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## Antipsychotic prescribing in nursing homes

### We need to understand why this practice continues despite the mortality risk

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Warnings about an increased risk of death in patients with dementia who receive atypical antipsychotics were first issued in 2004-5, after the publication of a meta-analysis of data from placebo controlled randomised controlled trials.[1 2] In 2008 analysis of databases led to similar warnings for older typical antipsychotics. At the time, inconclusive evidence suggested that the risk of death was greater for typical than for atypical antipsychotics.[3] The linked paper by Huybrechts and colleagues adds to the evidence on differential risk.[4] Using risperidone—the antipsychotic most widely prescribed for patients with dementia—as the comparator, they report an increased risk of mortality in nursing home residents taking haloperidol and a decreased risk in those prescribed quetiapine. Causation is not definitively proved but seems highly probable given the strength of the association that unmeasured confounders would need to have with both the use of haloperidol (or quetiapine) and mortality to account for the result.

**Few clinical problems place doctors in as tangled a web of clinical evidence, social policy, and ethical concerns as how to manage behavioural problems in patients with dementia**

Information on risk must be weighed against the potential benefits of a drug. Although meta-analyses have shown a small benefit for haloperidol on aggression in dementia there is no evidence that the benefit is greater than that for risperidone,[5] which seemed to be less harmful in the current study. This strengthens the argument for avoiding haloperidol on safety grounds. In contrast, there is no high quality evidence that quetiapine is effective for treating neuropsychiatric symptoms in dementia,[6 7] and the results of the current study should not support its use.

The use of any antipsychotic in dementia is undesirable given the increased risk of death and the many other adverse effects of these drugs, in addition to their limited efficacy against target behavioural and psychological symptoms.[7] Evidence on comparative safety must be evaluated in this light, but it is nevertheless important to extend our knowledge of the comparative efficacy and safety of antipsychotics for two reasons.

Firstly, although guidelines universally agree that the first line treatment for behavioural and psychological symptoms in dementia should be non-drug based, they also—in the absence of evidence for greater efficacy of other drugs—recommend the careful use of antipsychotics in the treatment of agitation, aggression, or psychosis that fails to respond to other measures and that reaches various severity thresholds, typically severe distress or serious risk to self or others.[8 9] Secondly, despite widely disseminated guidance aimed at limiting their use, antipsychotics are still widely prescribed to older people with dementia or in institutional care. Although published data typically lag behind what is happening in practice by several years, as an example, analysis of a primary care database covering England and Wales for 2008-9 found that 18.2% of patients in care homes were prescribed antipsychotic drugs. Among patients with a recorded diagnosis of dementia, 10.1% in the community and 30.2% in care homes received such drugs.[10] Clearly, doctors find compelling reasons to prescribe antipsychotics to patients with dementia, reasons that are unlikely to be found in the evidence base alone.

Few clinical problems place doctors in as tangled a web of clinical evidence, social policy, and ethical concerns as how to manage behavioural problems in patients with dementia. Many studies are now describing the demographic and institutional factors associated with the prescribing of antipsychotics. A complementary approach is to try to understand prescribing practice at the level of physician behaviour. A small qualitative study among psychiatrists in the north of England into prescribing for behavioural and psychological symptoms in dementia, although local, identifies themes with which many primary care doctors and psychiatrists will identify.[11] These include feeling pressurised to prescribe, believing that non-drug based approaches are unfeasible because of lack of resources and difficulties of implementation, and perceiving a failure at the societal level to provide the environment and resources needed for high quality innovative care. These could be seen as negative attitudes; equally, they may reflect the reality of the situations in which most dementia care occurs. Where care homes or community care services are inadequate and local clinical resources cannot compensate for them, doctors face genuine dilemmas about how to respond to distressed patients, relatives, and carers, often in ethically complex situations that involve a variety of risks. It is probably fair to say that many doctors think that the evidence based guidelines are not adequate for the day to day reality of practice.

