The piriformis syndrome: a report of a systematic review of its clinical features and the methodology developed for a review of case studies.

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Abstract

Background Piriformis syndrome (PS), sciatica caused by compression of the sciatic nerve by the piriformis muscle, has been described for over 70 years yet it remains controversial. The literature consists mainly of case series and narrative reviews. The few studies of diagnostic accuracy have had very small samples, a significant risk of bias or both. It has been suggested that PS may account for cases of sciatica where routine investigations fail to demonstrate herniated intervertebral disc or lumbar canal stenosis, the conventionally accepted causes.

Aims First, to estimate the frequencies of clinical features in patients reported as having PS. The existing evidence is composed largely of case studies so the objective was to make the best possible use of this limited evidence. Second, to identify research questions that are more specific than hitherto in order to progress future research.

Methods A systematic review of any study type that reported extractable data relevant to diagnosis was undertaken. The search included all studies up to 1 March 2008 in four databases: AMED, CINAHL, Embase, and Medline. In the absence of guidance or a consensus statement on the conduct of systematic reviews of case studies, appropriate methodologies for quality assessment and data synthesis were developed. Quality criteria focused on the completeness of reporting history and physical signs routinely assessed in sciatica and those said to be indicative of PS. Frequencies were calculated from individual data studies. Several denominators were used, thereby providing a range of estimates dependent on potential reporting biases. These denominators were: all cases, those cases with explicit reporting only, those with corroboration of the syndrome, and those with explicit reporting and corroboration. Two reviewers performed all screening, data extraction, and analysis independently.

Results Two hundred and twenty seven unique titles were identified. Fifty five studies were included: 51 provided details on individual patients (individual data studies), three provided only frequencies in case series (aggregated data studies), and one study reported some individual and some aggregated data. Quality of reporting was poor. Only 22 individual data studies scored all items on quality assessment and all but one of the aggregated data studies were so inadequate that their results were not considered further. Four features occurred most commonly across all denominators chosen: buttock pain, external tenderness over the greater sciatic notch, aggravation of the pain through sitting and augmentation of the pain with manoeuvres that increase piriformis muscle tension. Future research could start with comparing the frequencies of these features in sciatica patients with and without disc herniation or spinal stenosis and with measuring if they occur significantly together to warrant delineation of a syndrome.

Introduction

Background

History of sciatica and piriformis syndrome

Sciatica is musculoskeletal pain felt in the leg[33] along the distribution of the sciatic nerve and sometimes accompanied by low back pain. It may be caused either by disturbances of the nerve roots that comprise the nerve or of the nerve trunk.

Medical thinking was dominated for decades by the belief that the commonest cause of sciatica was compression of the nerve roots by a herniated intervertebral disc[138]. This belief owed its success to the work of Mixter and Barr who convincingly correlated clinical features with operative and histological findings in a series of sciatic patients. They were the first to perform a laminectomy specifically for a herniated intervertebral disc[83]. With time, the role of disc herniation was extended to implicate it as the commonest cause of low back pain even in the absence of sciatica [138].

Freiberg and Vinke, working in the same period as Mixter and Barr, argued that compression of the sciatic nerve by the piriformis muscle could cause sciatica [44][105]. They based this opinion on post-mortem studies and Freiberg later described cases of sciatica cured by division of the piriformis muscle[45]. The relations between the PM and the sciatic nerve are illustrated in figure 1. Robinson also described division of the PM for sciatica and is credited with coining the term piriformis syndrome, PS [105]. These arguments attracted less attention than the disc herniation theory. Nevertheless, case reports and case series of PS have continued to be published sporadically since the time of Freiberg.

Proposed mechanisms for PS include:

1. Contracture or spasm of the PM from trauma[44] [105];
2. Predisposition to nerve compression by congenital variations of the sciatic nerve or PM, in which the sciatic nerve or its divisions pass through the belly or tendinous portions of a normal muscle or the bellies of a bifid muscle [24] [98] [109]
3. Overuse and hypertrophy[12] [25] [97; 132].

