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ACC and back injuries: the relevance of pre-existing asymptomatic conditions revisited

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Abstract

The application of the New Zealand Accident Compensation Corporation (ACC) legislation in the management of patients who sustain back injuries requires a detailed knowledge of the pathogenesis of tissue injury, and the natural history of ageing and related conditions, so that the application of the ACC Act(s) is appropriate. We have reviewed the new information published in the last decade, and updated the previous knowledge basis in these fields, so as to assist the interpretation of the Act(s).

A decade ago we published a viewpoint article that focused on ACC entitlements for ACC patients with lumbar spine conditions who had a sustained a personal an injury by accident.[1] Our concern was that a number of ACC claimants were being declined on the basis of radiological findings (often discovered on then newer imaging modalities such as MRI scans) rather than an appropriate focus on the claimant's history.

Since that time changes have occurred to the ACC Acts, and their administration and the increased medical knowledge in relation to the prevalence and natural history of common spinal conditions has questioned accepted dogma. Over the same period a vast number of opinions have been published. These range from independent expert medical opinion, ACC medical advisory opinion, independent legal opinion, ACC review decisions, and up to District Court judgements. The latter of these can be considered expert legal opinion that establishes case precedents.

Our observation is that the quality of these opinions is highly variable, and this is reflected in the downgrading of the weight given to expert medical evidence.[2] Evidence based medicine and evidence based observational literature is now given greater weight than the expert opinion of an individual. From the outside, one might see parallels in the legal system where the higher courts form decisions with panels of judges but the lower courts rely on the expertise of a judge sitting alone whose judgement creates precedents for the future.

In New Zealand most ACC appeals end at the District Court, and it has been our observation that there are occasions when the judge has not clearly understood the medical details of the case,[3] or given apparently different decisions in similar cases thus creating both precedent and confusion.[4]

When the ACC legislation came into operation in 1974 it was the clear intention of the legislators to provide treatment, rehabilitation and compensation for patients suffering injury caused by accident. This intent has continued, but the ACC system does not cover congenital or developmental disorders, nor infective (de novo) nor malignant, nor chronic musculoskeletal conditions that occur other than resulting from trauma.

Given the increased diversity of opinions, viewpoints, and expertise paralleled by increasing knowledge of the prevalence of various symptomatic and asymptomatic spinal conditions, it is appropriate to review the medical knowledge in relation to recent Acts. As in the previous article we will focus on the Act(s) and the current and past relevant literature.

Changes in the Act through 2000, 2001, 2005, 2008 and 2010 have focused upon different areas of ACC administration and changes to the definition of personal injury and accident have been relatively minor.

Accident

The definition of accident in the current Act is unchanged from the 2001 Act:

Accident

(1) *Accident* means any of the following kinds of occurrences:

> (a) a specific event or a series of events, other than a gradual process, that—
>
> > (i) involves the application of a force (including gravity), or resistance, external to the human body; or
> >
> > (ii) involves the sudden movement of the body to avoid a force (including gravity), or resistance, external to the body; or
> >
> > (iii) involves a twisting movement of the body:

This definition leaves a wide scope for the interpretation of actions or activities that fulfil this definition. It does not comment on intention, although the 2010 Act excludes the effects of deliberate self harm (Section 119). Perhaps more importantly, the definition of accident does not require assessment of the magnitude of the force. There is no exclusion on the basis of a trivial injury.

Personal Injury

The current Act has modified Section 26 of the 2001 Act, although there is no change to the relevant clause discussed here.

(1) Personal injury means—

> (a) the death of a person; or
>
> (b) physical injuries suffered by a person, including, for example, a strain or a sprain; or ….

Personal injury requires a physical injury yet the nature of such an injury is not described but includes a sprain or a strain. Presumably physical injury would include laceration, haematoma, muscle or tendon rupture, fracture, and neural or vascular injury.

The Act does not require imaging or histological abnormalities to establish the diagnosis of an injury and does not give any other guidelines. It does not exclude changes at a cellular or neural level, which are the mechanisms now considered to be involved in pain syndromes,[5] with the biochemical and neural changes occurring after initiating traumatic events.

The Act does specify that cover includes a sprain or a strain.