Future research should be pragmatic. It should focus on identifying the key components of non-drug based interventions and on establishing the service structures that can deliver them as simply and efficiently as possible. Although international comparisons may be useful, much research of this nature will need to look for solutions that are compatible with local conditions. More locally based explanatory research into prescribing patterns will help inform service development. Continued debate on the ethical framework of dementia care, such as recent discussions on the usefulness of a palliative care model, should be encouraged.[12]

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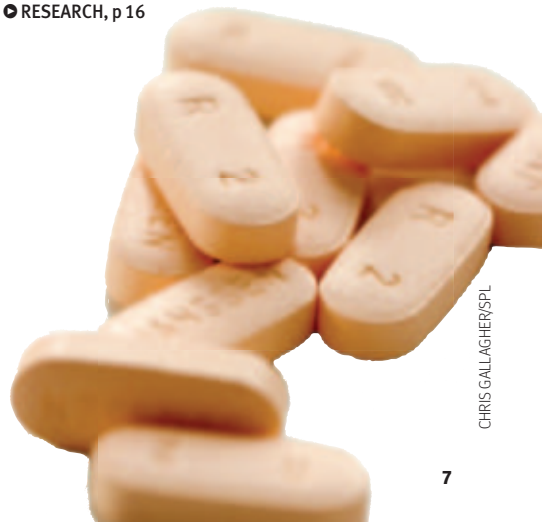
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**bmj.com/blogs**
- Helen Macdonald: Dangerous weekends—more complicated than just a lack of consultants
- Ken Taylor: Dr Foster on inpatient hospital mortality

# Higher senior staffing levels at weekends and reduced mortality

## The association is clear but the effects of the grade and specialty of key personnel are not

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Dr Foster Intelligence recently published a report that shows a clear association between reduced numbers of senior doctors in hospitals in the United Kingdom and increased mortality at the weekend.[1] How should patients, doctors, and commissioners interpret this finding and what can be done to improve patient outcomes at the weekend?

In the Western world mortality is 10% higher in patients admitted to acute hospitals at the weekend than during the week.[2 3] Medical conditions, especially cancer and cardiovascular disease,[2] account for most of this excess mortality, but increased weekend mortality has also been shown for surgical diagnoses such as ruptured aortic aneurysm.

The Dr Foster report compared hospital standardised mortality ratios (HSMRs) for patients admitted to English hospital trusts on two weekends in April 2011 with those admitted in the week. They then used self reported data on staffing levels from hospitals to assess the effect of numbers of doctors—both resident and on-call—on weekend mortality. Data were collected on all grades of doctor (and nurses), but interestingly only the number of senior doctors (registrars and consultants) was associated with a difference in weekend mortality.

The hypothesis that early assessment and intervention by experienced clinicians result in improved weekday mortality seems to be a "no brainer," and patient and professional organisations have called for seven day working to be put in place in all hospitals.[4] Lower levels of medical staffing in UK hospitals at nights and weekends are well documented,[5] and a large geographical variation exists. Indeed, the preponderance of hospitals with increased weekend mortality in the north of England is striking. However, low staffing levels are only one of the factors that potentially explain increased mortality in patients admitted at the weekend.

Evidence indicates that patients admitted at the weekend are sicker than those admitted during the week, and hospital coding (on which HSMRs rely) is not sensitive enough to allow correction for this. However, studies in which severity scores have been measured show that differences in mortality between the weekend and week disappear for many conditions when the severity of those conditions is adjusted for.[6 7] HSMRs have been shown to be highly sensitive to the variability of the coding process, and many clinicians do not trust them. Future measures of mortality need to be accurate and consider severity as well as comorbidity. Equally, clinicians need to recognise the importance of accurate coding.

