment to remain effective and relevant, our present understanding and paradigms need to be constantly challenged. This could be a double-edged sword, as this evolution may validate and consolidate some current practices, extinguish others and promote new ones. Nonetheless, as history often repeats itself in cycles, the current drive toward holism may, for manual medicine, represent both a step forward and a return to its origins at the same time.

The significance of muscle tissue and fascia in manual medicine

A literature report

Basics

Manual medicine likely initiates biomechanical and neurophysiological mechanisms.

Manual medicine produces a mechanical force, which is necessary to initiate a chain of neurophysiological responses.

The neurophysiological changes achieved by manual medicine mechanisms originate from peripheral mechanisms, spinal cord mechanisms, and/or supraspinal mechanisms. Inflammatory mediators and peripheral nociceptors interact in response to injury and manual therapy may directly affect this process.

Applied manual medicine is associated with pain relief, afferent discharge, motoneuron pool activity and changes in muscle activity, all of which may indirectly implicate a spinal cord-mediated effect.

The suggested model is intended to be applicable to all forms of manual medicine. While the biomechanical application of joint-, soft tissue- and nerve-focused manual therapy differs, the related neurophysiological responses are similar and adequately encompassed within the model given.

The comprehensive model is intended to explain the mechanisms of manual therapy on musculoskeletal pain ([43]; ◘ Fig. 12).

Joints and discs

In patients with sciatica, centralisation is common. Centralisation might affect the symptoms of patients with sciatica, no matter what type of disc lesion is found on MRI and despite the severity of the symptoms (leg pain) and neurological findings [45].

Please see also the Basic Outlines in chapter B 1.

Muscles

Myofascial trigger points are important because they are common. Myofascial trigger points are the main source of pain in 30% of patients who enter a general practitioner's practice and in 85% of patients who enter a pain centre [46].

An active myofascial trigger point is usually associated with a painful, restricted range of motion. Trigger points can be identified by gentle palpation across the direction of the muscle fibres. Palpation of a myofascial trigger point is painful and reproduces the patient's local and referred pain pattern. The most important feature of the trigger point, besides the jump sign, is the palpable taut band [142].

Palpable taut band, spot tenderness, jump sign and referred pain due to palpation are the most reliable criteria for palpatory diagnosis of myofascial trigger points. Experience of the observer is essential for accuracy. There is moderate evidence for the reproducibility of myofascial trigger point palpation with experienced observers [47–50].

In several publications, Mense and colleagues discussed a slight "neurogenic" inflammation under the neuromuscular endplate region of a myofascial trigger point [139].

There is evidence that a myofascial trigger point might be the result of increased excitability of spinal neurons, which is responsible for the increased muscular tension in the taut band and myofascial trigger point [51].

A feed-forward contraction of the abdominal muscles could be shown in asymptomatic individuals, whereas the activation of the transverse abdominis is delayed in individuals with long-standing groin pain [52].

Back pain patients have also a poorer ability to voluntarily recruit the multifidus muscle in order to obtain physiological lordosis [53].

It has been shown that women with disorders of continence and respiration have a significantly higher prevalence of back pain than women who do not have these disorders [54].

A greater fatigability of the superficial cervical flexors was identified for neck pain patients by electromyography (EMG) with respect to healthy subjects. Furthermore, an aberrant activity of the sternocleidomastoid muscle and the anterior scalene occurs during functional activities [55, 56].

Female athletes who developed lower back pain (LBP), as compared with those without LBP, were found to have hip extensor strength that was significantly more asymmetric [57].

It could be demonstrated that synchronisation between motor units in the medial and lateral quadriceps muscles during an isometric knee extension task is reduced in people with anterior knee pain compared to pain-free individuals [58].

The findings have important implications for the understanding of the effects of pain on motor control, because they show that motor unit synchronisation is changed with pain [53, 54, 56–59].

There is strong evidence for the existence of three myofascial chains: the superficial back line, the back functional line and the front functional line. Moderate-to-strong evidence is available for parts of the spiral line and the lateral line [59].

Most skeletal muscles of the human body seem to be directly linked by connective tissue. There is evidence that tension can be transferred between at least some adjacent muscles. The possibility of load transfer between muscles encourages targeting entire myofascial chains in the evaluation process, treatment and exercise. Therefore, more holistic diagnostic and treatment approaches seem appropriate for overuse conditions or radiation pain symptoms that involve several structures of a myofascial chain [60].

A statistically significant decrease in pelvic floor descent and an increase in diaphragmatic excursion with manual external pelvic compression has been determined [61].

Fasciae

Palmar aponeurosis has the typical features of a proprioceptive organ. The fascial innervation seems to be important in the perception of pain [62].

Myofascial pain has a high prevalence among individuals with regional pain complaints. Palpation is the only method available for the clinical diagnosis of myofascial pain [48, 63]. It has been shown that a semi-electronic tissue compliance meter appears to determine the stiffness of biological tissues with a sound reliability and validity [64].

Ligament creep has a significant effect on neuromuscular functions. It is suggested that prolonged ligament tension subjects joints to an increased risk of instability and potential injury due to its own laxity and via increased agonist activity without compensatory antagonist coactivation [65].

The inflamed thoracolumbar fascia showed an increase of presumably nociceptive fibres in the rat, which may explain the pain from a pathologically altered fascia [66].

Innervation density was found to be three times higher in thoracolumbar fascia than in the corresponding muscle of mice, and almost as high as at the muscle–fascia interface, where mechanical loads can be concentrated. Data of studies in humans show that the thoracolumbar fascia is a densely innervated tissue with marked differences in distribution of the nerve endings over the fascial layers. These findings support the view that the thoracolumbar fascia is potentially a major input to musculoskeletal pain [67].

Thoracolumbar fascia shear strain has been shown to be reduced in subjects with LBP of greater than 12 months duration compared to a control group with no LBP [68].

Studies have shown a critical codependent mechanism between deep abdominal and lumbar spinal muscles linked to each other, especially through the posterior layer of the thoracolumbar fascia [69, 70].

Tissue specificity is important in the pain perception associated with delayed-onset muscle soreness. Fascial tissue may have an important role in delayed-onset muscle soreness perception [67].

Chronic pain

Generic prognostic factors for musculoskeletal pain with strong or moderate evidence include widespread pain, high functional disability, somatisation, high pain intensity and long pain duration as well as a high depression and/or anxiety score [71].

Reduced proprioception in the spine has been identified in subjects with back pain in relation to those free from back pain [72].

A clear difference has been demonstrated between patients with LBP and subjects without LBP regarding their ability to actively control the movements of their low back. There is also a significant difference related to pain duration. Patients with chronic LBP have significantly more positive tests than those with acute or subacute LBP [73].

It has been shown that in both subjects with acute and chronic LBP, there are elevated levels of proinflammatory and nociceptive chemokines [74].

In persons with chronic LBP, a decrease of paraspinal cutaneous temperature has been shown in comparison with subjects without chronic LBP [75].

It has been shown that fatigue can possibly have an effect on ankle kinematics and kinetics during a jump [74].

