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Low Back Pain: a New Comprehensive Pathogenetic Model Supporting Methods of Medical Rehabilitation

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ABSTRACT
The pathogenesis of chronic low back pain remains elusive. It is still considered a «non-specific» condition, with severity loosely related to anatomical alterations of the lumbar spinal canal (e.g., disc herniation, spinal stenosis). Signs and symptoms may appear contradictory, such as pain aggravated by rest or spinal loading, opposite lumbar postures (flexed or extended) adopted by different patients, and others. Guidelines and reviews oscillate between a restrictive nerve compression model to large sets of epidemiologic factors (from lifestyle to chronic lumbar stress to genetic determinants). A new pathogenetic model is presented here, based on the variable interaction between three possible determinants: compression of nerve endings by disc herniation or arthritic spurs, engorgement of the epidural (Batson) venous plexus, and inflammation triggered by focal thrombophlebitis and fostered by fibrinolytic defects. Hence, the name Compressive-Venous-Inflammatory (CoVIn) is given to the model. Biological and clinical studies provide evidence for each of the three cited determinants. The integrated model explains many «unexplained» characteristics of LBP and provides a rationale for mechanical treatments targeting one or more of the three determinants. Active Lumbar Traction (auto-traction), water exercise, and Williams' flexor exercises look highly consistent with the model, which can explain their effectiveness.

KEYWORDS: low back pain, pathogenetic model, epidural venous plexus, active lumbar traction, balneotherapy, flexor exercises.

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Боль в пояснице: новая комплексная патогенетическая модель, подкрепляющая методы медицинской реабилитации

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РЕЗЮМЕ

Патогенез хронической боли в пояснице остается неясным. Она до сих пор считается «неспецифическим» состоянием, выраженность которого слабо связана с анатомическими изменениями поясничного отдела позвоночного канала (например, грыжа диска, спинальный стеноз). Признаки и симптомы могут быть противоречивыми, например, боль, усиливающаяся в покое или при нагрузке на позвоночник, противоположные позы в поясничном отделе (сгибание или разгибание), принимаемые разными пациентами, и др. Руководства и обзоры колеблются между ограничительной моделью компрессии нерва и большим набором эпидемиологических факторов (от образа жизни до хронического поясничного стресса и генетических детерминант). Новая патогенетическая модель основана на вариабельном взаимодействии трех возможных детерминант: компрессии нервных окончаний в результате грыжи диска или артритных шпор, ущемления эпидурального (по Бэтсону) венозного сплетения и воспаления, вызванного очаговым тромбофлебитом и способствующего развитию фибринолитических дефектов. Отсюда возникло название «компрессионно-венозно-воспалительная» (CoVIn) модель. Биологические и клинические исследования подтверждают наличие каждой из трех указанных детерминант. Комплексная модель объясняет многие «необъяснимые» характеристики боли в пояснице и дает обоснование для механических методов лечения, направленных на одну или несколько из трех детерминант. Активное вытяжение поясницы (ауто-тракция), водные упражнения и упражнения Уильямса на сгибание выглядят в высшей степени соответствующими модели, что может объяснить их эффективность.

КЛЮЧЕВЫЕ СЛОВА: боль в спине, патогенетическая модель, эпидуральное венозное сплетение, активное вытяжение в поясничном отделе, бальнеотерапия, упражнения на сгибание.

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Low Back Pain: a Set of Elusive Syndromes

«Benign» chronic low-back pain (LBP), i.e., pain lasting for more than six weeks, with or without radiation to the lower limbs, is the most prevalent disabling condition globally. Lifetime prevalence in adults of any age is estimated to be around 84 % [1], and prevalence around 23 % [2]. The «benign» qualification refers to syndromes caused by some mechanical dysfunction of the spine, excluding traumatic, inflammatory, infectious and neoplastic causes. This adjective may be over-optimistic. LBP is often chronic and intractable. It causes relevant individual suffering and loss of working capacity in the adult population [3]. The survival of a descriptive diagnosis reveals the scarce understanding of LBP pathophysiologic mechanisms. The state-of-the-art medical knowledge is well summarised in an authoritative «seminar» to which the interested reader is referred [2]. Attempts to classify the various clinical pictures abound [4–7], to say nothing of the proposed treatments, which have always been seen as contradictory (from rest to exercise, from drugs to surgery, from electrotherapy to psychiatric counselling) [8].