Although patients with certain conditions—such as trauma, alcohol associated conditions, and self harm—are more often admitted at the weekend, the main reason that sicker patients are admitted at the weekend is variation in referral practice. Out of hours primary healthcare services have changed dramatically over the past decade in the UK, with increasing reliance on "emergency medical services" rather than patients' own general practitioner. A better understanding of referral practice and medical staffing in the community would be useful when considering variation in mortality between hospitals. This offers a key improvement opportunity for the developing clinical commissioning groups in England.

Provision of hospital support services is reduced at the weekend, so fewer interventional procedures, are performed.[8 9] Increased mortality has clearly been associated with reduced provision of percutaneous coronary intervention at the weekend in parts of the United States.[8]

The provision of full support services is challenging for many hospitals, both in terms of the workforce and finances (especially in the current economic climate). This has led to the call for the creation of networks through rationalisation of services in parts of the UK. The Dr Foster report shows a reduction in HSMR for stroke in London since the formation of such a network, as well as lower weekend mortality.[1] London is unique in the number of large and small hospitals that are within close proximity to one another, but in many other areas the network model for trauma, vascular surgery, and stroke is well advanced. How successful such networks will be in terms of patient outcome, safety, and cost effectiveness remains to be seen.

Even when these other contributory factors are considered, the observation of increased mortality and low staffing levels cannot be discounted and poses a serious problem for the NHS. The process of increasing the number of doctors within the UK, especially senior doctors, is slow and expensive. Short term increases using doctors from outside the UK will probably have an effect on healthcare workforce planning in other European Union countries.[10] It is unclear which specialties and grades of doctor need to be increased in number for weekend mortality to be reduced. Future studies need to investigate this question.

Continuity of care must be maintained when remodelling weekend staffing. Emerging data show that working patterns for consultants influence mortality. Hospitals in which the admitting consultants work blocks of more than one day have lower excess weekend mortality than those with a "physician of the day" model (D Bell, personal communication, 2012).

Interestingly, Dr Foster defined first year registrars as senior doctors in their report,[1] and although this may be debated by some, it shows the importance of registrars in the provision of out of hours services. Indeed, the number of medical registrars (who run most hospitals at night) could easily be the defining predictor of hospital mortality. The current plan for the UK is to reduce the number of registrar posts in both surgery and medicine.[11] This may need to be re-thought but, given the potential profound impact on clinical outcomes, decisions must be based on sound evidence.

The Dr Foster report raises more questions than it answers. Hospitals and commissioners, with their clinicians, need a better understanding of the potential factors that cause higher mortality at the weekend. Among these—and with profound implications for planning—are community out of hours services, hospital staffing, and workforce configuration. All need to be reviewed against the knowledge of which conditions are associated with increased mortality at the weekend. This is an opportunity that, if tackled intelligently, will improve the care of some of our sickest patients for many years to come.

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News: Protective effects of antiretrovirals are confirmed in community setting (*BMJ* 2012;344:e1789)

# Antiretroviral therapy and sexually transmitted HIV infection

## Early treatment in infected partners reduces transmission and improves clinical outcomes

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Antiretroviral drugs have been used to prevent the transmission of HIV since the 1990s. The demonstration that zidovudine decreased the risk of HIV infection after percutaneous exposure,[1] and success in prevention of mother to child transmission,[2] have led to antiretrovirals being used for HIV prevention. However, transmission of HIV mostly occurs during sexual intercourse, and successes in this area have lagged behind. Prophylaxis after sexual exposure, although an established clinical practice,[3] has not been evaluated in a randomised controlled trial. More recently, large well conducted trials demonstrated the effectiveness of pre-exposure prophylaxis of uninfected people, using tenofovir and emtricitabine or 1% tenofovir vaginal gel.[4 5]

