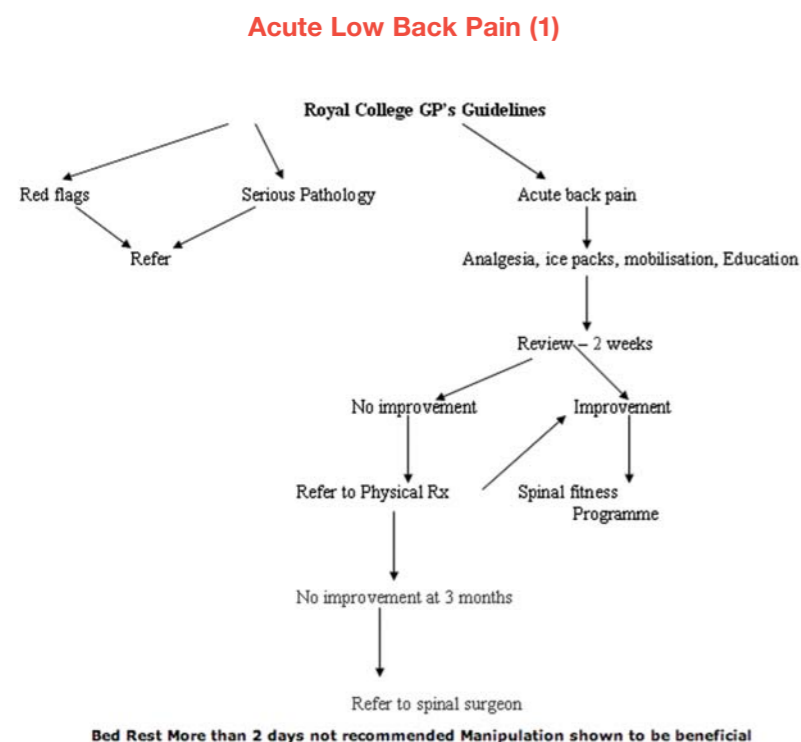


Referral Guidelines



Contact Information:

Mr M Krishna

FRCS, MCh(Orth). Spinal Surgeon.
kspine@gmail.com

NHS Practice

NHS Secretary
Tel 01642 624193
Fax 01642 624814

Email amanda.hardey@nth.nhs.uk
Address for Referrals

Spinal Unit, University Hospital of North Tees,
Hardwick Road, Stockton-on-Tees TS19 8PE

Private Practices

Private Secretary
Tel 07813 568585
Fax 01642 889564

Email spinesec@gmail.com

Addresses for Referrals

Nuffield Health Tees Hospital, Junction Road,
Norton, Stockton-on-Tees TS20 1PX

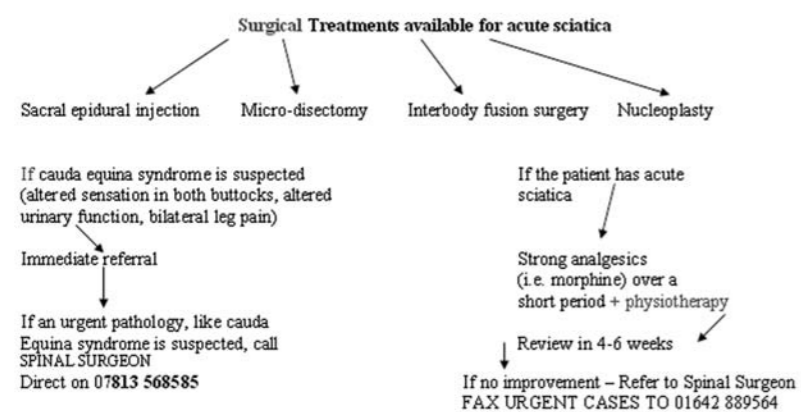
Nuffield Health York Hospital, Haxby Road,
York, YO31 8TA

Consulting Hospitals:

Nuffield Hospital Tees – 01642 360100
Nuffield Hospital York – 01904 715000

Leg Pain/Sciatica (2)

Altered sensation in the leg with a slight degree of weakness in the foot is not an indication for an urgent referral.



DO NOT SEND URGENT REFERRALS THROUGH THE POST

In brief



Manoj Krishna has been a consultant spinal surgeon for 14 years and works at the University Hospital of North Tees, in Stockton-on-Tees, and at the Nuffield hospitals in Stockton and York.

He trained in spinal surgery with spinal surgery fellowships in Brisbane, Hong Kong and Nottingham, having done his orthopaedic training in Pune, India, and Merseyside and Wales in the UK.

He performs over 300 major spinal operations a year. He treats the full range of adult spinal problems including neck, thoracic and lumbar pathology.