The Birth of the Discal-Arthritic Compressive Model

LBP and sciatica (for simplicity, including here also pain radiating anteriorly to the lower limbs) are still considered a «nonspecific» condition [2] mainly consisting of generic, unexplained suffering of back muscle or lumbosacral roots. The proposed aetiologies are multifactorial, variegate and controversial, from anatomical alterations to behavioural and psychological factors to genetic predisposition. The contemporary dominant model in clinical practice is based on the compression of the many nerve endings [9] inside the spinal canal. This model took off after Mixter and Barr's [10] seminal article, which identified disc herniation as a distinct nosologic entity (formerly considered a «chondroma») and associated it with neurological symptoms, including radicular pain. Until the '70s of the 20th century, the in vivo diagnosis could only be made with myelography adopting dangerous lipidic contrasts. Therefore, the diagnosis was limited to frank radicular syndromes, presenting with sensory or motor signs, root-related pain distribution, and the Lasègue's sign. This confirmatory approach probably explains the persistent belief that back pain without sciatica cannot be ascribed to lumbar disc herniation (by the way, the concept of «herniation» will include here, for simplicity, the related pictures of «protrusion» and «prolapse») [11]. Extruded disc herniation, despite its peculiar imaging, will also be included. With hydro-soluble contrast media for myelography and CT and MRI, diagnosis of lumbar disc herniation and spinal stenosis (see below) became easier and virtually harmless (Figure 1).

It also became apparent that herniation of lumbar discs is extremely common in adults, with a peak prevalence between 35 and 60 years of age. The posterior longitudinal ligament, reinforcing the posterior annulus, is tapering from L2 caudally so that lower discs are more prone to disc herniation (in 80 % of herniations, the 4th and 5th discs are affected). Disc herniation could be either asymptomatic or associated with a wide range of conditions, from a mild localised back pain to dramatic cauda syndromes. The CT and the MRI also made evident that another common source of compression of the lumbar spinal root is developmental spinal stenosis, first described by Verbiest [12]. In acquired spinal stenosis, compression of the nerve roots is caused by arthritic changes in the facets (posterior, inter-apophyseal joints), which cause typical claudication more frequently than pain [13]. Many other anatomical structures beyond the spinal roots may generate pain following compression or inflammation within the spinal canal: the discal annulus fibrosus, the posterior longitudinal ligament, the dural sac, the facet joints and the arterial and the venous vessels [9]. No lymphatic vessels can be found inside the vertebral canal [14]. Ganglion compression is enough to cause pain. By contrast, a compression of cauda roots alone causes paraesthesia and sensory loss without pain unless some inflammation exists. This «anatomy of pain» will be considered again in the following paragraphs.

Looking for a Unifying Rationale for Treatment

The disc-compression model soon provided a rationale for the most popular exercise methods, from the classic «flexor» Williams exercises [15] to the prevalently «extensor» McKenzie exercises [16].

These methods claim to fight the «compressive» pathophysiology by striving to widen the vertebral canal section and remodelling the disc herniation. However, results remain unsatisfactory in many single instances. Not surprisingly, most of the traditional treatments, like heat, cold, massage, electrotherapies, manipulations, acupuncture, etc., are merely symptomatic in that they target pain. From guidelines and reviews, LBP emerges as a uniform condition with no clear pathophysiology («non-specific»). The literature leans towards epidemiologic rather than pathophysiologic explanations. These explanations support a widely multifactorial (physical, psychological, environmental) origin of the syndrome and suggest various treatments in various combinations [2, 6]. The literature tends

