



## What does degeneration mean? The use and abuse of an ambiguous word

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### Abstract

The use of the word *degeneration*, particularly in the compensation arena, is not recommended. It is imprecise and is interpreted in different ways by radiologists, clinicians and insurers. Insurers use the word to conclude that any so called *degenerative* changes mean that there is age causation so that compensation can be denied. These changes can be caused by single or multiple injuries continuing heavy work and other causes. Each risk factor should be carefully assessed in each case.

Interpretation of the imprecise and pejorative terms *degeneration* and *degenerative* can be misleading and confusing. It is often assumed by clinicians that degeneration implies an *age relation* and then some insurers make a false assumption that a statistical relation to age equates age to causation. Dorland's Medical Dictionary defines degeneration as deterioration, change from a higher to a lower form, especially to a lower or less functionally active form.

In the compensation arena it is often stated that "*the observed changes are degenerative and therefore due to age and so are not caused by injury or a gradual process from chronic overload or other possible causes.*" This can lead to inappropriate refusal of insurance entitlement.<sup>1</sup> Those with osteoarthritis (OA), spinal disc disease or tendinopathy may be refused compensation on an assumption of age causation when age is not the substantial cause or has only a minor role. All possible risk factors should be considered in assessing the causation of all musculoskeletal conditions.

When radiologists use the word *degeneration* they understand that such appearances can result from the cumulative effect of repeated minor and major impacts and physiological use, not just age.<sup>2,3</sup> Usually changes called degenerative do not cause any symptoms and so are of no clinical importance.<sup>4</sup>

Radiologists expect clinicians to assess, what they report as *degenerative* changes, together with the clinical observations, as part of the normal clinical path leading to diagnosis, but some clinicians and insurers interpret *degeneration* as meaning the ageing process. These conditions would be more precisely described as osteoarthritis for synovial (diarthrodial) joints, spondylosis for the spine and tendinopathy for the tendons as these terms do not imply causation. Insurers may still take these terms to imply age causation.

It is important to distinguish immutable risk factors such as sex, age and genetics which predispose to injury but do not cause injury. An accident or other environmental change is necessary to cause the injury.

The New Zealand Accident Compensation Corporation (ACC) often deems injuries to be aggravating factors of pre-existing *degenerative* and so age-related conditions, no matter how minor the changes. More logically, these radiological changes should be regarded as being caused by the interaction of several causes (risk factors). The effects of these are cumulative and so will increase with age. This is a statistical age relationship which does not indicate age causation. This association can be coincidental. Minor OA usually does not cause any symptoms at all.<sup>4</sup>

For example it is inappropriate that a 28-year-old nurse with 10 years exposure to heavy lifting as a nurse is described as having *degenerative changes* at one level, only but is told by her employer and/or the insurer that her pain and loss of function were due to age-related *degeneration*. More likely, lifting heavy patients and to a lesser extent gardening and playing net ball have combined to produce the lumbar disc protrusion. She was too young to have the loss of tissue resilience of later life which could lead to multi-level changes. So, the decision not to grant compensation in this case was neither logical nor just.

## Spinal disorders

In the spine *degeneration* is often used to describe loss of disc height, traction spurs and annular osteophytes. The loss of disc height puts an abnormal load on the facet joints causing secondary OA. Though there are many papers written under the title *degenerative disk disease* we have not found an explicit definition of this term. Radiological reviews of *degenerative* diseases of the spine<sup>2,3</sup> consider that the main pathogenic factor is chronic overload and that such changes may not cause symptoms. The name spondylosis is more satisfactory as it does not imply a cause. *Degenerative* changes in the posterior synovial (diarthrodial) joints are better labelled as osteoarthritis, which may not be caused by age.

Freemont<sup>5</sup> explains that changes said to be *degenerative* in the spinal discs may result from the interaction of one or more of the following risk factors:

- Diffusion of nutrients and oxygen across the inter-vertebral disc matrix
- Soluble regulators of cell function
- Mechanical load including and
  - acute, repeated and gradual process injuries
  - excessive spinal loading or obesity
- Genetic influences\*
- Ageing and senescence\*

(\*Immutable predisposing factors)

Others have suggested that micro-fractures in the subjacent bone lead to breakdown of the disc.<sup>2</sup> This suggests injury causation.