Despite numerous case studies, the incidence and even the existence of PS remain controversial. A small survey of physical medicine and rehabilitation specialists in the USA found that only 21 out of 29 believed for certain that the condition exists [111]. Different authors have argued that there is over diagnosis[123], under diagnosis [34] and even both [16]. In contrast, lumbar spinal stenosis causing compression of the nerve roots has been progressively recognised as a cause of sciatica alongside HIVD.

Definitions

Several authors have credited Yeoman with the first descriptions of PS early in the twentieth century [44] [91; 116] [87; 94]. He reported that 36 out of 100 patients with sciatica had radiographic abnormalities of the sacro-iliac joint (SIJ)[142]. He speculated that sciatica could be caused by a "periarthritis", an inflammatory condition that might include the piriformis muscle. Modern musculoskeletal medicine does not recognise periarthritis as an entity. Yeoman's report had very few clinical details and nothing to demonstrate piriformis involvement so its relevance to PS is more historical than informative.

Robinson's original definition of PS had six "cardinal features":
"(1) A history of trauma to the sacroiliac and gluteal region
(2) Pain in the region of the sacroiliac joint, greater sciatic notch and piriformis muscle, extending down the leg and causing difficulty in walking
(3) Acute exacerbations of the chronic pain brought on usually by stooping or lifting, which can be relieved to a great extent by traction on the affected leg.
(4) The presence of a palpable sausage shaped mass over the PM, an acute exacerbation of the pain, which is markedly tender to pressure, is almost a pathognomic sign.
(5) A positive Lasegue's sign.
(6) Gluteal atrophy may be present depending on duration." [105].
This definition is unsatisfactory because it mixes aetiology (trauma), clinical features and late complications (muscle atrophy).

Some authors use PS for sciatica arising from nerve trunk compression by any cause, regardless of PM involvement, for example by osteophytes [73], haematomas[122], pseudo-aneurysms[81] [95], endometriotic cysts in the pelvis[32] and prolonged external pressure[27]. Fractures, of the femoral neck and of the ischial tuberosity, and hip arthroplasty too can cause sciatica [82] [145]. Such cases were called secondary PS by Foster[42] and pseudosciatica by others[108]. The wider definition of PS confuses attempts to describe the clinical features of PS, narrowly defined, which are related to the structure and function of the PM.

In an attempt to clear up the confusion, Foster divided PS into primary and secondary. Primary PS covers problems intrinsic to the muscle and secondary PS covers masses compressing the sciatic nerve or irritation of the sacro-iliac joint[42]. Hopayian[53] and Papadopoulos et al [93] have proposed the "term pelvic outlet syndrome" for Foster's secondary PS but this has not entered into common usage.

For the purposes of this review, the following definitions have been chosen:
PS is taken to be sciatica arising from pressure on the sciatic nerve trunk or its branches by the PM or disorders involving the muscle, whether or not congenital variations in anatomy are present. This excludes other causes of nerve trunk compression that have no relation to the PM, such as pseudoaneurysms and cysts.

Sciatica has been defined, in a consensus statement aiming to standardise terms for epidemiological studies[33]. Two definitions were agreed. The minimal definition is musculoskeletal pain felt in the leg. The optimal definition is

musculoskeletal pain extending below the knee. The minimal definition was chosen for this review because it has been widely used, including in the PS literature.

The definition of low back pain presented a difficulty in relation to PS. It has been defined within European guidelines as pain and discomfort, localised below the costal margin and above the inferior gluteal folds, with or without leg pain [134]. This definition includes both buttock pain and pain in the lumbar region. However, several reports of PS have differentiated between buttock pain and low back pain, suggesting that for those authors low back pain refers to lumbar pain. For example, some have reported that low back pain was absent while buttock pain present[2] [5] [109] [118], or that both low back pain and buttock pain were present [2] [4] [51] [68; 87] [132]. We have, therefore, taken low back pain to refer to pain in the lumbar region.