Isolated musculoskeletal chest wall pain can be due to dysfunctions of costovertebral joints or dysfunction of the sternum and muscles of the chest. Physiotherapy including manual medicine techniques is recommended in this case [76].

It has been shown that during simulated tennis play, patients with chronic tennis elbow employ an earlier, longer and greater activation of forearm extensors in their EMG pattern than healthy subjects. Such changes may be considered detrimental to the healing process [77].

Effects of manual therapy

In experiments in rats, it has been shown that interventions with manual therapy prevent functional declines, improve task performance, prevent behavioural changes indicative of discomfort and reduce neural inflammation, myelin degradation and extraneural fibrosis [78].

There is moderate evidence that neuromuscular manual treatment is capable of activating the central mechanisms responsible for pain control, modulation of autonomic functions and posture [79].

Treatment directed at cervical pain generators has produced significant headache relief in patients with headaches related to cervical spine pathology [80].

External pelvic compression (EPC) can be expected to decrease pain and improve static strength in some individuals with lumbopelvic disorders. EPC appears to improve pelvic stability, decrease pelvic laxity and sacral mobility, relieve pain, substitute for stabilising muscle activity and improve function (moderate evidence) [81].

It has been shown with moderate evidence that manipulation with HVLA influences various biochemical markers. After HVLA there is an increase in substance P, neurotensin, oxytocin and interleukin levels and there may be an influence on cortisol levels after HVLA. The use of HVLA may be considered a sound strategy to influence pain and inflammatory disorders [82, 83].

Mobilisation of the cervical and thoracic spine has an influence on sympathetic and parasympathetic nervous system reactions including heart rate variability, blood pressure and vasodilation. Chronic pain patients seem to have a higher sensibility to manual medicine techniques than control groups in terms of autonomous nervous system responses [75, 83, 84, 84–88, 88–92].

Neural mobilisation produces hypoalgesic effects (pressure pain threshold) [93].

Manual therapy applied with almost the same load produces plastic deformation for fasciae (plantar fascia, fascia lata) [94].

Manual therapy theoretically restores mobility by reoptimising the distribution of lines of force within fascia [95].

In vitro experiments could demonstrate that isometric strain induces an increase in fascial stiffness. An association between strain hardening and loss of tissue water could also be validated in vitro [96].

Experiments in vitro could show that interstitial flow alone may be sufficient to induce and sustain fibrosis, even in the absence of transforming growth factor alpha (TGF)-alpha secretion by other cells such as inflammatory, epithelial or tumour cells, and correlates with key features of the progression of an inflammatory state to a fibrotic pathology. Findings in studies suggest that interstitial flow may help to modulate fibroblast phenotypes and drive the progression of fibrotic diseases [97].

Manual therapy may improve fascia sliding by generating fluid pressure [98].

Stretching of connective tissue seems to decrease acute inflammation (in vivo), reduced neutrophil migration (ex vivo) and increased connective tissue pro-resolving mediators (in vivo and ex vivo), and the similar effects of active and passive stretching suggest a mechanical effect on the tissues [99].

Experiments in dogs could demonstrate reflex responses of the cardiac vagus nerve evoked by excitation of group II (A beta) and III (A delta) but not group I (A alpha) somatic nerves also applied to the somatic sympathetic reflex recorded simultaneously. The general pattern was excitation followed by long-lasting inhibition of tonic activity. Reflex excitation was also found to be produced in the vagal nerve in response to splanchnic nerve stimulation [75].

There is evidence that fascial techniques increase parasympathetic nervous system activity [86, 91, 100].

In rat experiments, a new form of long-term depression of excitatory synaptic transmission in substantia gelatinosa neurons from the rat spinal dorsal horn might have been demonstrated that can be induced by conditioning low-frequency stimulation of primary afferent A-delta fibres. Possibly, this long-term depression may be involved in long-lasting therapeutic effects of counterstimulation [89].

Fascial manipulation seems to have a preventive effect for sport injuries in individuals with chronic ankle instability with regard to improving ROM and symptoms [101].

It has been shown that there is evidence that fascial manipulation in postsurgical care after hip surgery (total hip arthroplasty) improves the functional outcome significantly [99].

Adhesions result from peritoneal trauma and aberrant wound healing processes and can therefore develop after any intraabdominal operation. Intraabdominal adhesions occur in 50 to 100% of patients with previous surgery. Particularly in patients with previous surgery, adhesion-related complications can occur at any time [102]. In rat experiments it has been shown that manually assisting free movement of the bowel following injury results in fewer adhesions. It seems to be possible to manually lyse postsurgical adhesions in a rat model [44, 102].

Examination

In the diagnosis and management of musculoskeletal pain there is a selection of special tests in physical examination which appear to be useful for screening red flags. There are individual items of clinical examination which have been found to be fairly reliable and several exist with an acceptable level of accuracy for determining red flags such as cervical or lumbar radiculopathy. A selection of these clinical tests uses a neural impairment reference criterion standard [103, 104].

Examination by spinal palpation shows acceptable results in tests for palpation of pain. The interobserver reliability is acceptable for palpation of osseous structures and soft tissue pain. Regional ROM tests are more reliable than segmental ROM tests. Paraspinal soft tissue palpatory tests have a low interobserver reliability in all regions of the spine [105].

Clinicians should include assessments of impairments of body function that can establish baselines, monitor changes over time and be helpful in clinical decision-making to differentiate several types of pain in the musculoskeletal system in the physical examination of patients with acute or chronic musculoskeletal pain [106].

There is significant international variation in the physical examinations and impairment scores in subjects with chronic pain [104, 107–111].

The minimal important change (MIC) values depend not only on empirical evidence but also on clinical interpretation and judgement [112–114].

Manual palpation of the fascia tissue represents a cost-neutral and widely used screening method aimed at assessing viscoelastic properties (e.g., stiffness). Studies show a limited reliability [79, 96].

Cervical spine

In their physical examination measures, physicians should include a cervical active ROM as well as examination of cervical and thoracic segmental mobility and segmental provocation signs for neck pain with mobility deficits and neck pain and the associated International Classification of Diseases (ICD) categories of cervicalgia and/or pain in the thoracic spine. It is recommended to include a cranial cervical flexion test and a deep neck flexor endurance test into the physical examination of patients with neck pain and movement coordination impairments and the associated ICD category of sprain and strain of the cervical spine. A combination of upper limb tension tests (shoulder abduction test), Spurling's test and the distraction test are proposed for patients with neck pain and radiating pain to increase the likelihood of a cervical radiculopathy [104, 106, 108–111, 115–121].