Seidler et al<sup>6</sup> found a strong dose-related relationship of cumulative physical load, lifting/carrying or extreme forward bending to lumbar spondylosis (osteocondrosis) in 229 men attending orthopaedic clinics compared with 197 controls. The same result was found for 135 cases who also had disc herniation.

Battie et al<sup>7,8</sup> in a large magnetic resonance imaging (MRI) study of twins noted that there is no agreed definition of degenerative disc degeneration. They did not state their inclusion and exclusion criteria nor did Sambrook et al<sup>9</sup> in a similar twin study.

In a more recent paper<sup>10</sup> Battie et al define degenerative disease of the spine (spondylosis) as decreased disc height and disc desiccation on MRI scan. This restricted definition precludes comparison with other studies. Battie et al<sup>7,8</sup> found that genetic predisposition had more effect than occupational workload.

Battie et al<sup>7,8</sup> did not show a relation to age for disc height narrowing, disc herniations or upper end plate changes and only showed a moderate increase for signal intensity, disc bulging, osteophytes or fatty infiltration. These MRI changes are usually described as *degenerative* but reduced disc height, disc herniations, disc bulging and end plate changes can occur in spinal injuries and chronic overload. We have not found direct evidence that age alone can cause such changes.

Using a summative “*degenerative*” scale to assess spinal MRI changes<sup>10</sup> in 120 subjects, over 40 years old, with chronic back pain, there was a relation to age and global “*degenerative*” change (disc height loss, number of narrowed discs, spinal stenosis, and spondylolisthesis). They found that physical occupational exposure, a heavier work load, pain duration and disability were associated with *degeneration*.

Some disc *degenerative* changes in the spinal discs could be more accurately labelled *internal disruption of the disc (IDD)*.<sup>11,12</sup> Annular tears usually show on MRI. Though this suggests injury causation this can be labelled “*age related degeneration*.”

Discography induces pain and indicates which disc causes the pain but this is not usually done as it is not without risk.

Schmorl’s nodes<sup>13</sup> which are heritable,<sup>14</sup> are often disregarded as irrelevant as they are usually asymptomatic but acute injuries can produce the same appearances with a break (fracture) in the end plate with herniation of disc material into the vertebral body and so can cause back pain. It is not clear whether such cases, or cases of IDD have been excluded in papers entitled *disc degeneration*. Presumably cases with nerve root involvement were excluded.

In a study of diagnostic labels and perceived diagnosis in chronic low back pain Sloan and Walsh<sup>15</sup> found that the use of degenerative terms, such as wear and tear, were associated by patients with a poor perceived prognosis.

### **Synovial joints—osteoarthritis**

Osteoarthritis of the synovial joints also results from the combination a number of causes (risk factors)<sup>17,18</sup> so it is misleading to call this *degenerative* arthritis. For example, osteoarthritis of the knee results from a combination of many causes such as:

- Fracture through joints
- Chondral injuries
- Meniscus tears
- Repeated heavy loading, prolonged bending, crouching and squatting<sup>18</sup>
- Repeated injury

- Obesity
- Knee deformity
- Inflammatory arthritis
- Heredity
- Hypermobility\*
- Some rare hereditary conditions\* and
- Sex\*

(\* Immutable factors)

The cumulative interaction of these factors determines the age of onset of symptoms and so there will be an increased prevalence and severity with age at least to retirement age. Studies will then show a statistical *relation to age* which is coincidental and so does not imply age causation. Yiuqin et al<sup>19</sup> have shown that the incidence of osteoarthritis falls after the age of 70 years.

This suggests that age alone is not an important cause. For instance knee cartilage and cruciate ligament injuries, which are so common in footballers, lead to a very high rate of secondary osteoarthritis.<sup>20</sup> The prevalence of this will increase with age though it is clearly not caused by age.

Linear (bucket handle) tears in the knee menisci result from acute injuries. More complex partial thickness meniscus tearing has been attributed to “*degeneration*” and so to age but this could also be due to chronic overload or repeated injuries.

### **Tendon disorders—tendinopathy**

Tendon disorders are often described as *degenerative* though there is no evidence of age causation. This applies to the tendons at the wrist, ankle, hip, rotator cuff tendons and elbow. Tendinopathy of the extensor origin tendons at the elbow (epicondylitis) is common. The suffix *itis* suggests inflammation which is usually not evident so the term tendinopathy is preferred as this does not imply inflammation. If the synovial sheath of the tendon is inflamed the name tenosynovitis is appropriate.