Epidemiology

Estimates of the incidence and prevalence of sciatica vary enormously between surveys. The reasons for this variation are differences in the definitions, the survey methods and whether occupational groups or the general population are surveyed[69]. The following estimates have been taken from general population surveys reported in a systematic review: lifetime prevalence, 12.2–27%; annual prevalence 2.2–19.5%; point prevalence 1.6–4.8%[69].

The proportion of sciatica cases due to disc herniation remains uncertain. In a series of 160 sciatica patients, only 131 (82%) had a corresponding disc herniation on MRI[63]. The same study found that there was no correlation between the degree of disc disease and severity of symptoms and signs. Those who argue that the role of disc herniation is overplayed point to the fact that although discectomy leads to more rapid recovery from the acute condition, the life time history of sciatica may not be altered[47]. In one study, aalthough surgery led to more rapid recovery from sciatica compared to non-surgical treatment, 20% of sciatica sufferers were symptomatic at two years whether or not they had had surgery [63].

It is harder to estimate the prevalence of PS than disc herniation in sciatica since the former has no accepted reference diagnostic test. Estimates of the ratio of PS to disc herniation from secondary or tertiary care are not straightforward because different definitions and denominators have been used. Furthermore, the reliability of estimates varies. Those based on coding of diagnoses at the time of procedures or clinic attendances[9; 56] are less open to selection bias than those based on a retrospective review of case notes [7][91].

Benson and Schutzer [7] retrospectively reviewed 93 cases referred to them by other orthopaedic surgeons. They identified 14 cases that did not improve with conservative management and went on to have surgery for PS, giving a prevalence of 15%. However, their cases included contused hip, whose frequency they did not report therefore the estimate is unreliable. Pace and Nagle[91] reported 45 cases out of “some” (sic) 750 sciatica patients but this lack of precision and the fact that cases were identified by recall renders any estimate very unreliable.

At the Hagevik Orthopaedic Hospital, Norway surgeons performed 19 operations for PS but over 1500 for disc herniation over 16 years[56] giving an

estimated prevalence of 1%. Bernard and Kirkaldy-Willis [9] reported only 5 cases out of a series of 1293 cases of back pain and sciatica over a 12 year period, giving a prevalence of <1%. Both these studies coded cases at the time of treatment and so were less prone to recall bias than the others. Therefore, the best estimate for PS as a proportion of sciatica seen in orthopaedic practice is ≤1%.

Diagnosis of piriformis syndrome

Investigations

There is no accepted investigation that can act as the reference standard for PS. Several candidates have been proposed.

Nerve conduction studies (NCS) and Electromyography (EMG)

Fishman et al attempted to set an operational definition of PS by demonstrating objective EMG findings with symptoms [41]. They measured the H Reflex on EMG in the FAIR position (described below), a test they called the modified H reflex. They found a delay in the modified H reflex in patients with PS compared to asymptomatic controls. An impressive large number of patients, 918, were studied. However, the study did not establish the accuracy of the H Reflex because it lacked symptomatic controls (patients with sciatica but not PS). Fishman et al also claimed that response to conservative therapy was greater in patients with a positive test but scrutiny of their results shows that they did not reach statistical significance. Furthermore, the study design and report did not meet the STARD criteria for a study of diagnostic accuracy [11]. Campbell and Landau cast doubt on the study by Fishman et al. They pointed out that the H reflex is difficult to elicit in people aged over 60 yet Fishman and colleagues appear to have elicited it in all their cases[17].

Slipman et al [112] calculated the positive predictive value of the modified H reflex and reported that it was too low to be useful, though many would argue that a sample of 6 patients might be too small to make such a bold attempt.