Cohort studies suggested as early as 1994 that treating HIV infected patients with antiretroviral therapy (ART) would decrease the likelihood of them infecting their sexual partners. Seven cohort studies have examined this question in discordant couples, in which one sexual partner is infected with HIV and the other is not. A systematic review found that these studies had a pooled relative effect of 0.34 (95% confidence interval 0.13 to 0.92), meaning that uninfected sexual partners of people taking ART had a 66% lower risk of becoming infected than those whose partners were not taking ART.[6] Another important early observation was that the risk of HIV transmission in untreated discordant couples is directly associated with the infected partner's serum viral load, which suggests that suppression of viral replication using ART could decrease infectiousness.[7]

Earlier this year, data from a large randomised controlled trial on the effect of ART on sexual transmission of HIV in discordant couples was published.[8] On 28 April 2011, the data safety and monitoring board for the HIV Prevention Trials Network (HPTN) Study 052 recommended release of study results.[7] The trial examined 1763 HIV discordant couples in nine countries; the infected partner in each couple had a CD4 cell count of 350-550×$10^6$ cells/L on entry and had not previously received ART, except for short term prevention of mother to child transmission

DIETER TELEMANS/PANOS

**A patient receiving ART**

of HIV. Participants were randomised into two arms: an early treatment arm in which infected partners began ART at study entry (median CD4 count 442×$10^6$ cells/L) and a delayed treatment arm in which infected partners began ART when their CD4 cell counts fell below 250×$10^6$ cells/L or they developed symptoms of advanced HIV disease. Thirty nine new infections were noted, four in the early treatment arm and 35 in the delayed treatment arm. Because the investigators used molecular virological techniques to link HIV strains between partners, they were able to show that only one of the transmissions in the early treatment arm and 28 in the delayed treatment arm could be linked to their partners' strains, indicating that the other infections were acquired from people outside of the study partnership. Thus, using virologically linked transmission as the end point, the relative risk of transmission in the early treatment group compared with the delayed treatment arm was 0.04 (0.01 to 0.28). In addition, the trial showed that patients in the early treatment arm had 40% fewer clinical end points, such as death, progression to World Health Organization stage 4 disease, severe bacterial infections, or pulmonary tuberculosis; this difference was most pronounced in patients who developed pulmonary tuberculosis. Moreover, the number of grade 3-4 clinical adverse events was evenly distributed between arms, although more laboratory adverse events were seen in the early

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treatment arm, as would be expected with longer exposure to antiretrovirals.

How should these results be used in clinical practice to balance the benefits of decreased infectiousness and improved clinical outcomes against the risks of cumulative toxicity and earlier development of antiretroviral resistance? Large cohort studies and data from the HPTN 052 study suggest that patients treated earlier in the course of disease do better and have fewer complications of chronic HIV infection than those treated later.[9 10] In low income and middle income countries, this new indication for ART offers a powerful new strategy for preventing transmission, especially in discordant couples, which in some countries with mature generalised epidemics account for most incident infections.[11] Couples, rather than individuals, will need to be tested to identify those most likely to benefit from this strategy. On the other hand, the World Health Organization's recent recommendation to begin ART when CD4 counts are at 350×$10^6$ cells/L, although clearly clinically important, has created a new burden on the procurement and flow of antiretrovirals, and concern exists about adding another indication while not everyone who has clinical indications for ART is being treated.

Clinical experience and observational studies, rather than additional large randomised controlled trials, will probably be able to inform these uncertainties. Understanding how to weigh toxicity, resistance, cost, and long term adherence against clinical benefit and decreased transmission will require long term cohort studies and the data from clinical registers. Nonetheless, HPTN 052 has made a historic contribution to our knowledge of how to prevent sexual transmission of HIV, and its results need to be translated rapidly into practice.

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# Tic disorders

## New European guidelines highlight best practice in diagnosis and management

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The European clinical guidelines for Tourette's syndrome and other tic disorders were recently compiled and published by the European Society for the Study of Tourette Syndrome.[1-5] The take home message of the guidelines is that tic disorders are common and complex neuropsychiatric conditions. Practising clinicians need to work together with medical specialists, parents, teachers, peers, and advocacy groups to disseminate knowledge and implement effective interventions.[2 3] In addition, despite recent scientific advances, more effort is needed to understand and treat these neurodevelopmental disorders.

