Review article

Lumbar spine traction: evaluation of effects and recommended application for treatment

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SUMMARY. Despite the widespread use of traction, little is known of the mode of effect, and application remains largely anecdotal. The efficacy of traction is also unclear because of generally poor design of the clinical trials to date, and because subgroups of patients most likely to benefit have not been specifically studied. These observations prompted this review, the purposes of which are to evaluate the mechanisms by which traction may provide benefit and to provide rational guidelines for the clinical application of traction. Traction has been shown to separate the vertebrae and it appears that large forces are not required. Vertebral separation could provide relief from radicular symptoms by removing direct pressure or contact forces from sensitised neural tissue. Other mechanisms proposed to explain the effects of traction (e.g. reduction of disc protrusion or altered intradiscal pressure) have been shown not to occur. We conclude that traction is most likely to benefit patients with acute (less than 6 weeks’ duration) radicular pain with concomitant neurological deficit. The apparent lack of a dose-response relationship suggests that low doses are probably sufficient to achieve benefit.

INTRODUCTION

Traction is widely used for the treatment of lumbar spine conditions accounting for approximately 7% of physiotherapy sessions in The Netherlands (van der Heijden et al. 1995), but its application is largely based on clinical experience because there has been no systematic evaluation of its practice. Traction is most commonly used for normalization of neurological deficits or painfully restricted neuromeningeal tension signs (Gillström & Ehrnberg 1985; Knutsson et al. 1988), the relief of pain (Cyriax 1980; Grieve, 1981) and for improving joint mobility (Grieve 1982; Maitland 1986). The little evidence available suggests that traction is more effective for pain reduction and return to activity than infra-red radiation (Mathews et al. 1987), corset and bed rest (Larsson et al. 1980), hot packs and rest (Lidstrom & Zachrisson 1970), hot pack, massage and mobilization (Lidstrom & Zachrisson 1970) and bed rest (Moret et al. 1998). However, no apparent advantage is produced by varying the application of traction, including the magnitude of force (Buerskens et al. 1997; Pal et al. 1986). The differing results from trials of traction shown in Table 1 have furnished conflicting evidence for the efficacy of traction and this has been further interpreted in clinical guidelines to mean that traction is an ineffective modality for the management of lumbar spine conditions (Bigos et al. 1994; New Zealand Ministry of Health 1997).

The controversy over the effectiveness of traction may result from the generally poor design of the studies. Design problems include comparison of heterogeneous study populations, application of several treatment modalities in each treatment session, uncertainty about the appropriate dose of traction, and the apparent lack of a valid sham intervention. Since many authors have assumed that intervertebral separation is essential for efficacy and that large forces are required to achieve separation, a large traction force has been considered a treatment and this has often been compared with a smaller force of 10–20% body weight (Table 1). However, there is evidence to suggest that even a small dose of 9 kg (Twomey 1985, 1989; Twomey & O’Shea 1988; Twomey et al. 1987) may be sufficient.
Lee & Evans (1992) provides a mechanical effect. Low forces of 5–10 kg are also recommended based on clinical observations (Maitland 1986).

Recently, Beurskens et al. (1997) demonstrated that high dose traction is as effective as a comparison sham treatment of low dose traction (10–20% body weight) for subacute and chronic non-specific low back pain (NSLBP) with or without leg pain. After 12 weeks, both treatment groups improved by approximately 50% in terms of global perceived effect and 4.3 on the Roland Morris Disability Questionnaire. That is, a greater force did not result in greater therapeutic effect. It might be argued that a ‘sham treatment’ that has an effect of 50% is a real treatment, and consequently that such studies suffer from deficiencies in design. These data, therefore, do not contribute to the argument that traction is an ineffective treatment; in fact, it could be argued that they help define the minimum dose.