A sprain is stretched or torn ligament while a strain is a stretched or torn muscle or tendon (US National Institute of Health). These are soft tissue injuries, diagnosed from the history of injury and the examination findings. X-rays may be taken to exclude a bony injury and may demonstrate soft tissue swelling.

Newer forms of imaging may demonstrate tissue discontinuity but this is not required for the diagnosis. Medically, we accept that the diagnosis of a sprain or strain involves a history of injury, pain, some restriction of motion and tenderness which will be more noticeable the more superficial the site of injury.

It is important to note the lack of any prescription of the force magnitude at the time of injury, nor any requirement for high tech imaging or histologic examination of the affected area to confirm the diagnosis, nor any limitation to the duration of cover that relates to "normal" times for recovery from specific injuries.

Exclusion

The Act includes provisions for exclusion of ACC entitlement in sections that relate both to the definition of accident and personal injury. In Section 25 of the Accident Compensation Act 2001 an accident is not a gradual process (accident means ... a specific event or series of events, other than gradual process...).

In Section 26 personal injury is excluded by the statement "personal injury does not include personal injury caused wholly or substantially by a gradual process, disease or infection unless ...". "Personal injury does not include a personal injury caused wholly or substantially by the aging process".

In summary, the diagnosis of personal injury by accident cannot include personal injury caused wholly or substantially by gradual process, disease, infection or the aging process. The intent of the legislation is clear. However, as the aging process is universal, the implications of these exclusion criteria warrant further attention. First, it is appropriate to consider the modifying descriptors for the exclusion criteria. "Wholly or substantially" defines the contribution of disease, gradual process, or aging that would lead to exclusion for cover.

The phrase "wholly" is unequivocal. "Substantially" leads to considerable arguments. In our 2000 article we used the Webster's dictionary for the word 'substantial', for which 'substantially' is "being largely but not wholly that which is specified" which translates to "in the most part" or "significantly". Thus for ACC cover to be excluded the major component of the ongoing personal injury needs to relate to gradual process, disease or the aging process. Thus, if the major component of the personal injury is the accident, and not gradual process, disease or aging on the balance of probabilities, (the legal standard of proof required by the Act,) then ACC coverage occurs.

In practical terms this raises the question as to what symptoms or personal injury the patient would have had had the accident not occurred? If the patient would likely have been symptomatic with a gradual process, disease or the aging process then the accident cannot be the whole or substantial cause of the symptoms or personal injury.

Conversely if the patient would likely have been free of symptoms had the accident not occurred, yet is subsequently suffering from symptoms after an accident, then it is the accident that is the whole or substantial cause of the subsequent personal injury. Using this latter consideration to determine whether personal injury is covered requires a clear understanding of the natural history of disease and the aging process. [Significantly there have been legal arguments and opinion that conclude that a mere component of aetiology, rather than more than half of the aetiology, can still be "substantial".]

Aging/spondylosis/degeneration

As gradual process, disease and the aging process are reasons for exclusion for cover it is essential to understand the natural history of these conditions. It is clear from the literature that it is very difficult to differentiate aging and degenerative disease in the lumbar spine.[6]

The concept of aging from a medical perspective is relatively clearcut. When tissues age there are changes at the cellular level, causing biochemical and tissue changes which change tissue behaviour and, importantly, biomechanical behaviour. These normal changes of age are manifest by skin thinning and wrinkling, the stiffening of the lens the eye, and loss of hair pigment, which are expected to occur.

It is unlikely that the founders of the ACC legislation wished to exclude cover for victims of personal injury just on the basis of age. ACC would accept a laceration caused by trauma in an elderly person with thinner skin, and a fracture of the hip in an elderly person with likely age related osteopenia. So what is the significance of aging changes in the lumbar spine? First it is necessary to clarify medical terminology.

The changes of lumbar spondylosis include disc space narrowing, osteophyte or spondylophyte formation and vertebral end-plate sclerosis shown on plain x-rays. MR scans will show more detail including disc desiccation, annular disruption with disc bulging, disc prolapse, annular tears, and end plate changes. These are considered as disc degeneration or spondylosis. If they are not symptomatic, then this can be clarified by the addition of the qualifying adjective "asymptomatic". If, however, the patient has mechanical axial pain in this setting then the changes can be considered as a disease and the term degenerative disc disease is appropriate.