Headaches related to nocigenerators in the upper cervical region exist. This is an important clinical entity. There are several nocigenerators in the spine, like the zygapophysial joints, intervertebral discs and myofascial trigger points. Diagnostic criteria for cervicogenic headache include unilaterality of pain; restriction in ROM of the neck; provocation of usual/regular head pain by neck movement or sustained awkward positions; provocation of usual/regular head pain with external pressure over the upper cervical or occipital region on the symptomatic side; ipsilateral neck, shoulder or arm pain; deviating muscle extensibility (incidence of muscle tightness) of neck or shoulder muscles, such as the upper trapezius, scalenes, levator scapulae, short cervical extensors, and pectoralis major and minor. The presence of painful SD in the upper three cervical joints as detected by manual examination most clearly identifies the cervicogenic headache subjects. There is an overlap in clinical features between cervicogenic headaches and migraines. Several studies have suggested that it is possible to discriminate between these headache types. Clinical studies have shown that pain from cervical spine structures can be referred to the head [73, 117, 118, 122].

Interobserver reliability values of the modified cervical nonorganic signs and modified physical dysfunction severity were both deemed acceptable based on observed intraclass correlation (ICC) values in a current study of patients with chronic neck pain [111].

Among spinal palpatory procedures used in the evaluation and management of back and neck pain, pain-provocation tests are the most reliable and soft tissue paraspinal palpatory diagnostic tests are the least reliable. Regional ROM tests are more reliable than segmental ROM tests, and intraobserver reliability is better than interobserver reliability. (The significance of observer experience is mentioned in Muscle Studies described in Sect. 5.3) [107].

Screening for upper cervical instability cannot be done accurately by physical examination tests at the moment [104].

Maximum neck flexor strength—peak force, peak force/body weight and average force seem to be significantly lower in patients with neck pain compared to controls [82].

Lumbar spine

Functional examination of the lumbar spine showed moderate interobserver reliability [55, 123].

The diagnosis of lumbosacral radiculopathy should always be arrived at through consolidation of sensory, motor and deep tendon reflex test results and not based on isolated test results [65].

No "gold standard" has been defined for motor control tests of the low back to date [66, 111].

Manual muscle testing, sensory testing, supine straight leg raise, Lasegue sign and crossed Lasegue sign are recommended for use in diagnosing lumbar disc herniation with radiculopathy. The supine straight leg raise, compared with the seated straight leg raise, is suggested for use in diagnosing lumbar disc herniation with radiculopathy [124].

There is no complete recovery of postural control demonstrated in the long term following lumbar discectomy [125].

There is insufficient evidence to date for certain physical findings to diagnose degenerative lumbar spinal stenosis [126, 127].

Growing evidence supports the use of manual physical therapy combined with exercise and aerobic training as a safe and effective intervention for patients with lumbar spinal stenosis [128].

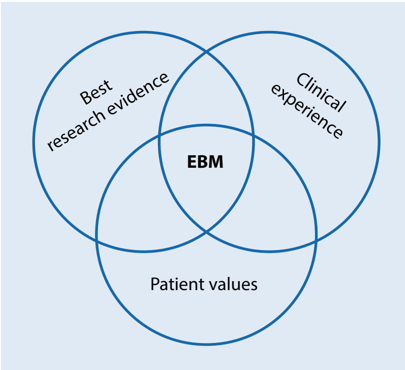

Fig. 13 ▲ Evidence-based medicine (*EBM*). (Modified from Haneline [144])

There is inconsistent evidence regarding the development of recommendations on MIC for commonly used measures of pain and function in (acute or chronic) LBP [116].

There is no "gold standard" for the diagnosis of axial spondyloarthritis; however, the current criteria do not include a physical examination of the lumbar spine [129–131].

Pelvis

Substantial agreement between two observers for the classification of nonspecific lumbopelvic pain into lumbar pain and pelvic girdle pain in pregnant women has been seen [111].

Evidence for the accuracy of provocative manoeuvres in diagnosing sacroiliac joint (SIJ) pain is limited. A combination of manual tests for examining the SIJ seems to be useful [132, 133].

Additionally, there is a recommendation to palpate the symphysis in the examination of the pelvis [61].

Extremities

Upper limb neurodynamic tests are plausible for detecting peripheral neuropathic pain. A positive upper limb neurodynamic test should at least partially reproduce the patient's symptoms and structural differentiation should change these symptoms [134].

The reliability of the examination of larger extremity joints (shoulders, hips, knees and elbows) fares slightly better than examining smaller extremity joints (wrists, ankles, fingers and toes) [135, 136].

Physical examinations of the cervicothoracic junction and the peripheral joints and muscles of the upper extremity are required in the thoracic outlet syndrome in addition to a required neurological examination, peripheral nerval entrapment test, and several tests of the cervical spine [137, 138].

Evidence in manual medicine

General considerations

Evidence-based manual medicine (EBM) is not different from evidence-based medicine in other medical specialties.

"Evidence based medicine is the conscientious, explicit, judicious and reasonable use of modern, best evidence in making decisions about the care of individual patients. EBM integrates clinical experience and patient values with the best available research information. […] The practice of evidence-based medicine is a process of lifelong, self-directed, problem-based learning in which caring for one's own patients creates the need for clinically important information about diagnosis, prognosis, therapy and other clinical and health care issues (◘ **Fig. 13**). It is not a 'cookbook' with recipes, but its good application brings cost-effective and better health care. The key difference between evidence-based medicine and traditional medicine is not that EBM considers the evidence while the latter does not. Both take evidence into account; however, EBM demands better evidence than has traditionally been used" ([143]).

The complexity of EBM described above is reflected in the development of European manual medicine since the middle of the 20th century.

During this period, a complex system of national and transnational scientific societies in Europe has developed from the activities of individual chirotherapists/manual therapists and individual "schools" of small groups of doctors, which have ensured that the criteria of EBM in the clinic, teaching and research are met.

The ESSOMM European Core Curriculum and Principles of Manual Medicine summarises the entire results of this development.

With these guidelines and principles agreed in consensus by the members of ESSOMM, an agreement which is based on the entire knowledge and fundamentals of modern medicine, an evidence-based teaching system of manual medicine has been inaugurated.

In regular meetings of the manual medicine societies, academies, teachers and expert commissions, opinions and convictions from clinical experience are agreed upon and published in relevant international journals. This corresponds to level IV of the evidence classes according to the recommendations of the Agency for Healthcare Research and Quality (AHRQ). A higher level of evidence is dependent on methodologically high-quality nonexperimental studies such as comparative studies, correlation studies or case–control studies (level III) and high-quality studies without randomisation (level IIb) as well as sufficiently large, methodologically high-quality randomised controlled studies (RCT; level 1b).

EBM is not limited to randomised controlled trials (RCTs) and meta-analyses. Nevertheless, these are to be regarded as the gold standard in the great majority of those questions that are about evaluating the benefits and risks of therapies.

A prerequisite for evidence-based diagnostics in manual medicine is good reproducibility, validity, sensitivity and specificity studies of the diagnostic procedures. To ensure the quality of such studies, the International Academy for Manual Musculoskeletal Medicine has developed a "reproducibility protocol for diagnostic procedures in MM" in recent years. "The protocol can be used as a kind of 'Cook Book Format' to perform reproducibility studies with kappa statistics. It makes it feasible to perform reproducibility studies in MM Medicine clinics and by Educational Boards of the MM Societies" [145].