Focus on Pathophysiology of Back Pain

There have been recent advances in our understanding of the pathophysiology of back pain which covers anatomy of the neural system and disc degeneration, the biomechanics of loading of the spine, and the role of Inflammatory mediators in the disc space. Combined together they have influenced the way we deal with back pain surgically, and improved our results of surgery.

Disc Degeneration, whats new? (Figure 1)

Cadaver studies have shown the growth of granulation tissue into the posterior part of the disc. This neo-vascular growth is accompanied by new nerve endings. Loading of this area causes pain.

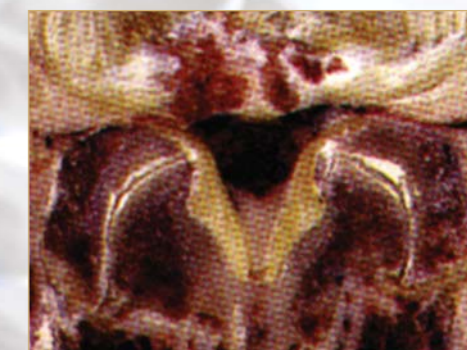


figure 1

Neural Anatomy (Figure 2)

We now understand that the sympathetic plexus plays a significant role in the innervation of the spine and the fibres travel along the side of the spine and enter the spinal column via the L2 Dorsal Root Ganglion. This rich sympathetic innervation may explain the link between back pain and depression, as well as the role of the patient's personality in the experience of back pain.

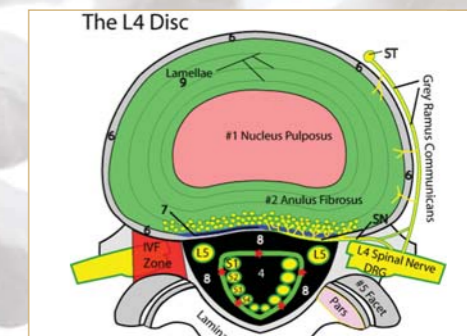


figure 2

We also now know that the sinuvertebral nerve innervates the posterior third of the disc space, and then joins the dorsal root ganglion at that level, and connecting from that to the main axial nerve root. This may explain why disc degeneration can cause leg pain, even though the MRI scan shows no neural compression. I have several patients now whose main presentation was with leg pain, though the MRI showed disc degeneration without neural compression. An interbody fusion at this level sorted it out.

Biomechanical Studies (Table 1)

Sato et al published a paper in SPINE in 1999, where they put a pressure sensor into the disc and measured loading across the disc in different postions. As the table clearly shows there is more than a 10 fold increase in the loading across a disc between lying down and sitting in flexion. There is thus a strong clinical correlation with the sitting pain that patients with mechanical back pain experience, which is eased on lying down. In terms of surgery, any procedure that restores loading across the disc by putting something in the disc space (Interbody fusion, Disc Replacement) has a higher chance of an excellent result compared to procedures that don't do this (Discectomy, Postero-lateral fusion). We also know that discs are not able to tolerate torsional stresses as well they tolerate axial loading. This may explain the pain that patients get at night on turning in bed, or the initial history of twisting as being a major triggering factor in development of back pain.

Continued overleaf

Loading through the L45 disc (kPa),
Measured using pressure sensors.

Prone	91
Standing - Upright	539
Standing - Flexion	1324
Standing - Extension	600
Sitting - Upright	623
Sitting - Flexion	1127
Sitting Extension	737

Sato et al, SPINE, 1999

table 1



MANOJ KRISHNA FRCS, MCh(Orth)

CONSULTANT ORTHOPAEDIC AND SPINAL SURGEON

“Physio - Heal Thyself!”



A fully fit Gavin

Gavin Cree, a physiotherapist from Newcastle, describes his journey from severe back pain, depression, to cycling 100 miles a day.

“Eight years ago I “caught” a 25 stone patient while doing a physiotherapy home visit and knew immediately that I had done something pretty bad to my back, however, I wasn’t ready for the rollercoaster of scans and surgery I was about to embark on. The first surgery was unavoidable with the MRI showing a prolapse at L3L4, L4L5 and L5S1. The nerve root pain was unbearable with weakness and loss of reflexes in my right leg . I was operated on by a neurosurgeon. He performed a micro discectomy at L4L5 and L5S1 and I began the slow road back to recovery.

I embarked on the usual lumbar back exercises and core stabilising work I could manage. I improved, but 18 months later was I was back to square one! I went back to the neurosurgeon for more decompression surgery. At this point I was told I would have to retire which was a pretty unpalatable given I was only 38!