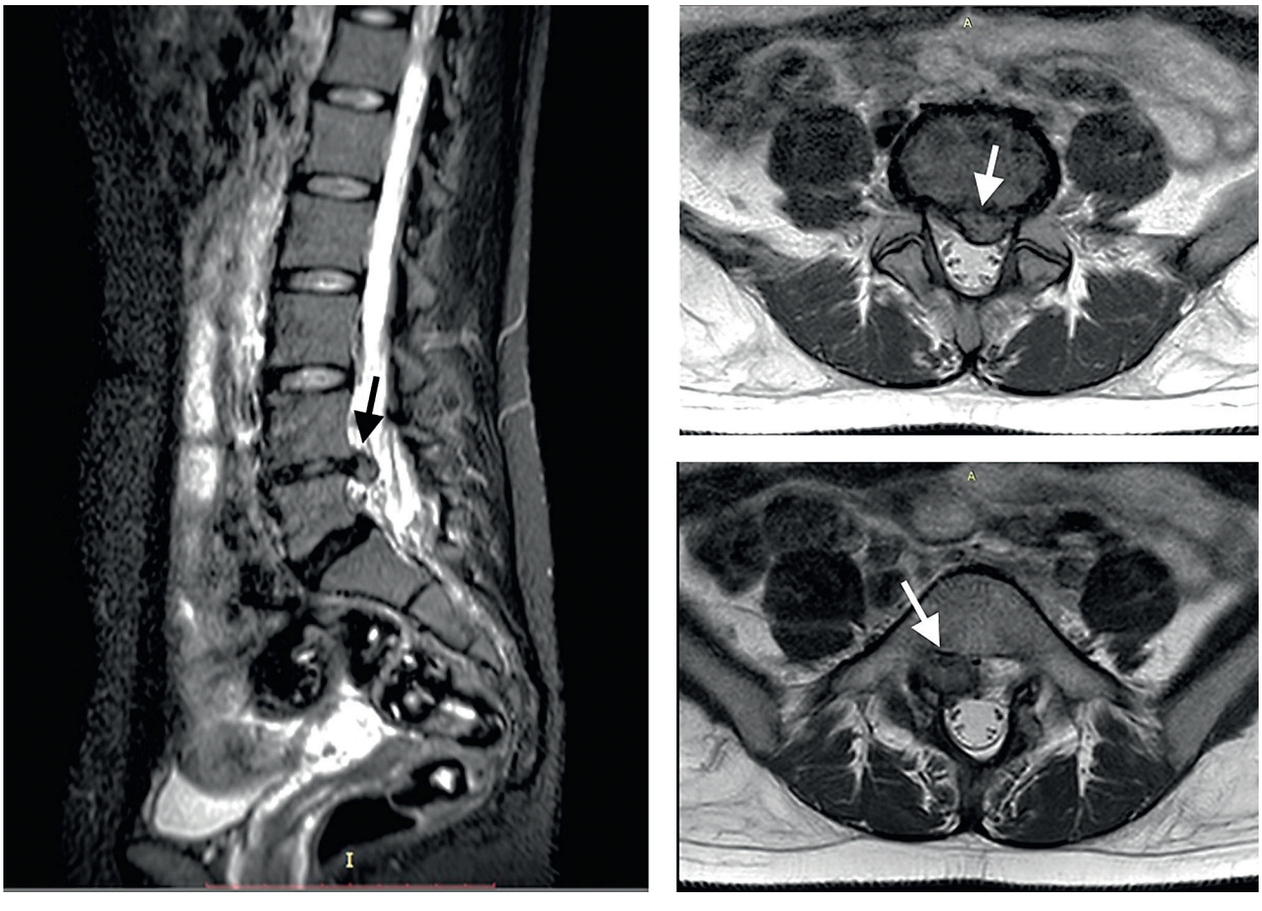

Fig. 1. Typical MRI images (T2 weighted) of a 4th lumbar disc, herniated bilaterally (though prevalently to the right).

Note: *In the sagittal view, on the left, an extruded nucleus pulposus is evident at the 4th disc level (black arrow). The 4th and 5th discs are dehydrated («black» discs). In the axial view, the disc herniation appears on the top right panel in the mediolateral left position (white arrow). The extrusion spreads downward in the bottom right picture and obliterates the right L5-S1 foramen. The patient is a 30-year-old woman. Five weeks before she suffered from an acute onset in the right back and sciatic pain (L5 territory) up to the foot dorsum. There was moderate weakness of foot dorsal flexion. A complete recovery was achieved at the time of the visit after the steroid treatment. The pictures highlight the precise details available on lumbar spine anatomy through MRI and, on the other hand, the scarce consistency between the radiological severity and the clinical findings (personal observation).*

to accept chronic LBP as an intractable condition. Current therapeutic approaches consider pain more like a disease than a symptom, so prevention of worsening and increased tolerance is recommended. Therefore, the most various treatments are attempted, with no clear classifications of patients. This may explain why evidence of the effectiveness of «exercise» is still weak [17]. This defeatist attitude is a waiver for research on a deeper pathophysiologic model.