It would appear that the efficacy of traction cannot be clearly interpreted from the literature. The relative efficacy of the various modes of application (continuous or intermittent, manual or motorized), the force applied and the duration and frequency of treatment have not been clearly investigated. Several issues therefore remain unresolved, particularly:

- selection of the traction application: this includes the traction technique, determination of an appropriate dose, and signs and symptoms to monitor immediately following treatment.
- identification of subgroups of patients who may or may not benefit from traction. There are few randomized controlled trials clearly establishing the outcomes of traction, and little clarity about the effects of traction. Research to date has investigated heterogeneous sample populations, and is therefore likely to have included those patient groups who do not respond to traction. However, preliminary work suggests that traction may be effective for subgroups of patients, such as

Table 1. Randomized controlled trials published in English that investigated the efficacy of traction

<table>
<thead>
<tr>
<th>Authors</th>
<th>Problem</th>
<th>Traction Treatment</th>
<th>Comparison treatment</th>
<th>Referred pain</th>
<th>Neuro deficit</th>
<th>Authors’ overall conclusion</th>
</tr>
</thead>
<tbody>
<tr>
<td>Matthews et al. (1987)</td>
<td>Acute and subacute LBP</td>
<td>Continuous motorised traction (&gt; 25% body weight)</td>
<td>Infrared radiation</td>
<td>Included</td>
<td>Excluded</td>
<td>Significant difference found in subgroup</td>
</tr>
<tr>
<td>Larsson et al. (1980)</td>
<td>Acute and subacute LBP</td>
<td>Autotraction + corset</td>
<td>Corset + rest</td>
<td>Included</td>
<td>Included</td>
<td>Significant difference found</td>
</tr>
<tr>
<td>Coxhead et al. (1981)</td>
<td>Mixed strata LBP</td>
<td>16 treatment groups. Treatment combination: intermittent motorized traction alone or + corset or exercises, or manipulation</td>
<td>i) No intervention ii) Exercises iii) Manipulation iv) Corset</td>
<td>Included</td>
<td>Not stated</td>
<td>Significant difference found</td>
</tr>
<tr>
<td>Lidstrom &amp; Zachrisson (1970)</td>
<td>Chronic LBP</td>
<td>Intermittent motorized traction (58–95 kg) and isometric abdominal exercises</td>
<td>i) Hot packs and rest ii) Hot packs massage and mobilization</td>
<td>Included</td>
<td>Not stated</td>
<td>Significant difference found</td>
</tr>
<tr>
<td>Weber et al. (1984)</td>
<td>Prolapsed lumbar disc</td>
<td>Manual traction</td>
<td>Isometric exercises</td>
<td>Included</td>
<td>Included</td>
<td>No significant difference</td>
</tr>
<tr>
<td>More et al. (1998)</td>
<td>Lumbar radicular syndrome</td>
<td>Traction and bed rest</td>
<td>Bed rest</td>
<td>Included</td>
<td>Included</td>
<td>Significant difference found</td>
</tr>
<tr>
<td>Ljunggren et al. (1984)</td>
<td>Chronic LBP: prolapsed lumbar disc</td>
<td>Autotraction + back school + bed rest</td>
<td>Manual traction + back school + bedrest</td>
<td>Included</td>
<td>Inclusion criterion</td>
<td>No significant difference</td>
</tr>
<tr>
<td>Weber (1973)</td>
<td>Prolapsed lumbar disc</td>
<td>Continuous motorised traction (40–70 kg)</td>
<td>Autotraction</td>
<td>Included</td>
<td>Included</td>
<td>No significant difference</td>
</tr>
<tr>
<td>Weber et al. (1984)</td>
<td>Prolapsed lumbar disc</td>
<td>Autotraction</td>
<td>Continuous motorised traction (10 kg)</td>
<td>Included</td>
<td>Not stated</td>
<td>Significant difference</td>
</tr>
<tr>
<td>Matthews &amp; Hickling (1975)</td>
<td>Mixed strata LBP</td>
<td>Continuous motorised traction (37–61 kg)</td>
<td>Continuous motorised traction (9 kg)</td>
<td>Included</td>
<td>Excluded</td>
<td>No significant difference</td>
</tr>
<tr>
<td>Pal et al. (1986)</td>
<td>Acute and subacute LBP</td>
<td>5.5–8.2 kg hospital continuous traction</td>
<td>1.2–1.8 kg hospital continuous traction</td>
<td>Included</td>
<td>Included</td>
<td>No significant difference in improvement</td>
</tr>
</tbody>
</table>

The force and type of traction application, and the comparison treatment are described. The study populations are briefly described in terms of duration of symptoms, and whether subjects with referred pain or neurological (neuro) deficit were included. Acute low back pain is defined as symptoms of < 6 weeks duration, subacute as symptoms lasting 6 weeks to 3 months, and chronic low back pain as symptoms lasting > 3 months. The authors’ conclusions about the efficacy of traction are also given.
those with radicular pain and neurological deficit (Larsson et al. 1980; Moret et al. 1998).