Tic disorders start in childhood and are characterised by multiple sudden, rapid, recurrent, and non-rhythmic movements (motor tics) or utterances (vocal tics), or both. The best studied chronic tic disorder is Tourette's syndrome, which has a prevalence of 0.3-1% in the general paediatric population. The syndrome is characterised by multiple motor tics as well as one or more vocal tics over a period of more than one year.[6]

The phenomenology and natural course of tic disorders are complex. Although some practitioners try to reassure families that their child will "outgrow" their tics, the long term course is variable. Tics may improve, but usually the tics are just one part of a larger neuropsychiatric syndrome. Specifically, the guidelines note that children with Tourette's syndrome often function poorly across numerous psychosocial domains and have high rates of hyperkinetic disorder: attention-deficit/hyperactivity disorder, obsessive-compulsive disorder, mood and anxiety disorders, learning disorders, and autism spectrum disorders.[2] Tics and their associated neuropsychiatric symptoms can negatively affect patients' quality of life, social and academic functioning, and lifetime achievements. Tics and related symptoms can be extremely troubling to the patient's family, and the entire family often needs care and counselling. At the time of evaluation, the child may be upset by his or her inability to control the tics and by criticism from parents, teachers, and peers who exhort him or her to control this strange behaviour, which they may believe the child can do.

Evaluation of a child with a tic disorder should include clarifying and dealing with family problems, such as parental guilt and misconceptions. Then, as the guidelines highlight, the central task is to educate everyone involved in the child's care.[4] To do this, clinicians first need to educate themselves (box).

Although the family may focus on the upsetting and socially stigmatising tics, the clinician needs to place the tics into the context of overall development so that the child's development is kept on track. This often involves refocusing the family's attention away from the tics and helping them find ways to build on the child's strengths and abilities.

Incremental progress in the study of this syndrome continues across a range of fields including phenomenology, natural course of the disorder, genetics, neurobiology, and treatment. Although the causes are heterogeneous, recent preliminary genetic studies have identified two genes that may contribute to the development of Tourette's syndrome. For example, in a two generation family with nine affected members, the gene that encodes the limiting step in histamine biosynthesis was non-functional.[7] This suggests that histaminergic neurotransmission may be a key factor for some families. Modulation of this system might be beneficial more broadly, not just to members of this family.[7]

Findings from postmortem brain studies may provide a deeper understanding of how the selective loss of striatal interneurones might contribute to the pathobiology of the syndrome.[8] Such studies generally indicate abnormalities in the basal ganglia circuitry but also suggest alterations in other brain regions and large scale brain networks.[9 10] Longitudinal studies are needed to make more informed interpretations of neuroimaging results.[11]

If tic suppressing drugs are needed, a two tier approach and monotherapy constitute the best practice. First tier drugs, notably α adrenergic agonists, are recommended for people with both tics and attention-deficit/hyperactivity disorder. Second tier drugs include various typical and atypical neuroleptics. However, few studies have compared the short term or long term efficacy and safety of different psychopharmacological agents, so no drug has been proved to be superior.[2] New agents are needed given the limited benefit and potential side effects, such as weight gain associated with the use of some neuroleptics. A randomised trial has shown that behavioural interventions such as comprehensive behavioural treatment for tics, exposure and response prevention, and parent management training and anger control training may help reduce tic severity, obsessive-compulsive behaviour, and disruptive and oppositional behaviours, respectively.[12] Depending on the resources available, it may therefore be sensible to consider beginning with behavioural treatment. However, such interventions require motivation from the patient and an ability for introspection, which may limit their use in younger patients.[4 5]