Limitations of the Compressive Model

A pathophysiologic model should explain most of the available observations. Unfortunately, the discal-arthritic compression model is far from satisfactory in this respect. The main eight observations missing a convincing explanation are listed in Table 1. A point-by-point explanation will be attempted in the following paragraphs based on the model proposed in this article.

The Venous Theory: the Core of a New Model

Backpain Following Visceral Problems

A seminal article by Batson [19] raised awareness in the medical community of the neglected epidural venous plexus. This is the fourth venous system (after the portal, caval and pulmonary systems). It runs in the epidural space merged with the epidural fat. It drains the venous outflow from the pelvis and the entire spinal column and has extensive anastomoses with retroperitoneal and intracranial veins. The epidural veins are valveless. They are virtually collapsed but can become engorged due to increased central venous pressure (e.g., in case of heart failure or pulmonary hypertension, or any increase of the abdominal strain), obstruction of the caval system, compression inside the spinal canal, or increased blood circulation (as in the case of pregnancy). Batson was only interested in understanding the spread of metastases (e.g., prostate metastases bypassing the liver and spreading to the lung, vertebrae, and skull). In the following years, LaBan highlighted the association of back pain with simple recumbency and with several visceral conditions like liver cirrhosis, chronic obstructive pneumonitis, and heart failure [20–23]. Pregnancy [24] and even the obstruction of the left renal vein in the aorto-mesenteric «nutcracker» [25] could also be related to LBP by mediating the plexus engorgement. Spinal stenosis also emerged as a condition favouring vein-related symptoms. The restless legs syndrome could also be ascribed to this mechanism [26]. Angiographic and MRI studies confirmed venous engorgement in these conditions [23].

«Primitive» Back Pain and Venous Engorgement

Curiously enough, the possibility that venous engorgement could contribute to «benign» LBP, beyond being a consequence of visceral problems, was not exploited. In 1991, Tesio formulated this hypothesis explicitly [27].

He looked for an explanation of the puzzling effectiveness of the Swedish auto-traction treatment (now Active Lumbar Traction) he imported to Italy in 1984 (see below). The technique, paradoxically, increases the discal pressure. Its effectiveness challenged the view that disc herniations were the dominant cause of pain, i.e., the the rationale for «decompressive» exercises [16]. Clinical observations suggested other relevant predisposing factors. These might be, for instance, sitting or erect posture (increasing the hydrostatic venous pressure at the trunk), increased abdominal pressure (e.g., constipation or Valsalva manoeuvres implied by coughing and sneezing), and spinal stenosis, all pointing toward epidural venous congestion as a possible pain generating mechanism, consistently with Batson's and LaBan's studies. Tesio also extended the «venous» hypothesis to the possibility of local phlebitis caused by venous stasis and fostered by fibrinolytic defects. This vascular-inflammatory hypothesis was supported by a series of rigorous studies by Jayson and coworkers [28–32]. These authors, however, emphasised the role of inflammation (e.g., triggered by the acid content of the nucleus pulposus or by fibrinolytic defects) while overshadowing the hydraulic mechanism provided by the a-valvular Batson plexus, prone to engorgement in a restricted space.

Based on the positive response to the paradoxical auto-traction treatment, Tesio also revitalised the potential role of venous stasis in back pain appearing during pregnancy, a well-known and substantially unexplained condition [33].

Table 1. Characteristics of benign chronic back pain unexplained by a purely disc-compressive hypothesis

a)	The discrepancy between the number and size of disc herniations and the severity of pain (if any)
b)	The pain-relieving posture adopted by the patients with evidence of lumbar disc herniation. As a rule, patients prefer a flexed/crouched lumbar stance. In some cases, an extended posture is chosen. Subjects selecting an extended pose usually suffer from a recent/acute episode
c)	LBP aggravated by rest or initiation of movement after rest: typically, with exacerbation in the morning after a nocturnal sleep. Paradoxically, recumbency and rest make the intradiscal pressure decrease
d)	LBP in pregnancy is a frequent condition [18] that is not significantly associated with lumbar disc herniation
e)	Evidence that the risk factors for developing LBP, with or without lumbar disc herniation, overlap with the risk factors for cardiovascular accidents (i.e., sedentary lifestyle, smoking, dyslipidaemia, fibrinolytic defects)
f)	Some LBP syndromes are permanent, while some can resolve spontaneously after various periods. The CT or MRI imaging is usually unrelated to the time course of pain
g)	When a conservative treatment effectively relieves or resolves pain, imaging before and after treatment may not change
h)	The long-lasting results of transient mechanical treatments