Again by way of clarification, we would emphasise that an asymptomatic, normally functioning individual is not diseased and therefore does not suffer from degenerative disc disease.

Over the last two decades it has become clear that the lumbar spine shows increased MR abnormalities with increasing age in asymptomatic individuals.[7,8] More recently and most importantly, it has become clear that these changes are not predictive of current or subsequent disability.

Boos et al[9] demonstrated the MR findings were much less predictive of subsequent disability than psychological and physical aspects associated with work. Borenstein et al[10] noted that the development of new low back symptoms in patients with previously abnormal MR scans was not related to the degree of MRI abnormality that predated the onset of symptoms or to changes in MRI appearance at a later stage.

Jarvik et al[11] concluded that depression was a more important predictor of low back pain than any MRI finding, except where new disc herniation had occurred when that herniation was clinically relevant. In essence, these authors showed that the common aging changes in the lumbar spine are not predictive of subsequent pain and disability and therefore the concept that a person with pre-existing MRI abnormality would have a high likelihood of going on to develop significant pain and disability is incorrect.

Asymptomatic spondylosis should not be regarded as a pending clinical problem. In this situation the diagnosis of any new state after injury might include a sprain to the back or new pathology may be considered to have occurred with new onset of symptoms, e.g. disc prolapse.[11] Thus the sudden onset of new pain and disability after an accident (in a person previously asymptomatic) is not likely due to the aging process / degeneration / disease, but at least substantially due to the accident. Stated another way, had the patient not sustained the accident, they would have likely remained asymptomatic.

For these reasons it remains our viewpoint that a patient who is symptom free prior to a clearly defined event should not be denied ACC cover. It is the accident that is the whole or substantial cause of the symptoms and not gradual process, aging or disease.

Post-traumatic imaging abnormalities

There are now improved experimental models that demonstrate how a normal disc may be damaged. Axial load produces increased pressure within the disc and may be combined with flexion, rotation and impulse creating a combination of annulus, end plate and nuclear damage. These changes reflect the clinical history and the events causing the symptoms. They probably can occur in previously normal spines and experimental models certainly favour this environment as facilitating disc damage.[6,12-18]

Given the clear evidence in large animal models of progressive disc degeneration after injury,[19] these changes are reasonably considered as post traumatic if they fit the clinical presentation (normal function prior to injury, clearcut accident and later discovery of spondylotic changes). We are unaware of any credible evidence in clinical practice that demonstrates that disc degeneration or spondylosis can be reversed.[6] The time for the development of injury changes is difficult to determine. Widespread osseous changes are likely to take time to develop, but loss of nuclear hydration alone is likely to occur rapidly. Loss of hydration is an almost universal finding in acute disc prolapse, and as noted, it is unlikely that a significant number of these discs were abnormal prior to the injury.

In summary, as with most musculoskeletal presentations, the history and examination are paramount and the imaging findings are supportive. Previously normal patients who develop low back pain and disability after accident should not be denied ACC cover based on the current Act and the current medical knowledge. However it must be acknowledged that the history given by the patient is not always accurate,[20] and evidence of pre injury medical or other consultation with pre injury imaging on file would cast doubt over a history of being "previously normal".

Spondylolysis and isthmic spondylolisthesis

Spondylolysis and isthmic spondylolisthesis represent a further area of challenge for the legislation. Again, we see people who have been asymptomatic and unaware of any existence of an abnormality, who subsequently sustain an accident with resulting symptoms and are denied cover. Again, it is important to look at the aetiology of a spondylolysis and its natural history.

Dysplastic spondylolisthesis in childhood can be considered developmental in the medical sense[21-23] - (developmental, pertaining to the development of a condition during growth, either in the intrauterine phase or early childhood (Dorlands Medical Dictionary). This form of developmental spondylolisthesis may be excluded from cover, being a gradual process. Degenerative spondylolisthesis occurs with facet joint arthritis and variable degrees of disc degeneration resulting in spondylolisthesis. It is generally a condition that occurs over time as a gradual process and is typically symptomatic over a variable duration so it could be excluded from cover, being both a gradual process and a degenerative disease.