Anti-tic drugs that work quickly, effectively, and with few side effects are clearly needed. For interventions currently in use, it will be important to determine the relative long term benefits of behavioural interventions when combined with drugs. Other emerging areas of research concern brain morphometry and immature and anomalous patterns of functional connectivity of various brain regions. Longitudinal studies of the trajectories of brain growth are currently under way to discover how the structure of the brain may change over time. Repeated functional magnetic resonance imaging studies are also needed to determine how the regional connectivity may change depending on the course and outcome of the disorder. With regard to prognosis, why are deficits in fine motor skill in childhood modestly associated with more severe tics in adulthood? Another key question concerns why tic symptoms improve or stop completely by early adulthood in most cases but persist and worsen in a small number of patients. Other basic questions involve the heterogeneous causes of tic disorders. For example, genetic studies of rare variants have led to promising, but at times, controversial results. In addition, a deeper understanding of the role of the immune system in Tourette's

**The phenomenology and natural course of tic disorders are complex . . . Tics may improve, but usually the tics are just one part of a larger neuropsychiatric syndrome**


PAUL BROWN/SPL

**Severity of vocaltics reduces in the 3rd decade**

## WHAT CLINICIANS NEED TO KNOW ABOUT TIC DISORDERS

Key aspects of the phenomenology and natural course of tic disorders include some of the following points. Tics, although they can be suppressed for brief periods, are often irresistible because of antecedent sensory urges, like the urge to cough or scratch, that call for an almost inevitable response.[2] As such, tics can be viewed as conditioned responses to these "premonitory" interoceptive stimuli, so that over time the associative interaction between the sensation and the resultant tic behaviour becomes stronger.

Motor and phonic tics occur in bouts over the course of a day and wax and wane in severity over the course of weeks to months. This can complicate treatment interventions. For example, if a drug is started during a period of waxing, when the tics are severe, and then the tics greatly improve over the following months, the uninformed clinician may presume that this improvement is caused by the drug when it is simply part of the natural course of the disorder.[3]

Understanding the cues and contextual factors that influence tic expression is key to refining and developing new behavioural interventions.[4] Specifically, tics are usually worse during periods of excitement, stress, and fatigue, and better during periods of goal directed behaviour that requires motor control, such as playing a musical instrument.

The severity of motor and vocal tics usually peaks early in the second decade, with a marked reduction by 19-20 years of age. However, in most cases, tics persist well into adulthood.[5] The most severe cases are lifelong and can be associated with self injurious behaviours. Although a growing body of evidence suggests that deep brain stimulation is helpful for some severe refractory adult cases, this procedure is experimental and is not always successful.[5]

Social, emotional, and academic outcomes in adulthood do not always reflect tic outcomes given the chronic course, the negative impact on peer and family relationships, and the variable number of co-occurring conditions.[1-4] Helping a child or an adult become a "self advocate" rather than being ashamed of his or her tics is often a helpful approach (www.tsa-usa.org/aPeople/ForPeople_main.html).

To ensure the best outcomes, clinicians, teachers, parents, and peers should be educated regarding the key phenomenological features and natural course of Tourette's syndrome and related disorders as described above.[1]

syndrome and related disorders is urgently needed. Key controversies in this area include whether a subset of cases with sudden onset (overnight) of obsessive-compulsive disorder and tics are caused by post-infectious autoimmune processes that parallel what is seen in other movement disorders such as Sydenham's chorea.

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**The linked systematic review and meta-analysis . . . finds little direct evidence to support the use of any drug for this condition**

# Treating sciatica in the face of poor evidence

## It may be necessary to extrapolate from evidence on treatment of other neuropathic pain syndromes

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The term "sciatica" is often applied to any presentation of low back and leg pain, although lumbosacral radiculopathy is a more specific term for the condition experienced by patients with low back pain who have impingement of lumbosacral nerve roots as they emerge from the spinal canal. This results in pain and sensory deficits in a dermatomal distribution and sometimes motor weakness in the corresponding myotomal distribution. Because the most commonly affected nerve roots are L4/L5 and L5/S1, pain typically radiates below the knee, and leg pain (elicited by performing the straight leg raise test) may be more pronounced than back pain. The most common cause of lumbosacral radiculopathy is intervertebral disc herniation, which occurs in about 3% of patients with acute low back pain.[1] Other causes include spondylolisthesis and foraminal stenosis owing to degenerative osteophytes.