An evaluation of the proposed effects of spinal traction has therefore been undertaken to derive a more rational therapeutic approach to the use of traction for low back pain.

**SELECTION OF TRACTION AS TREATMENT**

Traction is generally selected as a treatment of choice for patients with a neurological deficit (Grieve 1981), because the presence of an acute or unstable neurological deficit is considered to contraindicate management by passive accessory mobilization and high velocity manipulation. Traction is also recommended for spinal stiffness or bilateral pain referred into the lower limbs since the origin of bilateral symptoms is thought to be symmetrical or 'central' rather than unilateral (Grieve 1981). Thus, bilateral pain should logically be managed using manual techniques applied either 'centrally' (over the spinous process) or symmetrically using traction or physiological movements in the sagittal plane (Maitland 1986). Generalized spinal stiffness is also considered an indication for management by traction (Grieve 1981) since traction is thought to affect numerous spinal levels during a single application (Maitland 1986).

**EFFECTS OF TRACTION**

Traction could improve signs and symptoms by both biomechanical effects, such as separation of the intervertebral motion segment (Twomey 1985), and neurophysiological effects, such as modulation of nociceptive input in either the ascending (Watkins & Mayer 1982) or descending pathways (Zusman 1986), as postulated for SMT (Table 2). The division of traction effects into mechanical and neurophysiological is somewhat artificial, however, because most clinical effects are probably produced from a combination of the two. For example, the mechanical effect of vertebral separation may induce neurophysiological changes that are responsible for pain reduction. Therefore, the following discussion evaluates the effect of traction in terms of the proposed clinical effects, that is, relief of signs and symptoms.

**Normalization of neurological deficit and relief of radicular pain**

Neurological deficits associated with radicular pain are thought to arise from mechanical compromise, ischaemia or inflammation of the spinal nerve/dorsal root ganglion/nerve root complex (Hasue 1993), possibly associated with abnormalities such as intervertebral disc lesions and osteophytic encroachment into the intervertebral foramen (Lindblom & Rexed 1948). Such deficits have occasionally been found in association with specific subgroups of patients with low back pain (LBP). Inflammation of the spinal nerve/nerve root complex has been reported in association with disc disease (Grönblad et al. 1994a) but not with lumbar spondylosis (Nordström et al. 1994). Histological changes suggesting the presence of inflammation in zygapophyseal joints have also been reported (Cooper et al. 1995). Vascular changes have been reported in the spinal nerves of patients with motor and/or sensory neurological deficit accompanied by positive straight leg raise test and reduced spinal mobility. These changes are thought to result from mechanical compromise that obstructs venous outflow (Jayson 1992) and produces ischaemic damage and ultimately fibrosis (Cooper et al. 1995). A particularly interesting finding is that application of nucleus pulposus material to cauda equina nerve roots causes degenerative neural changes, including fibre atrophy, Schwann cell oedema and axonal vascularization in pigs (Olmarker et al. 1993). Although uncertain, it is possible that disc disruption in humans could also cause degenerative neurological changes around the nerve root (Lindblom & Rexed 1948). Finally, proteins associated with peripheral nerve damage have been

| Table 2. Proposed mechanical and physiological effects of traction and supporting evidence |
|-----------------------------------------------|-----------------------------------------------|------------------|
| Effect                                      | Evidence                                      | Authors         |
| Intervertebral separation                   | Strong in vivo and in vitro evidence to support | Colachis & Strohm 1969; Twomey 1985; Lee & Evans 1993 |
| Silencing of ectopic impulse generators     | Moderate evidence to support hypothesis in animal model | Howe et al. 1977; Bini et al. 1984 |
| Reduction of intervertebral disc protrusion | Weak evidence to refute hypothesis             | David 1992      |
| Altered intradiscal pressure                | Weak evidence to refute hypothesis             | Anderson et al. 1983 |
| Normalization of conduction in spinal nerves/nerve roots | Weak evidence to support hypothesis             | Knutsson et al. 1988; Onel et al. 1989; Tesio et al. 1989 |
| Pain relief:                                |                                              |                 |
| ● increase in non-nociceptive input         | Untested hypothesis                           |                 |
| ● recruitment of descending inhibition      | Untested hypothesis                           |                 |
| Increased joint mobility                    | Untested hypothesis in lumbar spine           |                 |
identified in patients with chronic lumbar pain, but not in patients with occasional episodes of NSLBP (Cameron et al. 1995). Thus, there is some evidence that inflammation, vascular changes and neural degeneration may be associated with LBP with a neurological deficit.