Spondylolysis (and isthmic spondylolisthesis) is generally accepted to be a failure or stress fracture of the pars interarticularis under flexion and that fails to heal.[24,25] This is an acquired condition usually occurring in late adolescence. Once acquired, it is usually stable and asymptomatic. Conventional orthopaedic teaching has been that the incidence of spondylolysis is 6% in the community and 3% of adults have a low grade spondylolisthesis. A recent observational study from the Framingham Heart Project indicates that the incidence of pars defects may be as high as 11% in a cross sectional study.[26]

Clearly all of these people do not present for treatment. Studies in the Scandinavian literature suggest that there is no evidence that patients with a spondylolysis or low grade isthmic spondylolisthesis have increased risks of back disability through life.[27-29] This has been confirmed by Frederickson et al whose long-term study did not find any increase in problems throughout life for spondylolysis and isthmic spondylolisthetic patients when compared with normals.[30] The Framingham Study also found that patients with spondylolysis or isthmic spondylolisthesis had no increase in lumbar spine symptoms when compared with the non spondylolytic or non isthmic spondylolisthesis patients.

In summary, there is no high quality observational literature that suggests that spondylolysis or isthmic spondylolisthesis predisposes to increased back pain or disability in adult life, but there is now good quality evidence that it does NOT predispose to an increased risk or rate disability. Given that spondylolysis (and isthmic spondylolisthesis) is not a gradual process (yet may occur after a specific event or series of events - the normal fracture or stress fracture aetiology), and neither can it be considered a disease in the asymptomatic individual (which may represent 10% of the community) and it is clearly not part of the aging process, there seems no justification for exclusion of these patients from cover if they have been previously asymptomatic and have a clear history of new symptom and a personal injury caused by accident.

The notion that “the accident has brought the condition to light, and the effects of the accident might now be spent leading to the underlying spondylolysis and

spondylolisthesis as being the whole or substantial cause of the symptoms or personal injury" has no support from the current quality observational literature.

Conclusion

As in all branches of medicine, and none more so than in the diagnosis and management of back pain a detailed history is fundamental to forming a diagnosis and optimising treatment. Over-reliance on modern high quality imaging increases the chances of unnecessary or inappropriate treatment. The practitioner must consider the widespread tissue changes that can occur with age.

In medicolegal decision-making where there is a need to apportion weight to the contribution of accident to subsequent symptoms or personal injury, it is essential that the decision-making relies is on quality observational population studies, rather than expert opinion that may be generated from skewed referral patterns in a previous practice life. As we have remarked before, the history of accident and the history of pre accident status must be the foundation for the correct application of the ACC Act.

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References:

1. Robertson PA, Nicholson OR. ACC and back injuries: the relevance of pre-existing asymptomatic conditions. N Z Med J. 2000;113(1102):16-9. http://www.nzmj.com/journal/113-1102/2202/content.pdf
2. Wright JG, Swiontkowski MF, Heckman JD. Introducing Levels of Evidence to The Journal. J Bone Joint Surg Am. 2003;85:1-3.
3. Beattie MJ. Burke vs ARCI Corporation, District Court Decision No 198/98; 5 August 1998.
4. Beattie MJ. Arapai vs ACC. District Court Decision No 142/2006. 7 June 2006.
5. Latremoliere A, Woolf C. Central sensitization: a generator of pain hypersensitivity by central neural plasticity. The Journal of Pain. 2009;10(9):895-926
6. Adams MA, Roughley PJ. What is intervertebral disc degeneration, and what causes it? Spine 2006;31(18):2151-61.
7. Boden SD, Davis DO, Dina TS, et al. Abnormal magnetic-resonance scans of the lumbar spine in asymptomatic subjects. A prospective investigation. The Journal of Bone and Joint Surgery. 1990;72(8):1178.
8. Jensen MC, Brant-Zawadzki MN, Obuchowski N, et al. Magnetic resonance imaging of the lumbar spine in people without back pain. N Engl J Med 1994;331:69-73.
9. Boos N, Semmer N, Elfering A, et al. Natural history of individuals with asymptomatic disc abnormalities in magnetic resonance imaging: predictors of low back pain-related medical consultation and work incapacity. Spine 2000;25(12):1484.
10. Borenstein D, O'Mara J, Boden S, et al. The value of magnetic resonance imaging of the lumbar spine to predict low-back pain in asymptomatic subjects: a seven-year follow-up study. The Journal of Bone and Joint Surgery. 2001;83(9):1306.
11. Jarvik J, Hollingworth W, Heagerty P, et al. Three-year incidence of low back pain in an initially asymptomatic cohort: clinical and imaging risk factors. Spine 2005;30(13):1541.