In the following years, anatomic [34] and refined imaging studies [35] confirmed Batson's anatomical findings and the association of epidural vein dilatation with back pain or sciatica [36, 37]. Consistently with the critical role of the venous outflow in back pain syndromes, a human peculiarity was evidenced by other authors in the vascular supply of lumbosacral roots. These are exceptionally long in humans and are stretched some 3 cm during lumbar and lower limb flexion, challenging blood supply. Cauda's roots contain arterio-venous anastomoses with a spiraliform (pig-tail) shape. This may ensure an arterial flow through the veins during root stretching. Of course, the venous pressure must not be too elevated [38]. However, the above observations and research lines did not converge in an organic model. Furthermore, some observations remain to be explained (see Table 1 and below). An updated pathophysiologic model is simplified and suggested here.

The Compressive-Venous-Inflammatory (CoVIn) Back Pain Model

The puzzling observations listed in Table 1 can all find a reasonable explanation if the discal/arthritic compression model is integrated with the hypothesis of epidural venous engorgement and local thrombophlebitis. I dubbed this model Compressive-Venous-Inflammatory (CoVIn). The model is sketched in Figure 2.

What follows is a step-by-step response to the questions covered in Table 1.

a) The discrepancy between anatomical and clinical findings can originate from the variable concurrence of discal compression, canal section reduction, epidural veins' engorgement, patient's harmful movements, and local inflammation. Not all of these factors are detectable through CT or MRI.

b) The flexed lumbar posture widens the spinal canal. A colourful debate originated around the displacement of the nucleus pulposus. It seemed reasonable to sustain the idea that it migrates toward the direction of a widening intervertebral distance, e.g., posteriorly in flexion and anteriorly in extension. Clinical experience, however, clearly shows that chronic LBP patients tend to avoid lumbar extension. Several studies on anatomical specimens and living subjects (even with invasive contrast discography) gave contradictory results for decades. Looking at the heterogeneous literature on this topic I think that the following convincing conclusions can be drawn:

- During flexion, the nucleus tends to migrate posteriorly, without causing meaningful discal bulging, only if the annulus is integer. In any stage of disc degeneration, the nucleus migrates anteriorly. The phenomenon reflects the loss of elastic resistance of the annulus so that on the side of vertebral rapprochement (i.e., anteriorly), the annulus sags and opposes less resistance to the internal hydraulic pressure. The posterior annulus (and the posterior longitudinal ligament) is elongated and tightened during flexion.
- The opposite occurs during extension. Extension causes a disc prolapse of 1 to 4 mm even in recumbency, primarily due to the annulus sagging. It has been shown that extension alone restricts even by 20 % or more the osseous transverse section of the spinal canal at L3-L4 and L4-L5 levels compared to flexion because of the increased overlap between the posterior joint facets, much more than because of disc prolapse [39, 40]. This restriction is even more prominent in the case of posterior bulging of the disc and can become dramatic in spinal stenosis [35].

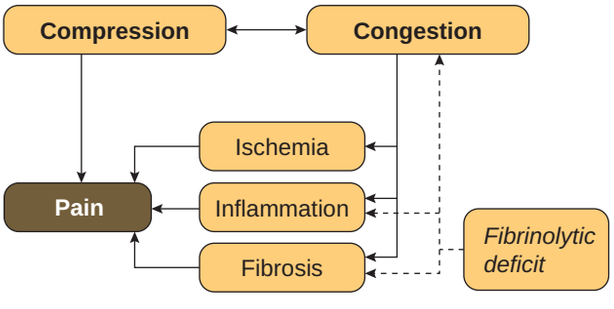

Fig. 2. A sketch of the Compressive-Venous-Inflammatory (CoVIn) pathogenetic model of low back pain
Note: *Compression of neural endings and dilation of the epidural veins (Batson's plexus) can both lead to pain either alone or by reciprocal enhancement. Venous congestion may act through ischemia of nerve endings or an inflammatory thrombophlebitic process, possibly evolving into local fibrosis. Fibrinolytic defects foster the phlebitic processes. Ideally, physical rehabilitation should contrast both arms of this model.*