Again, I improved, but by the time another 18 months had past I had deteriorated again. Most days I couldn’t get out of bed without being sick with the pain. Ridiculous levels of Gabapentin, Tramadol, Diclofenac, Codeine and Diazepam weren’t helping and I was looking down the barrel of losing my business and my sanity.

I was then referred to see Mr Krishna. He suggested stabilising L4L5 and L5S1 with a Posterior Lumbar Interbody Fusion (PLIF) as well as putting in a small hinge in the stabilization at L3L4 to decrease the chance of too much load on the levels above the stabilization. A disco gram was done initially to confirm the levels causing my symptoms and then on to the surgery.

I needed a day in intensive care but even waking up there I knew something was different. Suddenly there was stability in my spine, a sensation I hadn’t felt for nearly five years!

As my stamina improved so I was able to stop all of my medication which was one of the most liberating moments of the entire process. Never wanting to go back into that drug fueled depression was all the motivation I needed to keep going with my rehabilitation.

In amongst all the rehabilitation I was back at work running my own practice no longer contemplating retirement but looking forwards to working for years to come as the Physio I knew I was capable of being . And here I am today, writing this , sitting on a train with three friends going to Aberdeen with our bikes to spend the next three days cycling back home to Newcastle. If you had offered me that four years ago before I met Manoj I would have probably pulled the covers back over my head and reached for the Gabapentin!”

Mr Krishna comments, ‘Gavins story illustrates how the advances in basic science have resulted in improved surgical results. the key here is the removal of the painful disc, restoring loading by doing an interbody fusion , and then intensive rehabilitation.’

To read this story in full, and for more information on the rehabilitation programme Gavin devised for himself, visit www.kspine.net



Pre-op MRI Scan



Post-op lateral X-ray

Continued from front page

Inflammatory Mediators (Figure 3)

Research suggests that mechanical and chemical causes, acting together, may play a role in the production of mechanical LBP. Components of the nucleus pulposus, most notably the enzyme phospholipase A2 (PLA2), have been identified in herniated disc material. This PLA2 may act directly on neural tissue, or it may orchestrate a complex inflammatory response that manifests as LBP.

Glutamate, a neuroexcitatory transmitter, has been identified in degenerated disk proteoglycan and has been found to diffuse to the dorsal root ganglion (DRG) affecting glutamate receptors. Substance P (pain) is present in different neurons, including the DRG, and is released in response to noxious stimuli, such as mechanical compression of the nerve. The breakdown of the annular fibers allows PLA2 and glutamate, and possibly other as-yet unknown compounds, to leak into the epidural space and diffuse to the DRG. Injury to a disc may stimulate the release of substance P. Substance P, in turn, stimulates histamine and leukotriene release, leading to an altering of nerve impulse transmission. This may explain why some patients have severe leg pain, but no evidence of neural compression on an MRI scan. Leakage of chemicals from a degenerate disc on to the neural structures may cause neural irritation.

Muscle wasting and reaction time (Figure 4 and 5)

Research has shown that after an acute episode of back pain, the erector spinae muscles waste and weaken by up to 15%. This is not visible or noticed by the patient. It does however set up a cycle where the spine becomes more prone to subsequent attacks of back pain. It is vital therefore that patients do a spinal fitness program to reverse this process (available on www.kspine.net).

Figure 4 is a CT scan through healthy spinal muscles and Figure 5 through unhealthy muscles in a patient with back pain, showing a large amount of fat between the muscles bellies. The reaction time of the trunk muscle stabilizers is also reduced in patients with chronic low back pain by about 0.2 milliseconds. These trunk muscles are responsible for stabilizing the trunk. This may explain why patients with back pain report a slight twisting action (to bend and pick up something), can leave them on the floor. Muscle research has made Spinal Rehabilitation Programs more effective. These now focus on core stability, aerobic fitness, flexibility, endurance time, reaction time of muscles and progress from static to dynamic rehabilitation.

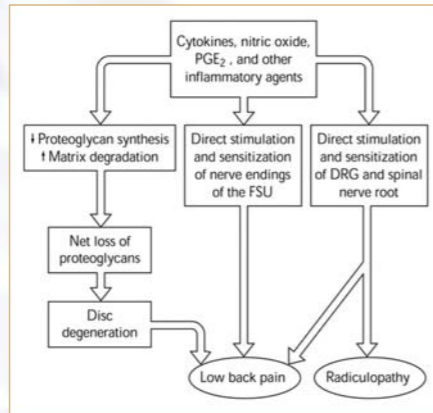


figure 3



figure 4



figure 5

Click onto our new website and see the difference. We have upgraded our website to make it easier, quicker and more informative.

It is bursting with advice, help and numerous patient stories. The site can be visited by yourself, as a fellow professional or there for you to refer patients to, or both!

www.kspine.net