The linked systematic review and meta-analysis by Pinto and colleagues (doi:10.1136/bmj.e497) finds little direct evidence to support the use of any drug for this condition.[2] Drugs commonly used to treat lumbosacral radiculopathy include analgesics such as paracetamol, non-steroidal anti-inflammatory drugs (NSAIDs), and opioids; drugs for neuropathic pain such as anticonvulsants and certain antidepressants; skeletal muscle relaxants; and systemic corticosteroids, whose anti-inflammatory effects may relieve pressure from herniated intervertebral discs on lumbosacral nerve roots.

Pinto and colleagues found few randomised trials of the effectiveness of such drugs, and, when meta-analysis was possible, pooled estimates generally showed no benefits over placebo for either pain or function. Furthermore, available trials typically had small sample sizes, did not perform long term follow-up, and had methodological shortcomings. The authors therefore concluded that for most drugs the quality of evidence was very low to low. Systemic corticosteroids were the one potential exception, with a meta-analysis of two small (n=78 and n=60) trials showing positive effects on short-term (more than two to three months) pain compared with placebo, although the benefit was small (about 10 points on a 100 point pain scale).

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**Points at which pain of sciatica commonly manifests**

A shortcoming of the systematic review is that it focused on mean improvements in pain or disability scores and did not evaluate the likelihood of experiencing a clinically meaningful benefit,[2] which is often considered a better measure of treatment effects. Several trials of systematic corticosteroids in the review reported this information, with mixed results. The systematic review also did not include some relevant older negative randomised trials of systemic corticosteroids, which reported dichotomous outcomes.[3-5] Thus, the available evidence is insufficient to support a treatment recommendation for systemic corticosteroids, although a trial currently in progress has a target sample (n=270) substantially larger than any previous study and should help clarify their role (http://clinicaltrials.gov/show/NCT00668434).

Given the paucity of evidence available from drug trials, how should clinicians select a treatment for this common, usually painful, problem? Clinicians still need to make treatment decisions even when evidence is suboptimal. In such situations it is necessary to use "indirect" evidence by extrapolating from the findings of trials evaluating drugs for other conditions and making assumptions about generalisability.[6] In this case, it is reasonable to assume that true lumbosacral radiculopathy should respond to drugs in a similar way to other types of neuropathic pain. Therefore, for patients with symptoms and signs typical of lumbosacral radiculopathy, clinicians may consider drugs that are effective for other types of neuropathic pain, such as pregabalin or gabapentin, and certain selective serotonin-noradrenaline (norepinephrine) reuptake inhibitors and tricyclic antidepressants.[7] However, more research is needed to confirm that lumbosacral radiculopathy responds to drugs similarly to other types of neuropathic pain. Pinto and colleagues found that NSAIDs had small but unclear benefits, which is consistent with the perception that NSAIDs are generally not effective for neuropathic pain, although the evidence is limited.[8] Opioids, although effective for neuropathic pain,[9] are not a first line drug owing to the potential for misuse and overdosing. Opioids should be reserved for severe or intractable lumbosacral radiculopathy, in appropriately selected and monitored patients. Skeletal muscle relaxants and benzodiazepines are not recommended as a first line drug because they have not been well studied for neuropathic pain and can have sedative effects.

Regrettably, no evidence is available to guide drug choices in patients with back and leg pain with features inconsistent with lumbosacral radiculopathy (for example, the pain is in a non-dermatomal distribution), although it is probably reasonable to follow general guidelines on the management of low back pain.

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