On behalf of the ESSOMM, the Research Advisory Center of the GSMM in 2019 carried out a literature search on current study results (2009–2019) on diagnostics and therapy in manual medicine. Items for search: ("Manual Medicine" OR "Manual Therapy") AND (functional OR musculoskeletal OR disorder). The search identified 1499 unique citations limited to humans. After screening titles and abstracts, 482 full-text manuscripts were retrieved for further assessment, 216 of which were systematic reviews. The individual publications were subdivided by hand according to their target content: diagnostics ($n=85$), specific therapy ($n=119$), basics and safety ($n=39$).

Based on the available scientific materials, the author believes to be able to conclude that a general EBM level III is available, with individual studies reaching level II or Ib, which fulfils the prerequisite and creates the ability to perform tasks to a satisfactory or expected verification (validity) of manual medicine diagnostic and therapeutic techniques.

Two studies are mentioned here as examples of good-quality studies (level Ib): the first focused on functional disorders and pain in the lower spine, the second on functional disorders of the head joints in babies.

The first is "demonstrating a clear difference between patients with LBP and subjects without back pain regarding their ability (in 5 of 6 tests) to actively control the movements of the low back" [141]. The second used a setting with 202 infants aged 14–24 weeks with postural and movement findings examined in four study centres using a standardised four-item symmetry score. Result: the single manual medicine treatment significantly improved postural and motor asymmetries in infants with articular and segmental dysfunctions causing tonic asymmetric positions [146].

A large systematic review searching prognostic factors for musculoskeletal pain (MSK) in primary care "involves more than 48,000 participants with 18 different outcome domains. 51 studies were on spinal pain/back pain/low back pain, 12 on neck/shoulder/arm pain, 3 on knee pain, 3 on hip pain and 9 on multisite pain/widespread pain—total quality scores ranged from 5 to 14 (mean 11) and 65 studies (83%) scored 9 or more—provides new evidence for generic prognostic factors for MSK conditions in primary care. Such factors include pain intensity, widespread pain, high functional disability, somatization and movement restriction. This information can be used to screen and select patients for targeted treatment in clinical research as well as to inform the management of MSK conditions in primary care" [140].

Recently, an ESSOMM literature search found 24 relevant systematic reviews or meta-analyses related to manual therapy. The individual reviews are aimed at different therapeutic goals for different complaints in different parts of the body. The search results have not yet been evaluated coherently.

An update of the Bone and Joint Decade Task Force on Neck Pain and its Associated Disorders by the OPTIMa collaboration [147] concluded: "Our review adds new evidence to the Neck Pain Task Force and suggests that mobilization, manipulation (HVLA), and clinical massage are effective interventions for the management of neck pain. It also suggests that electroacupuncture, strain–counter strain, relaxation massage, and some passive physical modalities (heat, cold, diathermy, hydrotherapy, and ultrasound) are not effective and should not be used to manage neck pain."

The double-blinded randomised controlled Spinal High-Velocity Low-Amplitude Manipulation in Acute Nonspecific Low Back Pain [148] trial, in which 47 subjects received spinal manipulation, showed in a subgroup of patients with acute nonspecific LBP that "spinal manipulation was significantly better than nonsteroidal anti-inflammatory drug diclofenac and clinically superior to placebo".

Other reviews are dedicated to:

- Low back pain [12, 149, 150]
- Neck pain and/or low back pain [151, 152]
- Upper limb pain [153]
- Knee pain [154, 155]
- Function of the temporomandibular joint [156, 157]

Practically all studies have limiting factors that limit their informative value. For example, one of the limiting factors is that there is often no distinction between manipulation and mobilisation.

The results of this systematic review showed

- that spinal manipulation and mobilisation, acupuncture and massage treatments were significantly more efficacious for neck/LBP than no treatment, placebo, physical therapy or usual care in reducing pain;
- that spinal HVLA procedures are cost-effective treatments to manage spinal pain when used alone or in combination with GP care or advice and exercise compared to GP care alone, exercise or any combination of these;
- that spinal HVLA procedures have a statistically significant association with improvements in function and pain improvement in patients with acute LBP;
- preliminary evidence that subgroup-specific manual therapy may produce a greater reduction in pain and increase in activity in people with LBP when compared with other treatments. Individual trials with low risk of bias found large and significant effect sizes in favour of specific manual therapy;
- that upper cervical manipulation or mobilisation and protocols of mixed manual therapy techniques presented the strongest evidence for symptom control and improvement of maximum mouth opening;
- that musculoskeletal manipulation approaches are effective for the treatment of temporomandibular joint disorders—here is a larger effect for musculoskeletal manual approaches/manipulations compared to other conservative treatments for temporomandibular joint disorder;
- that the results of the available reviews and the evidence found on the effect of manual medical treatment form the basis for the inclusion of manual therapy in guidelines for the treatment of acute and chronic pain in the musculoskeletal system, especially in the spine, joints and muscles.

All reviews mentioned call for further qualitative studies in order to further consolidate and increase the level of evidence.

The previous initial shortcomings of the studies must be overcome:

- Clear elaboration of the question;
- Exact description of manual medical practice;
- Lowering the bias in patient inclusion.

Conclusion

The EBM-oriented doctors in the manual medicine of tomorrow have three tasks:

1. To use evidence summaries in clinical practice.
2. To help develop and update selected systematic reviews or evidence-based guidelines in their area of expertise.
3. To enrol patients in studies of treatment, diagnosis and prognosis on which medical practice is based [151].

Safety in manual medicine

A review of the existing literature. Adapted from the FIMM guidelines for basic training and safety [158].

The subject safety of spinal manipulations has been extensively discussed in many publications [159–196].

Risks of cervical spine high-velocity thrust therapy

General considerations from the literature. The degree of serious risks associated with manipulation of the cervical spine is uncertain, with widely differing results being published [197].

A 1996 Danish chiropractic study confirmed the risk of stroke to be low, and determined that the greatest risk is with manipulation of the first two vertebra of the cervical spine, particularly passive rotation of the neck [198].

Serious complications after manipulation of the cervical spine are estimated to be 1 in 4 million manipulations or fewer [199]. A Rand Corporation extensive review estimated "one in a million" [200]. Dvořák, in a survey of 203 practitioners of manual medicine in Switzerland, found a rate of one serious complication per 400,000 cervical manipulations, without any reported deaths, among an estimated 1.5 million cervical manipulations [201].

Jaskoviak reported approximately 5 million cervical manipulations from 1965 to 1980 at the National College of Chiropractic Clinic in Chicago, without a single case of vertebral artery stroke or serious injury [202]. Henderson and Cassidy performed a survey at the Canadian Memorial Chiropractic College outpatient clinic, where more than half a million treatments were given over a 9-year period, again without serious incident [203]. Eder offered a report of 168,000 cervical manipulations over a 28-year period, again without a single significant complication [204]. After an extensive literature review performed to formulate practice guidelines, the authors concurred, "the risk of serious neurological complications (from cervical manual technique) is extremely low and is approximately one or two per million cervical manipulations" [205].