Patients preferring an extended posture usually represent acute cases suffering from the classic «witches' blow». In this case, a dural sac involvement must be considered. Defensive reactions to meningeal pain overtake the responses to other forms of pain (including discal-arthritic pain). The dural sac is elongated during spine flexion and shortened during extension [41]. Therefore, in case of inflammation, any manoeuvre stretching the sac (spinal flexion but also the neurologic signs of Lasègue, Kernig, and Brudzinski) exacerbates pain. Spinal extension attenuates pain. The acute pain (witch's blow) is usually transient, lasting no more than a couple of days, and can respond quickly to steroid medication (primarily dexamethasone). The «extended» patient frequently turns into a «flexed» patient after the acute phase has elapsed.

c) Rest may decrease the discal pressure but prevents the muscles' physiological «venous pumping» activity. Therefore, venous volume is increased during any static posture, particularly during night recumbency. In restricted bony spaces, venous congestion can become very painful because of direct compression on nerve endings or ischemia; the same happens with sinusitis, dental pulpitis, and osteomyelitis. Back pain looks just a variant of this phenomenon. Of course, increased pressure on the disc and overload on the facet joints may result from heavy physical activity thus contributing to pain. It may well happen that some patients suffer from pain both at rest and during certain motor activities or only during the latter.

d) Pregnancy causes a remarkable increase in blood flow. In the last three months of pregnancy, the caval venous return shows a 6-fold increase. The epidural plexus thus becomes a timely escape route at the cost of venous engorgement within the narrow, inextensible spinal canal.

e) Venous stasis may be the first step towards potential phlebitis, implying thrombosis and chronic fibrosis. The

epidural fat in patients undergoing repeated unsuccessful surgery for disc herniation (causing arachnoiditis) is similar to the subcutaneous fat in superficial thrombophlebitis [29]. This explains why chronic LBP and cardiovascular diseases share some risk factors (sedentary lifestyle, smoking, dyslipidemia), all known to decrease the fibrinolytic capacity of the plasma. This notwithstanding, concerning these associations (in particular, smoking), «the underlying mechanisms remain obscure» for the dominant Literature [2].

f) Venous stasis and inflammation may be transient phenomena, as it is well known for other conditions (from haemorrhoids to subcutaneous phlebitis to deep vein thrombosis). The venous-inflammatory component of the model may thus explain both the chronicity and the temporality of the pain symptoms in chronic LBP.

g) A successful treatment must not necessarily modify the CT or MRI images of the spine. First, imaging does not highlight the pressure exerted by compressive structures (disc or arthritic spurs) on nerve endings. Micro-displacements may cause clinically meaningful decompressions. Second, routine MRI imaging does not target the decrease of venous engorgement, although it should be possible to conduct specific studies nowadays [42–44].

h) The favourable results can be permanent for many reasons (e.g., slow neural adaptation and long-term atrophy of a prolapsed nucleus pulposus). Still, the end of a phlebitis status is a sufficient explanation in most cases.

The Efficacy of Some Mechanical Treatments Supports the CoVIn Model

If the CoVIn model and the above explanations hold, conservative mechanical treatments (i.e., physiotherapy) should aim at

- Widening the lumbar canal section.
- Fostering decongestion of the epidural plexus.

Ideally, both of these related aims should be pursued. I'm now describing three mechanical treatments with established effectiveness that seem most consistent with the CoVin model. These treatments are Active Lumbar Traction (originally, Auto-traction), «flexor» (Williams's) lumbar exercises, and water exercises. The perspective is not one of asserting that, among the dozens of other conservative approaches available, these should receive priority in any instance. Instead, they are brought as clinical evidence further supporting the CoVIn pathophysiological model.

Auto-Traction Treatment / Active Lumbar Traction

Active Lumbar Traction (originally «auto-traction») is a mechanical treatment of benign chronic LBP performed on a specially designed traction bench. Auto-traction was invented by the Swedish physician Gertrud Lind in 1974 [45]. The original table was manually operated. It was provided by electro-hydraulic mechanisms by Lind's pupil Emil Natchev. Natchev also formalised and disseminated the method through a manual, seminars and one-week intensive courses [46]. In 1984 Luigi Tesio attended one of Natchev's courses in Stockholm, introduced the technique in Italy, designed a simplified bench and further simplified the method, which a physiotherapist can now learn in a few days [47, 48]. As shown in Figure 3. (Tesio's method), the technique requires a bench electrically operated. The bench is transversally divided into two sections.