These pathological changes reported to accompany neurological deficit could theoretically be relieved by traction. For example, separation of the vertebrae, thereby increasing the diameter of the intervertebral foramen could reduce radicular pain and normalize neurological deficits by relieving direct pressure or contact forces in sensitized neural tissues (Colachis & Strohm 1969; Twomey 1985). Ectopic impulse generation thought to result from these factors (Lindblom & Rexed 1948) could be reduced or ablated, thus reducing radicular pain and symptoms (Howe et al. 1977).

Other hypotheses, although largely unfounded, have also been advanced to support the use of traction for patients with radicular pain accompanied by neurological deficit. These hypotheses include the possibility that the traction force causes separation of the vertebrae resulting in reduction of disc protrusion or altered intradiscal pressure (Andersson et al. 1983).

Intervertebral separation

Intervertebral separation has been investigated both in vitro and in vivo. Lumbar intervertebral separation has been demonstrated when isolated lumbar spine specimens are subjected to sustained traction loads (Twomey 1985; Lee & Evans 1993). Twomey (1985) applied a distraction load of 9 kg for 30 minutes to lumbar spine specimens, finding that most distraction occurred immediately the weight was applied with 15% more separation to a maximum of 7.5 mm separation occurring as a result of creep. Interestingly, both Lee and Evans (1993) and Twomey (1985) found that most intervertebral separation occurred when their specimens were positioned to flatten the lordosis, a position that simulates the typical traction position when the hips are flexed to approximately 90° to place the legs on a stool. Furthermore, vertebral separation measured in vivo using plain radiography was shown to occur with a traction force of 50 lb (approximately 20 kg) applied either as a static or intermittent force in normal subjects (Colachis & Strohm 1969; Bridger et al. 1990). The poor reliability for plain radiography (Nachemson 1988) indicates caution in interpreting these results, although the findings are consistent with those found for isolated lumbar specimens (Twomey 1985; Lee & Evans, 1993). These findings suggest that intervertebral separation does occur during the application of traction with greatest separation occurring when the patient is positioned in supine with the hips flexed, and immediately after the distraction force is applied.

Low forces have not been studied in vivo, however, the low forces used for in vitro studies (body positioning) are unlikely to generalize to in vivo applications, because in vivo, some of the traction force would be dissipated in surrounding tissues. However, forces as large as those studied in vivo (50 lb) may not be necessary to incur the small amount of intervertebral separation sufficient to increase the size of the intervertebral foramen and slow or silence ectopic impulse generation.

Silencing of ectopic impulse generators

Ectopic discharge can occur in the presence of endoneurial oedema in animals (Howe et al. 1977) and humans (Nordin et al. 1984) in the dorsal root ganglion (DRG) following damage that occurs distally in the neural pathway. Endoneurial oedema within the DRG may result from inflammatory exudate compressing the DRG (Chatani et al. 1995). It is possible that intervertebral separation during traction may relieve pressure on the DRG or spinal nerve at the intervertebral foramen, and silence ectopic impulse discharge. Reduction in discharge has been shown in cats when such compression was removed (Howe et al. 1977). Alternatively, it is possible that mechanical stimulation of large diameter myelinated fibres may decrease pain associated with ectopic nerve stimulation (Bini et al. 1984) although findings are inconsistent (Howe et al. 1977). Traction may constitute an adequate mechanical stimulus for large diameter fibres and thereby decrease pain. Thus, traction may reduce radicular symptoms by reducing ectopic discharge from the generating site.