Understandably, vascular accidents are responsible for the major criticism of spinal manipulative therapy. However, it has been pointed out that "critics of manipulative therapy emphasize the possibility of serious injury, especially at the brain stem, due to arterial trauma after cervical manipulation. It has required only the very rare reporting of these accidents to malign a therapeutic procedure that, in experienced hands, gives beneficial results with few adverse side effects" [206]. In very rare instances, the manipulative adjustment to the cervical spine of a vulnerable patient becomes the final intrusive act, which results in a very serious consequence [190, 207–209].

According to an expert opinion, HVLA manipulation of the cervical spine is estimated to have no effectiveness and to be dangerous [210], while this has not been confirmed by others. As it has been revealed, this expert opinion does not fulfil the criteria of evidence level III [211].

In a 2007 follow-up report in the Journal of the Royal Society of Medicine, Ernst concluded: "Spinal manipulation, particularly when performed on the upper spine, is frequently associated with mild to moderate adverse effects. It can also result in serious complications such as vertebral artery dissection followed by stroke. Currently, the incidence of such events is not known. In the interest of patient safety we should reconsider our policy towards the routine use of spinal manipulation" [212].

In 2007, the French Medical Society for Manual Medicine and Osteopathy SOFMMOO recommended in a scientific article based on a literature search that for the cervical spine, despite a lack of data in the literature, prudence and medicolegal issues justify the performance of systematic radiography prior to cervical spine manipulation therapy and generally in case of back or neck pain in patients of less than 25 years of age [213].

A paper by Michell et al. published in 2004 reported on an investigation on the effects of cervical spine rotation on vertebral artery blood flow [214]. The question was whether cervical spine rotation, as used in the standard vertebrobasilar insufficiency test, is associated with a measurable change in intracranial vertebral artery blood flow. Transcranial Doppler sonography was used to measure intracranial vertebral artery blood flow in 30 young, healthy, female subjects, with the cervical spine in the neutral position and with sustained, end-of-range rotation. Statistically significant decreases in blood flow were demonstrated, with contralateral rotation particularly, in the left and right vertebral arteries. Despite this change in blood flow, signs and symptoms of vertebrobasilar insufficiency were not demonstrated in these subjects. The author concluded that the use of the vertebrobasilar insufficiency test, in the absence of a more specific, sensitive and valid test, should be recommended to assess the adequacy of hindbrain blood supply to identify those patients who may be at risk of serious complications post-manipulation.

On the other hand, in 1997, a Canadian research group was unable to demonstrate that the extension–rotation test is a valid clinical screening procedure to detect decreased blood flow in the vertebral artery [215]. They concluded that the value of this test for screening patients at risk of stroke after cervical manipulation is questionable. They tested 12 subjects with dizziness reproduced by the extension–rotation test and 30 healthy control subjects using Doppler ultrasonography examination of their vertebral arteries with the neck extended and rotated.

Yet maximal rotation of the cervical spine may significantly affect vertebral artery blood flow, particularly when used in the treatment of patients with underlying vascular pathology. In 2003, Mitchell [216] investigated intracranial vertebral artery blood flow in normal male and female subjects, aged 20 to 30 years, in neutral and maximally rotated cervical spinal positions using transcranial Doppler sonography. The sample consisted of 60 male subjects and 60 female subjects (240 vertebral arteries). He found a significant decrease ($P = 0.001$) in intracranial vertebral artery blood flow following cervical spine rotation, irrespective of side but greater on the contralateral side, in the total sample and in male subjects. Female subjects had a significantly higher blood flow than male subjects.

In 1998, Licht et al. presented a randomised, controlled and observer-blinded study comparing flow velocity in the vertebral artery before and after spinal manipulative therapy using Doppler ultrasound technology [217]. Investigated were 20 Danish university students with a "biomechanical dysfunction" in the cervical spine. The research group found no significant changes in these subjects. They concluded that major changes in peak flow velocity might in theory explain the pathophysiology of cerebrovascular accidents after spinal manipulative therapy. However, in uncomplicated spinal manipulative therapy, this potential risk factor was not prevalent.

Vertebrobasilar accidents and cervical spine high-velocity thrust therapy. In 2002, Haldeman and collaborators reported in an extensive paper on vertebrobasilar accidents in relation to cervical spine manual therapy [11, 218]. According to these authors, stroke represents an infrequent adverse reaction associated with cervical spine manual therapy. Attempts to identify the patients at risk and the type of manual technique most likely to result in these complications of manual therapy have not been successful. A retrospective review of 64 medical legal cases of stroke temporally associated with cervical manual therapy of the spine was performed to evaluate characteristics of the treatment rendered and the presenting complaints in patients reporting these complications. These files included records from the practitioner who administered the manual therapy, post-stroke testing and treatment records usually by a neurologist, and depositions of the patient and the practitioner of manual techniques as well as experts and treating physicians. A retrospective review of the files was carried out by three (2 in 11 cases) researchers, using the same data abstraction instrument, to independently assess each case. These independent reviews were followed by a consensus review, in which all reviewers reached agreement on file content. Whereas 92% of cases presented with a history of head and/or neck pain, 16 (25%) cases presented with sudden onset of new and unusual headache and neck pain often associated with other neurological symptoms that may represent a dissection in progress. The strokes occurred at any point during the course of treatment. Certain patients reported onset of symptoms immediately after the first treatment, while in others the dissection occurred after multiple manual treatments. There was no apparent dose–response relationship to these complications. These strokes were noted following any form of standard cervical manipulation technique including rotation, extension, lateral flexion and non-force and manual techniques in neutral position. The results of this study suggest that stroke, particularly vertebrobasilar dissection, should be considered a random and unpredictable complication of any neck movement including cervical manipulation. They may occur at any point in the course of treatment with virtually any method of cervical manual technique. The sudden onset of acute and unusual neck and/or head pain may represent a dissection in progress and be the reason a patient seeks manual therapy that then serves as the final insult to the vessel leading to ischemia.

Finally, the authors conclude that the literature does not assist in the identification of the offending mechanical trauma, neck movement or type of manual therapy precipitating vertebrobasilar artery dissection or the identification of the patient at risk. Thus, given the current status of the literature, it is impossible to advise patients or physicians about how to avoid vertebrobasilar artery dissection when considering cervical manual therapy or about specific sports or exercises that result in neck movement or trauma.

In another paper, after analysing 64 cases of cerebrovascular ischemia after manual therapy, Haldeman et al. stated that cerebrovascular accidents after such therapy appear to be unpredictable and should be considered an inherent, idiosyncratic and rare complication of this treatment approach. It seems not to be possible to identify factors from the clinical history and physical examination of the patient that would assist a physician in attempting to isolate the patient at risk of cerebral ischemia after cervical manual therapy [216].