Repetitive tendon overload in athletes causes tendinopathy and sudden excess load can rupture the tendon. Age-related muscle atrophy is associated with elasticity changes in the tendon<sup>21</sup> implying greater susceptibility to injury.

The alleged *degenerative* changes in tendons may be caused by a combination of factors such as:

- Repeated overload
- Single injuries
- Multiple injuries
- Sport injuries
- Vibration
- Obesity<sup>22</sup>

- Cefloxacin
- Age (immutable)
- Genetic<sup>2,3</sup>

Tendinopathy may be found in the absence of symptoms in the shoulder. The study by Allander<sup>24</sup> showed a decrease in the prevalence and incidence of shoulder pain and epicondylitis past the age of sixty. This is contrary to expectation if age was the main cause and suggests that occupation, and/or the other factors listed above are the explanation.

## The rotator cuff

Acute injuries and sustained overloads can cause partial or complete tears of the rotator cuff tendon without causing symptoms. Again a relationship to age may be assumed to imply age causation without considering the alternative causes.

In a 20-year prospective study of 883 asymptomatic subjects,<sup>25</sup> 63 developed chronic shoulder disorders. Work exposure to repetitive shoulder movements increased the risk (odds ratio [OR] 2.3) and vibration (OR 2.5) of developing shoulder disorders. For three of these risks, lifting heavy loads and working in awkward postures the risk increased to an odds ratio (OR) of 4. *“The effects seem to be long-term so that the accumulation of damage in shoulder tissues can be seen several years after work life has ended.”*

Age relationship was only significant for women and only body mass for men. Highly repetitive arm activity and sustained 60 degrees flexion or abduction can cause rotator cuff injuries.<sup>26</sup> In asymptomatic volunteers<sup>27</sup> full thickness rotator cuff tears increased with age up to 50 years but did not increase past 50. Shoulder tendinitis was more common in bricklayers, rock blasters and with those with vibration exposure compared to foremen.<sup>28</sup>

Similarly tendinopathy and/or rupture of the Achilles and other tendons can be caused by acute injuries and repeated overload in sport and similar occupational activities.<sup>29</sup>

## Conclusion

Radiologists, clinicians and insurers frequently put different interpretations on the word *degeneration* leading to confusion. It is suggested that this ambiguous word should be abandoned and replaced by osteoarthritis, spondylosis and tendinopathy as these terms do not imply causation. This would prevent false assumptions of age causation leading to flawed legal decisions in the New Zealand environment hindering early rehabilitation to the disadvantage of the patient. The various risk factors for each disorder should be carefully assessed in each case. Prevention, control strategies and compensation decisions would then be more logically based.

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## References:

1. Wigley RD. What is degeneration? The misuse of an ambiguous word [letter]. *N Z Med J.* 2009;122(1303). <http://www.nzmj.com/journal/122-1303/3808/>
2. Galluci M, Linbucci N, Paonessa A, Splendiani A. Degenerative disease of the spine. *Neuro-imageing clinics of North America.* 2007;87-103.
3. Internal derangement of Joints. Resnick D, Kang S, Pretterklieber M. *Synovial joints: Degenerative disorders.* Saunders Elsevier. 2007. Chapter 6.
4. Robertson P, Nicholson R. ACC and back injuries: The relevance of pre-existing asymptomatic conditions. *NZ Med J.* 2000;13:16-19.
5. Freemont AJ. The cellular pathology of the degenerative intervertebral disc and discogenic pain. *Rheumatology.* 2009;48:5-10.
6. Siedler A, Bolm-Audorff U, Heiskel H, et al. The role of cumulative physical work load in lumbar spine disease: risk factors for lumbar osteochondrosis and spondylosis associated with chronic complaints. *Occup Environ Med.* 2001;58:735-746.
7. Battie MC, Videman T, Parent E. Lumbar disc degeneration. Epidemiology and genetic influences. *Spine* 2004;23:2679-2690.
8. Battie MC, Videman T, Kaprio J, et al. The twin spine study: contributions to a changing view of disc degeneration. *Spine J.* 2009;9:47-59.
9. Sambrook PN, MacGregor AJ, Spector TD. Genetic influences and lumbar disc degeneration: a magnetic resonance imaging study in twins. *Arth & Rheum.* 1999;42:366-372.
10. Mariconda M, Galasso O, Imbimbo L, et al. Relationship between alterations in the lumbar spine, visualised with magnetic resonance imaging, and occupational variables. *Euro Spine J.* 2008;33:255-266.
11. Crock HV. Internal disc disruption. *Spine* 1985;11:650-653.
12. Karasek M, Bogduk N. Twelve month follow-up of a controlled trial of intradiscal thermal annuloplasty for back pain due to internal disc disruption. *Spine.* 2000;25:2601-2607.
13. Pifirrmann CWA, Resnick D. Schmorl's nodes of the thoracic and lumbar spine. Radiographic-pathologic study of prevalence, characterisation, and correlation with degenerative changes of 1650 spinal levels in 100 cadavers. *Radiology.* 2001;219:368-374.
14. Williams FM, Manek NJ, Sambrook PN, et al. Schmorl's nodes : common, highly heritable and related to lumbar disc disease. *Arth & Rheum.* 2007;57:855-860.
15. Sloan TJ, Walsh DA. Explanatory and diagnostic labels and perceived prognosis in chronic low back pain. *Spine.* 2010.35:E1120-1125.
16. McMillan G, Nichols L. Osteoarthritis and meniscus disorders of the knee as occupational diseases of miners. *Occup Environ Med.* 2005;62:567-575.
17. Jensen LK. Knee osteoarthritis. Influence of work involving heavy lifting, kneeling, climbing stairs or ladders or kneeling/squatting combined with heavy lifting. *Occup Environ Med.* 2008;65:72-89.
18. Dahaghin S, Tehrain-Banishashemi A Faezi FT, Davatchi F. Squatting, sitting on the floor, or cycling: Are life-long daily activities for clinical osteoarthritis? Stage III results of a community based study. *Arth & Rheum.* 2009;61:1337-1342.
19. Yiuqing Z, Jordan JM. Epidemiology of osteoarthritis. *Rheum Dis Clin North America.* 2008;34:515-529.
20. Lohmander LS, Eglund PM, Roos EM. The long term consequence of anterior cruciate and meniscus injuries: osteoarthritis. *Am J Sports Med.* 2007;35:1756-1769.
21. Reeves ND, Narici MV, Maganaris CN. Myotendinous plasticity to ageing and resistance to exercise in humans. *Exp Physiol.* 2006;91:483-498.
22. Roquelaure Y, Catherine HA Rouillon C, et al. Risk factors for upper-extremity musculo-skeletal disorders in the working population. *Arth Care & Research.* 2009;61:1425-1434.

23. Posthumus M, Collins M, Cook J, et al. Components of the transforming growth factor beta family and the pathogenesis of human Achilles tendon pathology – a genetic association study. *Rheumatology* 2010;2090-2097.
24. Allander E. Prevalence, incidence and remission rates of some common rheumatic diseases or syndromes. *Scand J Rheumatol.* 1974;3:145-153.
25. Miranda H, Punnett L, Vikari-Juntura E, et al. Physical work and chronic shoulder disorder. Results of a prospective population-based study. *Ann Rheum Dis* 2008;67:218-223.
26. Musculo-skeletal disorders and work place factors. A critical review of epidemiological evidence of work related musculoskeletal disorders of the neck, upper extremity and low back. Ed Bernard BP. 1997. (NIOSH publication 97-141) Chapter 3.
27. Worland RL, Lee D, Orozco CG, et al. Correlation of age, acromial morphology, and rotator cuff tear pathology diagnosed by ultrasound in asymptomatic patients. *J Southern Orth Assoc.* 2003;12:2328.
28. Stenlund B, Goldie I, Hagberg M, Hogstedt C. Shoulder tendinitis and its relation to heavy manual work and exposure to vibration. *Scand J Environ Health.* 1993;19:42-49.
29. Biundo JJ, Irwin IW, Umpierre E. Sports and other soft tissue injuries, tendinitis, bursitis and occupation-related syndromes. *Curr Opin Rheumatol.* 2001;13:146-149.