Reduction of intervertebral disc protrusion

Intervertebral disc abnormalities diagnosed from CT (Wiesel et al. 1984; Haldeman et al. 1988; Jackson et al. 1989), radiography (Goldie & Reichmann 1977; Korber & Bloch 1984; Nachemson 1992) and MRI (Jackson et al. 1989) have been shown to be poorly correlated with symptoms. Nevertheless, it has been hypothesized that traction improves symptoms by reducing intervertebral disc protrusions (Andersson et al. 1983). The evidence for this hypothesis is unclear (Mathews 1968; David 1992). Mathews (1968) injected contrast medium into the lumbar spines of three patients and took lateral radiographs before, during and after spinal traction. Multiple disc protrusions were reduced in two patients during the application of 54.5 kg for 30–40 minutes, however, 14 minutes after release of the traction force, the protrusions had reappeared, although not to the original size. Design problems with this pilot study
include the lack of a control group, lack of accuracy inherent to radiographic measurements and failure to correlate observed changes in disc contours with signs and symptoms. Despite these limitations, this study is frequently cited as evidence that distraction of the spinal vertebrae either creates a suction force that reduces disc prolapse, or tightens the posterior longitudinal ligament such that the disc is forced back to its original location, thus reducing symptoms (Gupta & Ramarao 1978; Andersson et al. 1983; Saunders 1986).

In contrast, no correlation between signs and symptoms and reduction of intervertebral disc pathology was found in a more recent study. David (1992) studied four patients with severe, constant low back pain who were treated with hospital traction, followed by out-patient traction and bed rest until full recovery (between 2 weeks and 2 months). Patients’ lumbar spines were scanned using CT before treatment and after full recovery. After full recovery, disc herniation was reduced in two patients but unchanged in the other two (David 1992). This pilot study was limited by subject numbers and the poor reliability of CT, however, the findings suggest that there is no clear correlation between symptoms and observable ‘abnormalities’ on scans, and further, that recovery is not related to alteration of disc ‘abnormalities’.

Altered intradiscal pressure

Altered intradiscal pressure, another commonly cited consequence of spinal traction, is also largely unsupported. It is thought that decreased intradiscal pressure may relieve symptoms caused by severe disc degeneration (Cyriax 1980; Saunders 1986; Fast 1988). The single investigation of this hypothesis, however, found that, while no alteration in pressure was recorded in the nucleus pulposus of healthy L3/4 intervertebral discs during application of motorized traction for 30 seconds using an unspecified force, a considerable increase in intradiscal pressure was reported using patient-generated traction for 2 minute with a force of 500 N (Anderson et al. 1983). These results cannot be generalized to ‘abnormal’ discs and it is not possible to study abnormal discs using these methods. Given the lack of relationship between disc abnormalities and symptoms (Haldeman et al. 1988), and the fact that traction effects on disc protrusion probably dissipate in less than 14 minutes (Matthews 1968), it is unlikely that any reduction in pressure is responsible for symptomatic improvement. Furthermore, any reduction in pressure would be unlikely to continue after the patient assumed an upright posture, since 49% of body weight is above the L3/4 of disc (Judovich 1955).

Normalization of conduction

Traction has been shown to normalize sensation, reflexes and muscle power by some authors (Knutsson et al. 1988; Onel et al. 1989; Tesio et al. 1989) but not others (Pal et al. 1986). Normalization of deficits may result from restoration of normal conduction in large diameter myelinated afferent and efferent nerve fibres. Conduction could be restored in a number of ways. An increase in intervertebral foramam diameter is likely to result in improved blood flow within the spinal nerves and intra-foraminal blood vessels, and thus reduce any existing ischaemia, although the duration of such effects after cessation of traction is unknown. Increased blood flow could, in turn, remove inflammatory exudate. In addition, traction could alleviate mechanical compression which is a possible cause of neurogenic inflammation. Compression is a confirmed stimulus for ectopic impulse generation (Groen et al. 1988; Grönblad et al. 1994b) and its removal would theoretically also remove the cause of conduction block.

Improvement in the straight leg raising test

Traction has also been shown to improve painfully restricted SLR (Larsson et al. 1980; Pal et al. 1986), probably by increasing the diameter of the intervertebral foramens, thus decompressing neural tissue and reducing neural sensitivity to movement. Nociceptive responses are rarely evoked by stretch or compression of healthy spinal nerves and nerve roots (Howe et al. 1977). When nerve is structurally damaged or inflamed, however, ectopic and nociceptive impulses can be generated as a result of increased sensitivity to stimulation, such as from tension, the mechanism proposed to underlie observed reductions in SLR (Smyth & Wright 1958; Howe et al. 1977; Boland 1995). Inflammation of neural tissues has been correlated with decreased range of motion of SLR (<70°) (Kawakami et al. 1994). It is possible that inflamed neural tissue could limit SLR by increased reflexogenic muscle activity, because during SLR the inflamed DRG may be subject to stimulation by direct pressure or tension (Smith et al. 1993). The resultant stimulation generated in the inflamed nerve roots may cause sufficient nociceptive discharge to stimulate the reflexogenic drive to the hamstring alpha-motoneurones as demonstrated in animals (Jaenic & Koltzenburg 1991; Woolf et al. 1994). Increased EMG activity in the hamstrings muscle group in response to SLR has been described in a single patient with SI radiculopathy (Hall et al. 1998), but not in response to other tension tests, such as the ‘slump’ or prone knee bend tests (Lew et al. 1994; Hall et al. 1998). Thus, there is limited evidence to support these hypotheses.
Pain relief