Depending on the response to the treatment, the patient lies supine, prone or side position while «anchored» to the foot end of the bench by a corset and a chain. Each section of the table can be tilted or rotated by the therapist. Whichever position the therapist selects, the patient exerts «auto-traction» manoeuvres, lasting 4–6 seconds, by grasping ad hoc bars on the head section of the table. Gradual relaxation then follows for the next 6–10 seconds. During the traction efforts with the upper limbs or between subsequent pulling actions, the patient can exert pushing efforts with the lower limbs against ad hoc bars on the foot end of the table to modify the lumbosacral orientation. The treatment begins with the search for the least painful back position. The patient is then asked to provide subsequent traction efforts (about 20 per session). The therapist gradually moves the patient towards the formerly painful positions by tilting and/or rotating the table's sections during the efforts or the pauses. Efforts should become painless. In 3 to 9 half-hour sessions in 5 to 10 working days, the whole range of lumbar movement must become painless or much less painful. As a consequence, active trunk mobility also increases. Neurological and thermographic signs can also improve, likely due to removing pain-related neural inhibition [33]. The treatment is discontinued if no significant improvement is observed after three sessions. Results are usually stable (at least three months, but several years — if not permanently — in the Author's experience). In about 75 % of the cases, pain can disappear or decrease to less than 30 % of the pre-treatment values (whichever pain scale is adopted) [49, 50]. No aftercare of any type is advised. The clinical and radiological severities

Fig. 3. The active lumbar traction (auto-traction) method for low back pain (see text and Manual for details; pictures from [48] — permission granted)

are loosely related to prognosis [51, 52]. The method may be successful in chronic LBP in pregnancy [53]. Lind's, Natchev's, and Tesio's method versions provide superimposable results [52]. Contraindications and side effects are minimal (see Tesio's Manual for details). Two isolated reports suggest that other custom-made exercises, inspired by Lind's method, might work without a dedicated bench [54, 55].

How Does it Work? Active Lumbar Traction is a Form of Vigorous Exercise

Evidence for the effectiveness of Active Lumbar Traction is sound [47]. However, the orthopaedic community was sceptical from the beginning of the method because the traction manoeuvres increase the discal pressure [56] through the contraction of paravertebral muscles. The name of the method may have needed to be more accurate. Lind thought that «auto» traction caused vertebral distancing and disc decompression, a mechanism ascribed (quite optimistically) to a traditional passive lumbar traction. For this reason, Tesio highlighted that Lind's «auto-traction» is a form of active exercise and renamed the method Active Lumbar Traction [48]. In light of the CoVIn model, the most likely mechanism of action appears to be the decongestion of the Batson plexus — preventing or attenuating local phlebitis — through the muscular venous pump, associated with selective canal widening and remodelling of the interface between the disc and painful nerve endings. From his perspective, a transient increase in discal pressure looks no longer like a paradox; the same holds for many of the «unexplained» features of LBP syndromes listed in Table 1. The accurate 3D positioning of the patient is consistent with the validated principle of matching the patient's «directional preference» [57]. From the CoVIn perspective, this ensures that the increase of discal pressure is harmless and funnels both the venous outflow and the disc remodelling in the right direction.

Water Exercise

For centuries, balneotherapy has been adopted for treating LBP and various rheumatic disorders. Its effectiveness in this field is established [58–61]. The effects of the chemical and thermal properties of the various types of water adopted and the purely physical properties of water still need to be disentangled. The physical effects of immersion in «neutral» water will only be considered here. A temperature of around 34 °C is considered «neutral» because immersion does not cause any physiological response to heat or cold in humans. Other physiological responses, however, abound. The immersed body receives a push towards the surface (Archimedes principle), which is stronger the greater the water density and the volume of the body submerged. The body lightening, minimising the need for muscular contractions, probably explains the notorious relaxing effect of immersion. Most importantly for the present discussion, the body is «wrapped» by a hydrostatic pressure increasing with the depth of the body immersion. In the case of vertical immersion, the highest effect on venous «squeezing» occurs when the water level reaches the compressible abdomen, thus generating an extra-venous flow towards the heart. In a human with a height of 1.8 m, the heart volume can increase from about 560 to 800 ml when vertically immersed up to the axillae [62]. The over-distension of the right atrium is (erroneously) interpreted by the heart as hypervolemia. The heart stroke becomes stronger (Starling's law), the systolic pressure increases, the diastolic pressure decreases, and the heart rate drops by at least 15 %. In addition, atrium distension enhances the secretion of the atrial natriuretic hormone and elicits, via the Vagus nerve and the Hypothalamus, an increased diuresis [63]. In 20–30 minutes, the interstitial fluid is also «squeezed» into the venous network, thus prolonging the cardiac and diuretic reaction to immersion. Meanwhile, the plasma is diluted, its viscosity decreases, and blood cells' concentration decreases. These changes attenuate the heart overwork. All these effects may outlast an immersion period of 30–40 minutes by a couple of hours [64–66].