Again, Haldeman and collaborators studied clinical perceptions of the risk of vertebral artery dissection after manual therapy of the cervical spine in 2003 [219]. The purpose of the study was to assess the effect of referral bias on the differences in perceived incidence of vertebral artery dissection after manual cervical therapy between neurologists and chiropractors in Canada. In a retrospective review, cases where neurological symptoms consistent with cerebrovascular ischemia were reported by chiropractors in Canada for the 10-year period 1988 to 1997 were included: there were 23 cases of vertebral artery dissection after cervical manipulation reported. Based on the survey, an estimated 134,466,765 manual treatments of the cervical spine were performed during this 10-year period. This gave a calculated rate of vertebral artery dissection after manual treatment of the cervical spine of 1:5,846,381 manual cervical spine treatments. Based on the number of practicing chiropractors and neurologists during the period of this study, 1 in every 48 chiropractors and 1 in every 2 neurologists would have been made aware of a vascular complication from manual treatment of the cervical spine that was reported during their practice lifetime.

In 2004, the Cochrane Collaboration stated that mobilisation and/or manipulation, when used with exercise, are beneficial for persistent mechanical neck disorders with or without headache. Done alone, manipulation and/or mobilisation was not beneficial; when compared to one another, neither was superior [220].

The quite extensive 2005 guidelines of the Canadian chiropractic profession stated on the basis of a broad analysis of the current evidence that none of the predisposing factors hypothesised in the literature definitively predict a dissection-related "cerebrovascular ischemic event" and, therefore, none is a contraindication to manipulation [221].

Also in 2005, Haneline and Lewkovich analysed the aetiology of cervical artery dissections in the years 1994–2003 [222]. They conducted a literature search of the

MEDLINE database for English-language articles published using the search terms cervical artery dissection (CAD), vertebral artery dissection and internal carotid artery dissection. Articles were selected for inclusion only if they incorporated a minimum of five case reports of CAD and contained sufficient information to ascertain a plausible aetiology. In total, 1014 citations were identified, 20 met the selection criteria. There were 606 CAD cases reported in these studies; 321 (54%) were internal carotid artery dissection and 253 (46%) were vertebral artery dissection, not including cases with both; 371 (61%) were classified as spontaneous, 178 (30%) were associated with trauma/trivial trauma, and 53 (9%) were associated with cervical spinal manipulation. If one apparently biased study was dropped from the data pool, the percentage of CADs related to cervical spinal manipulation dropped to approximately 6%. The authors concluded that this aetiologic breakdown of CAD did not differ significantly from what has been portrayed by most other authors.

In a paper presented in 2007, Smith and collaborators demonstrated that cervical spinal manipulation therapy is an independent risk factor for vertebral artery dissection [223]. The data were previously presented in 2003 [224]. They concluded that their case-controlled study of the influence of cervical spine manipulation therapy and cervical arterial dissection shows that this therapy is independently associated with vertebral arterial dissection, even after controlling for neck pain. Patients undergoing cervical spine manipulation therapy should be consented for the risk of stroke or vascular injury from the procedure. A significant increase in neck pain following cervical spine manipulation therapy warrants immediate medical evaluation.

In 2008, Cassidy et al. investigated a 10-year period with 818 stroke cases due to vertebrobasilar artery lesion, hospitalised in a population of more than 100 million person-years; 75% were treated by chiropractors, 25% by general practitioners. They concluded that stroke due to the vertebrobasilar artery is a very rare event in the population. The increased risk of vertebrobasilar artery stroke associated with chiropractic and general practitioner visits is likely due to patients with headache and neck pain from vertebrobasilar artery dissection seeking care before their stroke. There is no evidence of an excess risk of vertebrobasilar artery stroke associated with chiropractic care compared to primary care [171].

In 2009, Dittrich and collaborators compared 47 consecutive patients with cervical artery dissection with 47 consecutive patients of similar age with ischemic stroke due to aetiologies other than cervical artery dissection [225]. They found no association between any single one of the above risk factors and cervical artery dissection. Recent infections were more frequent in the cervical artery dissection group but failed to reach significance. However, the cumulative analysis of all mechanical trigger factors revealed a significant association of mechanical risk factors as a whole in cervical artery dissection. The authors concluded that mild mechanical stress, including manual treatment of the cervical spine, plays a role as possible trigger factor in the pathogenesis of cervical artery dissection. Cervical spine manipulation therapy and recent infections alone, however, failed to reach significance in the investigation.

Marx and collaborators evaluated in 2009 all cases with the diagnosis of cervical artery dissection submitted between 1996 and 2005 to the *Schlichtungsstelle für Arzthaftpflichtfragen der Norddeutschen Ärztekammer* for assessment of the accusations brought against the therapists who conducted the cervical spine manipulation therapy [188]. In neither in the 7 carotid nor in the 9 vertebral artery cases could a causal link be made between the dissection and the manipulation. However, in 5 of the 7 carotid and 7 of the 9 vertebral artery dissections was there clear evidence or a high probability that the dissection was present prior to the manual therapy and had caused neck pain, SD and, in some cases, even neurological symptoms. In no case were high-velocity thrust techniques the unique cause of such a negative event. Stroke after manual therapy of the cervical spine was mostly due to embolisation of thrombotic material from the dissected artery. As cervical arterial dissection and cervical spine disorder usually cause similar signs and symptoms, physicians must differentiate between these two entities prior to any manual treatment of the spine.

In 2010, the relationship between vertebrobasilar dissection stroke (VADS) and cervical manipulative therapy (CMT) was checked from all data available at the time. According to current data, the relationship between vertebrobasilar artery dissection stroke and manipulation of the cervical spine is not causal, but patients with VADS often have initial symptoms which cause them to seek care from a chiropractic physician and have a stroke sometime after, independent of the chiropractic visit. This new understanding has shifted the focus for the chiropractic physician from one of attempting to "screen" for "risk of complication to manipulation" to one of recognising the patient who may be having VADS so that early diagnosis and intervention can be pursued [226].

Finally, a prospective national survey in the UK to estimate the risk of serious and relatively minor adverse events following cervical spine manipulation therapy conducted by Haymo and collaborators [227] in 2007 dealt with data obtained from 28,807 treatment consultations and 50,276 cervical spine manipulations. There were no reports of serious adverse events. This translates to an estimated risk of a serious adverse event of, at worst, approximately 1 per 10,000 treatment consultations immediately after cervical spine manipulation therapy, approximately 2 per 10,000 treatment consultations up to 7 days after treatment and approximately 6 per 100,000 cervical spine manipulations. Minor side effects with a possible neurologic involvement were more common. The highest risk immediately after treatment was fainting/dizziness/light-headedness in, at worst, approximately 16 per 1000 treatment consultations. Up to 7 days after treatment, these risks were headache, in at worst approximately 4 per 100, numbness/tingling in upper limbs, in at worst approximately 15 per 1000, and fainting/dizziness/light-headedness, in at worst approximately 13 per 1000 treatment consultations. The study group concluded, consistent with an Italian group [228], that although minor side effects following cervical spine manipulation treatment were relatively common, the risk of a serious adverse event, immediately or up to 7 days after treatment, was low to very low.