Causal pathology can be identified in approximately 15% of patients with low back pain (Waddell 1998). In the remaining 85% of patients there is no pathology that is currently recognized to explain the pain, and such pain is therefore termed non-specific low back pain. It is occasionally recommended that traction be used to relieve such pain, particularly when symptoms are bilateral (Grieve 1981).

There is substantial evidence to explain pain generated by tissue insult. Nociceptive specific receptors innervated by small diameter unmyelinated fibres and polymodal receptors are stimulated by noxious stimuli. Nociceptive information is conveyed to higher centres in the brain, where it is perceived as pain, by five major ascending pathways (Jessell & Kelly 1991). The majority of nociceptive information is conveyed via two tracts, the spinothalamic tract and the spinomesencephalic tract. The spinothalamic tract is composed of axons of nociceptive-specific and wide dynamic range (WDR) neurones that terminate in the thalamus after decussation, and the spinomesencephalic tract projects to the periaqueductal grey region (PAG). The PAG has reciprocal connections with the limbic system (responsible for the emotional response) and the spinal cord (Jessell & Kelly 1991).

Once pain is generated, the response to non-noxious input can be exaggerated by central sensitization, expansion of receptive fields and peripheral receptor hyperactivity. In addition, ectopic impulses can be generated in the dorsal root ganglion if the nerve is damaged more distally (Wall & Devor 1983). These changes can occur within hours of injury because of the plasticity of the nervous system (Bennett & Xie 1988), perhaps to dissipate the increase inafferent traffic (Coghill et al. 1991; LaMotte et al. 1991). Continued nociceptive input could maintain neuronal hyperexcitability (Gracely et al. 1992), and tonic excitation of WDR neurones may result in decreased inhibition (Alkon & Rasmussen 1988; Collingridge & Singer 1990). It has been hypothesized that interventions such as traction may provide non-noxious input to reverse these events.

Pain may be modulated in a number of ways, including by increasing non-nociceptive input and recruitment of descending inhibition. Relevant neurophysiological mechanisms of pain modulation have been reviewed in the context of the effect of spinal manual therapy on NSLBP (Zusman 1986) and more recently on acute and chronic muscle spasm (Kataovich 1998). Since similar mechanisms are likely to be recruited by an application of traction for NSLBP, and information specifically related to traction is currently unavailable, the reader is referred to these reviews. The role of traction for relief of radicular pain accompanied by neurological deficit has been previously evaluated.

Increased joint mobility

The effect of traction on range of motion has not been investigated in the lumbar spine, but there is some evidence to suggest that a transitory increase in physiological range of motion occurs following the application of intermittent cervical traction (Goldie & Landquist 1970; Lidström & Zachrisson 1970; Zylbergold & Piper 1985). Design problems in all studies, however, suggest that the results should be interpreted with caution.

It is hypothesized that the mechanism responsible for such changes is the alteration of length and mobility of connective tissue structures (Threlkeld 1992). Connective tissue such as ligaments, joint capsule and periarticular fascia provide resistance to forces acting on joints and if abnormally shortened may alter joint motion (Frank et al. 1984; Twomey & Taylor 1991; Threlkeld 1992). Separation of the vertebral bodies may provide a stretch to the spinal soft tissues that is adequate to induce a transitory increase in length. In addition to the stretch stimulus, distraction forces have been shown to increase the length of spinal tissues by creep and hysteresis (Twomey & Taylor 1992). In vitro studies of lumbar intervertebral discs (Twomey 1985) have shown that elongation of the tissues is greater in health (approximately 2 mm) than in the presence of degeneration (approximately 1 mm) and is of longer duration in older specimens (30 minutes) than in young (0 minutes) (Twomey 1985). This preliminary evidence suggests that, if traction is used with the aim of lengthening spinal tissue, the optimal dose is likely to involve prolonged application and to involve decreasing force with increasing age.