12. Adams MA, Hutton WC. Prolapsed intervertebral disc. A hyperflexion injury 1981 Volvo Award in Basic Science. Spine 1982;3:184-91.
13. Adams MA, Freeman BJ, Morrison HP, et al. Mechanical initiation of intervertebral disc degeneration. Spine 2000;25(13):1625-36.
14. Simunic DI, Robertson PA, Broom ND. Mechanically induced disruption of the healthy bovine intervertebral disc. Spine 2004;29(9):972-8.
15. Pezowicz CA, Schechtman H, Robertson PA, Broom ND. Mechanisms of anular failure resulting from excessive intradiscal pressure: a microstructural-micromechanical investigation. Spine 2006;31(25):2891-903.
16. Veres SP, Robertson PA, Broom ND. ISSLS prize winner: microstructure and mechanical disruption of the lumbar disc annulus: part II: how the annulus fails under hydrostatic pressure. Spine 2008;33(25):2711-20.
17. Veres SP, Robertson PA, Broom ND. The morphology of acute disc herniation: a clinically relevant model defining the role of flexion. Spine 2009;34(21):2288-96.
18. Veres SP, Robertson PA, Broom ND. The influence of torsion on disc herniation when combined with flexion. Eur Spine J. 2010 19(9) 1468-78
19. Osti OL, Vernon-Roberts B, Fraser RD. 1990 Volvo Award in experimental studies. Anulus tears and intervertebral disc degeneration. An experimental study using an animal model. Spine. 1990;15(8):762-7.
20. Don AS, Carragee EJ. Is the self-reported history accurate in patients with persistent axial pain after a motor vehicle accident? Spine J. 2009;9(1):4-12.
21. Tsirikos AI, Garrido EG. Spondylolysis and spondylolisthesis in children and adolescents. J Bone Joint Surg Br. 2010;92(6):751-9.
22. Marchetti PC, Bartolozzi P. Classification of Spondylolisthesis as a guideline for treatment. In: Bridwell KW, deWald RL, eds. The textbookof spinal surgery. Second Ed. Philadelphia: Lippincott-Raven;2005:1211-54
23. Hammerberg KW. New concepts on the pathogenesis and classification of spondylolisthesis. Spine 2005;30(6 Suppl):S4-11.
24. Wiltse LL, Widell EH Jr, Jackson DW. Fatigue fracture: the basic lesion is inthmic spondylolisthesis. J Bone Joint Surg Am. 1975;57(1):17-22.
25. O'Neill DB, Micheli LJ. Postoperative radiographic evidence for fatigue fracture as the etiology in spondylolysis. Spine 1989;14(12):1342-55.
26. Kalichman L, Kim D, Li L, et al. Spondylolysis and Spondylolisthesis - Prevalence and Association With Low Back Pain in the Adult Community-Based Population. Spine 2009;34(2):199.
27. Virta L, Ronnemmaa T, Osterman K. Low back pain in the middle-aged and elderly spondylolisthesis population. Rehabilitation Res Centre of the Social Insurance Institution, Turka and the Orthopaedic Hospital of the Invalid Foundation, Helsinki, Finland.
28. Osterman K, Schlenzka D, Poussa M, et al. Isthmic spondylolisthesis in symptomatic and asymptomatic subjects, epidemiology, and natural history with special reference to disk abnormality and mode of treatment. Clin Orthop 1993;297:65-70.
29. Hefti F, Brunazzi M, Morscher E. [Natural course in spondylolysis and spondylolisthesis]. Orthopade 1994;23:220-7.
30. Beutler WJ, Fredrickson BE, Murtland A et al. The natural history of spondylolysis and spondylolisthesis: 45-year follow-up evaluation. Spine 2003;28(10):1027-35.