By conclusion and in agreement with the Bone and Joint Decade 2000–2010 Task Force on Neck Pain and its Associated Disorders, the best available evidence suggests initial assessment for neck pain should focus on triage and those with common neck pain might be offered primarily noninvasive treatments if short-term relief is desired before the evaluation of cervical spine manipulation therapy [229].

The effectiveness of thrust manipulation for neck pain has been examined in many high-quality systematic reviews as well as in evidence-based clinical guidelines and health technology assessment reports. When combined with recent randomised trial results, this evidence supports including manipulation as a treatment option for neck pain, along with other interventions such as advice to stay active and exercises. However, when risk, benefit and patient preference are considered, there is currently no preferred first-line therapy, and no evidence that mobilisation is safer or more effective than manipulation [230].

Risks of lumbar spine manipulation therapy

In a 1993 study J.D. Cassidy and coworkers concluded that the treatment of lumbar intervertebral disk herniation by side posture manipulation is "both safe and effective" [231].

Oliphant graded prospective and retrospective studies and review papers according to quality in 2004, and results and conclusions were tabulated [232]. From the data published, an estimate of the risk of lumbar spine manipulation therapy causing a clinically worsened disk herniation or cauda equina syndrome in patients presenting with lumbar disk herniation was calculated. This was compared with estimates of the safety of NSAIDs and surgery in the treatment of lumbar disk herniation. As a result, an estimate of the risk of lumbar spine manipulation therapy causing a clinically worsened disk herniation or cauda equina syndrome in a patient presenting with lumbar disk herniation was calculated from published data to be less than 1 in 3.7 million. The author concluded the apparent safety of spinal manipulation, especially when compared with other medically accepted treatments for lumbar disk herniation, should stimulate its use in the conservative treatment plan of lumbar disk herniation.

In 2005, Oppenheim and collaborators reviewed medical records and radiographic studies of appropriate subjects to better clarify the spectrum of nonvascular complications following lumbar spine manipulation therapy, and to help define the risks of lumbar spine manipulation therapy. In total, 18 patients were identified who had received lumbar spine manipulation therapy and whose neurological condition had immediately worsened. Injuries were sustained to the cervical, thoracic and lumbar spine, and resulted variously in myelopathy, paraparesis, cauda equina syndrome and radiculopathy; 89% required surgery. Outcome was excellent in 50% and good in 37.5%. Three patients died from unrecognised malignancies. The authors concluded that spinal manipulation can be associated with significant complications, often requiring surgical intervention. Pretreatment scanning may help identify patients with significant risk factors, such as substantial disc herniation or occult malignancies. Prompt evaluation and intervention is necessary when symptoms worsen or neurological deficits develop [233].

Dvořák and collaborators published a survey among members of the Swiss Medical Association for Manual Medicine in 1999. Based on this survey, LBP problems are approached by means of manual therapy on average 805 times per year and physician. On average, each case with LBP is treated 1.4 times by a general practitioner with experience in manual medicine, while specialists dealing with more complex cases treat on average 4 to 5 times. Based on the survey, side effects and complications due to lumbar spine manipulation therapy are extremely rare [234].

In 1993, Dvořák's research group published from an earlier survey undertaken 1989. Informative data were given by 425 respondents on the frequency of complications of spinal manipulation therapy. The number of thoracolumbar manipulations during 1989 was 805 for each respondent, and the number manipulations of the cervical spine 354. Thus, the total number of thoracolumbar manipulations was 342,125, and the total number of cervical manipulations was 150,450. The overall incidence of side effects of transient complications due to cervical spine manipulation such as disturbance of consciousness or radicular signs was 1:16,716. In addition to increased pain, 17 patients (ratio 1:20,125) showed a transient sensorimotor deficit with precise radicular distribution after lumbar spine manipulation therapy. Nine of the 17 patients (ratio 1:38,013) developed a progressive radicular syndrome with sensorimotor deficit and radiologically verified disc herniation and had to be referred for surgery. Side effects and complications of cervical and lumbar spine manipulation are rare. Taking in to account the yearly number of manipulations performed by a single physician in Switzerland and the rate of complications, it can be calculated that a physician practicing manual medicine will encounter one complication due to manipulation of the cervical spine in 47 years and one complication due to lumbar spine manipulation in 38 years of practice [235].

By conclusion, the evidence of today suggests that consistent with a randomised placebo-controlled double-blinded trial [236], after an initial assessment to exclude patients with contraindications, lumbar spine manipulation therapy is safe compared to other noninvasive treatment modalities.

Risks of thoracic spine and rib manipulation therapy

There is very little literature available on specific risks of thoracic spine or rib manipulation therapy. During the past 30 years there have been only four case reports on epidural thoracic hematoma (partially combined with leakage of cerebrospinal fluid) [237–239] and one case report of oesophageal rupture [240] following unclassified, but presumably direct chiropractic manipulations. In addition, there is one case report on rib fractures in an infant following chiropractic manipulation for the treatment of colic [241]. The overall data from the literature available in terms of lumbar spine manipulation therapy suggests the assumption that, after an initial assessment to exclude patients with contraindications, medical thoracic spine or rib manipulation therapy is safe compared to other noninvasive treatment modalities.

Risks of manipulation therapy of the pelvic ring (sacroiliac joints)

There is no literature available on specific risks of manipulation therapy of the pelvic ring or the sacroiliac joints. The data from the available literature in terms of lumbar spine manipulation therapy suggests the assumption that, after an initial assessment to exclude patients with contraindications, manipulation therapy of the pelvic ring or the sacroiliac joints is safe compared to other noninvasive treatment modalities.

Glossary

Adopted from the FIMM glossary. Only terms used in this document are listed.

Convergence
- In the neural system: afferents of different tissues converge to dorsal horn neurons (multireceptive; wide dynamic range neuron, WDR) in the spinal cord and in the medulla oblongata. In biomechanics: position of the facet joints (convergence/divergence).

Counternutation
- Counternutation is the minimal movement of the sacrum. The base of the sacrum shifts backwards and upwards, the tip frontwards and slightly downwards (0.5–1.5°). The countermovement is called nutation.

Diagnosis in manual medicine
- Diagnostic skills in manual medicine build upon conventional medical techniques with manual assessment of individual tissues and functional assessment of the whole locomotor system based upon scientific biomechanical and neurophysiologic principles.

Dry needling
- Intramuscular application of acupuncture needles in order to release contracted muscle areas (myofascial trigger points) by mechanical microstimulation and microtraumatisation.

Free direction
- Free direction is the direction of movement in an articular system in which the intensity of nociceptive afference is not enhanced. Opposite: the direction of movement provoking increase of nociception (direction of painful movement).

Global range of motion
- See range of motion.

HVLA thrust
- High-velocity, low-amplitude thrust.

Hypermobility
- Increase in mobility resulting from congenital, constitutional, structural or functional changes of the joints or soft tissue. It may occur locally, regionally or generalised.

Joint play
- All passive movements of a joint controlled exclusively by gravity or external forces.