Summary

In light of the evidence presented, it is clear that many of the clinical anecdotes concerning the effects of traction cannot be supported. That is, there is no lasting effect on the IVD, and any transitory effect is not related to symptom relief. However, traction has been shown to separate the IV motion segment, although the clinical value of IV separation is unknown. Intervertebral separation is likely to be most clinically relevant in patients with LBP with radicular symptoms according to current understanding of the involved pathology. The mechanism of pain modulation also remains unproved, but conforms with pain theories accepted in other disciplines.
RECOMMENDATIONS FOR THE CLINICAL APPLICATION OF TRACTION

Clinical guidelines are usually constructed from the best evidence currently available. In the absence of well-designed RCTs that have investigated the therapeutic efficacy of traction, recommendations for clinical application must be derived from the available evidence about its mechanical and physiological effects. It is evident from the present review that all the suggested effects of traction, only interspinous separation in vitro and in vivo has been clearly demonstrated. Pain modulation by traction has not been proven, but the scientific basis for an effect can be constructed from available knowledge. Theoretically these mechanisms could explain symptom relief, particularly when associated with neurological deficit, and thus provide a reasonable basis for the application of traction until further information becomes available.

Choice of traction table

The following discussion is based on the assumption that forces delivered from a traction apparatus are transferred to the patient, i.e. little force is lost in overcoming friction between the patient and the bed. This notion is valid if patients are treated with a traction apparatus that has a split-table function, where the lower part of the body rests on a mobile part of the bed that can slide away from the upper body when the traction force is delivered through a belt around the pelvis. Goldish (1989) demonstrated that a horizontally aligned traction force delivered 96% of the applied force to a simulated body. The remaining 4% of force was lost to factors that included overcoming friction associated with the sliding bed. The first recommendation must therefore be for the therapist to consider factors that promote the efficient delivery of traction forces. A split-table should be used and, logically, the segment immediately above the region of interest (such as, the L4 vertebrae) should rest on the fixed part of the traction table so the region to be distracted (L4/5 level and below) rests on the mobile part of the bed (Judovich, 1955). Goldish (1990) contends that hospital bed rest traction is ineffective because the low forces involved cannot overcome friction and the tensile properties of tissues.

Patient selection

Treatments are usually selected on the basis of the pathological or clinical diagnosis, and the stage of the condition. Since traction has been shown to increase the diameter of the IV foramina, it is likely to benefit patients whose condition is due to pathology in the IV foramen. The most common such condition is LBP with radicular symptoms that may be caused by mechanical compression, ischaemia or inflammation of the spinal nerve/DRG/nerve root complex.

The stage of LBP most likely to improve with traction is the acute stage (duration 6 weeks). This hypothesis is based on current understanding that SMT (‘mechanical’ treatment) is most effective in the acute phase (Di Fabio 1992) and less effective in the subacute (duration of 6 weeks – 3 months) and chronic phases (duration >3 months) (Maher et al. 1999). Effective treatment for LBP of greater than 6 weeks’ duration is likely to be achieved by active rather than passive modalities, such as an exercise programme (Maher et al. 1999). Since traction is a passive, ‘mechanical’ treatment, it is likely to be most effective in the acute stage of the disorder. Therefore, it seems reasonable to recommend that traction will provide the greatest benefit to patients with acute LBP, radicular symptoms and neurological deficit.

Selection of dose of traction

Traction dose is influenced by the variables of magnitude, frequency, ‘constancy’ (i.e. whether the traction is intermittent or sustained), duration and direction of the applied distraction force. None of these variables has been systematically investigated, however, information about magnitude of the distraction force can be derived from several studies (Lidstrom & Zachrisson 1970; Weber 1973; Twomey 1985; Beurskens et al. 1997). Until further evidence is available, it seems prudent to recommend the use of the minimum duration, force and frequency that achieves the desired outcomes. There is little information to guide recommendations about the ‘constancy’ or direction of the applied force.