Immersion in neutral water can thus be beneficial in many instances, from lymphoedema to heart failure, cirrhosis, chronic kidney diseases, varicosities, and hypertension [67–70] and, not surprisingly, oedema in pregnancy [71]. Not surprisingly, a simple «tank» dedicated to immersion therapy was projected [72]. However, it is of interest here that immersion may help decongest the epidural venous plexus. This looks like the most reasonable mechanism explaining its effectiveness in LBP and, in so doing, most of the «paradoxes» listed in Table 1.

Williams' «Flexor» Exercises

Two seminal twin papers by Williams must be recalled here. In the first paper, Williams proposed a typical history of lumbar spine degeneration [73]. Chronic traumas (mostly minimal and unnoticed) on the lumbosacral joints would trigger disc degeneration and increase lordotic posture. This leads to posterior 5th disc prolapse and a rapprochement of the adjacent vertebrae. Collision, subluxation and arthritic deformation of the facet joints cause the vertebral canal to be restricted. Williams also cited that local venous engorgement in a restricted canal might contribute to pain. The attention paid to the canal section inspired the second paper [15]. This paper proposed a set of exercises fostering a less lordotic lumbar posture. These exercises included stretching the hip flexors, strengthening the hip extensors, and active flexion (self-elongation) of the lumbar spine. Since the 1930s, Williams's exercises have been a popular standard. They are entirely consistent with the CoVIn model as long as they emphasise the widening of the spinal canal. In successive decades, the original set of passive and active exercises was widened, but still, they bear Williams' name. Their effectiveness is established, although, like for all types of exercises designed for LBP, evidence is of low grade (grade III or IV) [17, 74, 75].

Final Considerations

The CoVIn model is supported by epidemiological, clinical, anatomical, biochemical and in vivo imaging studies. The CoVIn model can also accommodate other «benign» syndromes (their discussion goes beyond the scope of the present article). One example is provided by the acute «extensor» cases (witch's blow), which can be explained based on a meningeal irritation. Another example is provided by post-surgical arachnoiditis, which is, in essence, a phlebitis/fibrosis process imprisoning nerve roots [76]. The CoVIn model explains more characteristics of the elusive chronic-benign back pain syndromes than other, more popular models. On an «anatomical» extreme, the latter rest

on root compression, generic «inflammation», or «stress» of muscles and joints. On an epidemiologic extreme, they rest on environmental causes (lifestyle, occupational hazards), psychological predisposition and genetic constitution. Given the complex and variable pathogenesis of pain, the former approach seems too simple, while the latter seems applicable to populations, not individuals.

Can we derive therapeutic suggestions from the CoVIn model? Regarding Physical and Rehabilitation Medicine, the model legitimises at least Active Lumbar Traction, water exercises, and flexor (Williams's) exercises. The above methods might be proposed as reasonable first-line approaches before more invasive procedures (e.g., surgery, epidural injections) are attempted. Of note, these three treatments may claim for an aetiologic, not only symptomatic, rationale. Therefore, they seem preferable to purely symptomatic procedures, mainly targeting pain as a symptom (e.g., painkillers, electrotherapy, acupuncture, massage, diathermy, etc.). Further reflection, however, is needed on drugs. The venous arm of the model suggests that old drugs might play a new, more-than-symptomatic role: these drugs are, for instance, flavonoids and heparinoids, whenever a phlebitis/fibrosis process can be suspected.

The CoVIn model is not conceived as a mechanistic prescription but as a logical framework helping medical reasoning. Two notes of wisdom, therefore, are needed. First, each patient is unique. Depending on medical judgment, invasive or purely symptomatic treatments may be more indicated than, or compatible with, treatments inspired by the CoVIn model. Second, despite converging evidence of various kinds, the effect of any mechanical treatment of LBP on epidural venous engorgement still waits for direct experimental demonstration, reinforcing a large list of clinical studies.

ADDITIONAL INFORMATION

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