Locomotor system
- In the context of manual medicine, the locomotor (or musculoskeletal) system includes the muscles, aponeuroses, bones and joints of the axial and appendicular skeleton, ligaments and those parts of the nervous or visceral system associated with or significantly affected by their function.

Manipulation
- Traditionally, the term manipulation has been understood to refer to the technique of high-velocity, low-amplitude thrust (HVLA). With the development of other techniques, manipulation is understood to refer to a variety of methods that restore normal anatomic and functional relationships within the musculoskeletal system. In most European countries, the term is used exclusively for the technique of HVLA thrust.

Manual medicine
- Manual medicine is the medical discipline of enhanced knowledge and skills in the diagnosis, treatment and prevention of reversible functional disorders of the locomotor system.
- The term defines all scopes of manual medicine and the noninvasive part of musculoskeletal medicine.

Manual medicine physician
- Physician who performs manual medicine.

Manual medicine techniques
- Methods, procedures or manoeuvres taught in a recognised school of manual medicine or employed by a manual medicine physician for therapeutic purposes.

Mechanoreceptor
- Encapsulated nerve endings (receptor endings classified by the method of Freeman and Wyke meeting the following three criteria: (1) encapsulation, (2) identifiable morphometry and (3) consistent morphometry on serial sections) are believed to be primarily mechanosensitive and may provide proprioceptive and protective information to the central nervous system regarding joint function and position.

Mechanotransduction
- The process by which cells convert mechanical stimuli into a chemical response. It can occur in both cells specialised for sensing mechanical cues such as mechanoreceptors, and in parenchymal cells whose primary function is not mechanosensory.

Mobilisation
- Passive, slow and repeated motion of axial traction and/or rotation and/or translatory gliding with increasing amplitude in order to improve restricted articular mobility.

Multireceptive dorsal horn neuron
- Is a dorsal horn neuron especially represented in lamina V in which

a variety of afferents with different qualities and from different organ systems (joints, muscles, skin, viscera, etc.) converge. This results in the first summary of information of the dorsal horn.

Musculoskeletal medicine
- Musculoskeletal medicine embodies all medical disciplines that deal with the diagnosis of acute and chronic conditions affecting the musculoskeletal system in adults and children, including the psychosocial impact of these conditions. Musculoskeletal medicine is a branch of medicine that deals with acute or chronic musculoskeletal injury, disease or dysfunction. Its aim is to address somatic dysfunction, which is an impaired or altered function of the components of the somatic (body framework) system. The somatic system includes the skeletal, arthrodial and myofascial structures with their related vascular, lymphatic and neural elements.

Neuromuscular techniques (NMT)
- A group of manual techniques that incorporate mobilisation by using the contraction force of the agonists (NMT 1), mobilisation after post-isometric relaxation of the antagonists (NMT 2) or mobilisation using reciprocal inhibition of the antagonists (NMT 3).

NMT
- See neuromuscular techniques.

Nocigenerator
- Nocigenerator is an organ or anatomical region that contains C-fibres. It gives information to the central nervous system (CNS) that there are ongoing activities threatening the body, e.g., tissue damage inflammation, mechanical irritation, etc.

Nocireaction
- Is the response of connective tissue, sympathetic and parasympathetic systems, endocrine system, motor system and spinal, subcortical and cortical structures to nociafferent input to the body (hurt, heat, acid, mechanotrauma).

Nutation
- Nutation is the minimal movement of the sacrum. The base of the sacrum shifts forwards and downwards, the tip backwards and slightly upwards (0.5–1.5°). The countermovement is called counternutation.

Painful minor intervertebral dysfunction
- A term used in some European countries to describe the nature of painful dysfunction.

Pain-provocation test
- A test which stresses the body part(s) being tested with functional or physical force in order to elicit diagnostic pain.

Pre-tensioning
- Is part of the preparation of an articular structure in order to perform HVLA thrust.

Prevention in manual medicine
- Patient involvement in the therapeutic activity, resulting from the detailed diagnosis, helps in the prevention of recurrence of somatic dysfunction.

Range of motion
- Range of motion refers to the distance and direction a joint can move between the flexed position and the extended position.

Referred pain
- The nocigenerator being not in the painful tissue (e.g., "Head" zone).

Reversible dysfunction
- A peripheral articular or segmental dysfunction is responsive to manual medicine techniques in the sense of improved or restored function. Manual medicine deals primarily with the diagnosis and treatment of reversible dysfunction.

Segmental celluloperiosteal myalgic syndrome
- Painful minor intervertebral dysfunction causes reflex reactions within the same metamer leading to spinal somatic dysfunction (*syndrome cellulo-périosto-myalgique segmentaire*).

Segmental dysfunction
- Segmental dysfunction is a reversible uni- or multicausal alteration of the normal or physiological vertebral segmental function.

Segmental irritation
- Activation of afferent neurons followed by nocireaction.

Self-mobilisation
- Self-stretching techniques that specifically use joint traction or glides that direct the stretch force to the joint capsule or the muscles involved.

Sensitisation
- The receptive fields are enlarged, threshold in first (peripheral) or second (central) neuron is lowered leading to hyperalgesia.

Soft tissue treatment techniques
- Inhibition technique using digital compression for 1 min of a tender point. Deep transverse friction: strong friction of a structure thought to be malfunctioning (e.g., muscle, tendon). Stretching in a direction perpendicular or parallel to the muscle fibres without tightening the skin.

Somatic dysfunction
- Impaired or altered function of related components of the somatic system (skeletal, arthrodial, myofascial) and related neural, vascular and lymphatic elements. Somatic dysfunction is a reversible dysfunction.

Spinothalamic projection neuron
- See: multireceptive dorsal horn neuron.

Stabilising techniques
- Stabilising techniques in manual medicine consider sensory and motor components related to the locomotor system for optimal stabilisation of the core, the spine or a joint.

Strengthening techniques
- Strengthening techniques involve exercises increasing muscle strength

by putting more strain on a muscle than it is accustomed to receiving. This increased load stimulates the growth of proteins inside each muscle cell that allow the muscle as a whole to increase contraction strength.

Tender point
– Secondary local hyperalgesia without structural lesion (e.g., widespread pain syndrome with multilocular tender points).

Tensegrity
– An architectural principle in which compression and tension are used to give a structure its form.

Trial mobilisation
– A testing manoeuvre to predict possible adverse reactions of manual medicine treatments.

Trial tensioning
– See: trial mobilisation.

Trigger point, myofascial
– Structural lesion within myofibres by contraction of a part of the fibre producing referred pain.

Undirected movement dysfunction
– More than one movement direction in an articular system causing nocireaction.

WDR
– Wide dynamic range neuron, special kind of dorsal horn multireceptive neuron predominantly found in lamina V (see convergence).

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Declarations

Conflict of interest. H. Locher, M. Bernardotto, L. Beyer, M. Karadjova, and S. Vinzelberg declare that they have no competing interests.

For this article no studies with human participants or animals were performed by any of the authors. All studies mentioned were in accordance with the ethical standards indicated in each case.

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