Various assumptions underlie the force advocated as optimal (i.e. >20% body weight) in the clinical literature. First, it is assumed that IV separation is required for therapeutic efficacy and second, that 20–50% body weight is required to achieve such separation (Beurskens et al. 1997). Since the IV separation occurs when the lumbar lordosis is flattened (Twomey 1985), these assumptions can be questioned. In addition, if therapeutic benefit does result from IV separation, the available evidence suggests that separation is achieved at low forces (e.g. 9 kg, or approximately 10–20% body weight) (Twomey 1985; Lee & Evans 1992). Furthermore, therapeutic efficacy is not improved by application of larger forces. In the seven randomized controlled trials reviewed by van der Heijden et al. (1995), traction was compared to a ‘placebo’ treatment which in all cases was a smaller traction force (approximately 20% body weight). An improvement in outcome was demonstrated in all studies, but no difference was found between the large or small force...
applications. While this was interpreted to mean that the treatment group had no effect, these data could instead indicate that a large force is unlikely to confer greater therapeutic benefit than a small force. Thus, we propose that to relieve acute LBP, it is likely that even small forces could provide the desired IV separation.

Although the purpose of spinal traction is to achieve maximum relief of symptoms, it is common practice to aim for partial rather than total relief of severe pain within one treatment to prevent an exacerbation of symptoms after release of the distraction force (Hickling 1972; Grieve 1981; Maitland 1986; Saunders 1993). The rationale is that sudden reloading of the pathological spinal segment on completion of traction causes an increase in firing of nociceptors and mechanoreceptors. This is not considered during the application of other manual treatments, does not have a sound theoretical basis, and has not been investigated. However, if exacerbation of symptoms is of concern or the response on release of traction suggests that the patient should exercise care on rising, the patient could reload the spine and surrounding structures prior to standing by isometric muscle contractions or gentle active movements, such as rotation in supine.

In the presence of a neurological deficit, good results could potentially be attained using sustained traction applied at low forces for prolonged treatment periods, e.g. less than (10 kg for 20–30 minutes. It is also worth noting that positioning patients in the typical traction position, with the hips and knees flexed to approximately 90° has been shown to increase the length of the lumbar spine (Twomey 1985) and therefore alters dimensions of the IV foramina and spinal relationships in vivo. We suggest that traction should be sustained rather than intermittent to increase foramina dimensions for prolonged periods (20–30 minutes), allowing removal of inflammatory by-products from within spinal nerves and perhaps the DRG, and to promote patency within periradicular venous and arterial vessels.

Traction is also advocated for the improvement of joint mobility. Because of the viscoelastic properties of connective tissues, the strain that results from a tensile load varies with the rate of loading. That is, tissue is deformed more with a slow rate of loading than a fast rate of loading, and with sustained loading (Threlkeld 1992; Lee & Evans 1993). There are probably more effective means by which to achieve improved mobility, although it is possible that longitudinally directed forces have unique effects. Furthermore, older spines experience creep at a slower rate than young spines (Twomey 1985). It may be appropriate, therefore, to use small forces with longer duration of traction for older patients, but the patient should be closely monitored by reassessment of signs and symptoms.

Monitoring the effect of traction
Prescription of any treatment is based on predicted effects. However, the treatment effects associated with traction are not wholly predictable and evidence is lacking. Thus to justify the use of traction, a therapist must observe improvement in signs and symptoms and in so doing, modify treatment dose as necessary. The outcomes monitored should reflect the purpose of the treatment. Since it is recommended here that traction should be selected as the treatment of choice in the presence of radicular signs and symptoms, the patient’s neurological status should be monitored. Furthermore, there is some evidence that deficits in SLR, reflexes, muscle power and sensation can be improved with traction (Larsson et al. 1980; Knutsson et al. 1988; Onel et al. 1989). Therefore, the magnitude of the traction force could be determined by the response of these signs and symptoms, in addition to the pain response before, during and after traction (Grieve 1981; Ljunggren et al. 1984; Gillstroem & Ehnberg 1985; Maitland 1986; Knutsson et al. 1988).

CONCLUSION
The efficacy of traction for acute NSLBP is currently unclear, although it appears to be less effective in the subacute and chronic phases than in the acute phase (Table 2). Of the mechanisms proposed to explain efficacy of traction, only IV separation has been demonstrated, although pain modulation has a sound scientific basis. Therefore, the patients most likely to derive benefit from traction are those with acute LBP with associated radicular symptoms and neurological deficit. Based on the evidence reviewed here, a small force could be sufficient to achieve the desired effects of IV separation. The selected dose should be monitored, by reassessment of pain intensity and location, the status of any existing neurological deficit and by assessment of SLR. Future research should be directed towards investigation of changes in these variables in the defined group of patients with acute LBP and radicular signs.

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