Chang et al tested NCS with magnetic stimulation[21] comparing 23 patients and 15 volunteers. The mean motor nerve conduction velocity of the sciatic nerve at the gluteal segment in L5 component in patients with PS was slower than the mean value in healthy controls (P= 0.014). They claimed a diagnostic sensitivity by magnetic stimulation of 0.467. However, sensitivity estimation requires symptomatic controls and even if this figure were correct, a sensitivity of 47% is low. Their findings have not been reproduced.

Imaging

Filler et al have championed the use of MRI neurography to identify nerve entrapment. In their case series of 162 patients[40], they classified patients with sciatica who responded to local anaesthetic and steroid injections into the PM as *confirmed muscle based PS* and those who improved with surgery as *surgically confirmed muscle based PS*. Abnormal findings on MRI neurography in confirmed and surgically confirmed PS patients were reported to have an important predictive value. However, Tiel has argued that response to an injection of anaesthetic into the

PM does not prove that the lesion lies in the PM and that the abnormalities reported by Filler et al may have been artefacts[131].

Lewis et al also found MRI neurography was associated with abnormalities in the PM or sciatic nerve but in a small series of 14 patients and without any comparison group[75].

Broadhurst et al used ultrasound to evaluate the texture and size of the PM. They examined 27 patients with LBP and buttock pain comparing the affected side with the opposite muscle. They reported that 18 had an abnormality on the affected side but did not state what the abnormality(ies) was/were. Further detail was sought from the authors but no reply was received. Pecina reported on 10 people with PS who had MRI then surgery. All 10 had an abnormality of the PM and four had a difference in sizes between affected and asymptomatic sides. The difference was reported to be at least 20% but the actual figures were not given. The authors were contacted but did not provide the raw figures (Personal communication, M Pecina, Dept of Orthopaedic Surgery, Zagreb, 14 December 2009). Doubts on the importance of unequal sizes of PM have been raised by the findings of Russell et al. They reviewed 100 sequential patients having routine pelvic MRI for sciatica and who did not have PS symptoms. Sixteen had a difference in size of their PMs between 4 and 8mm. Actual measurements were requested but the original data were not available (Personal communication, Prof Mark Kransdorf, Dept of Radiology, Mayo Clinic, 17 December, 2009).

In summary, no single investigation has been validated in the diagnosis of PS.

Specific tests for PS

Several signs have been reported as specific to PS (table 1).

(1) On inspection, tonic external rotation of the hip may arise from shortening or spasm of the PM[116]. This sign has been referred to by some as the 'piriformis sign' [34].

(2) Tenderness of the PM may be found on external palpation over the greater sciatic notch or on internal palpation per vaginam or rectum[34] [105] [141].

(3) Several tests reproduce sciatica by augmenting PM tension:

   a. by passively stretching the muscle, Freiberg[44] and FAIR tests[143]

   b. by contractin the muscle against resistance, the Pace[91] and Beatty tests[5].

Campbell and Landau[17] have rejected these last two tests for being contradictory. They argue that it cannot be the case that pain is reproduced by both manoeuvres that stretch a muscle and those that contract it. However, this principle does not appear contradictory to practitioners of orthopaedic medicine who use it for a wide range of soft tissue diagnoses; tests that reproduce pain on contraction use *resisted* contraction[89].

Case studies

The value of case studies

Case study reports comprise case reports (reports of single cases) and case series (more than one case). Their role in education is the easiest to expound. Case reports remind us of the unusual, warn us of pitfalls and help us to learn by pegging theory onto real examples. This may explain why case reports remain among the most widely read, though not most widely cited, contents of journals [79]. In contrast, their role in research is less appreciated. Many modern clinicians remain dismissive, as demonstrated in a rapid response to the BMJ in 2003: "The plural of anecdote is not data." [48]. A survey of institutional review boards (research ethics committees) in US medical schools found that only 15% classed case reports as research at all[92].

Knowledge gained from case studies has limitations. Generalising from particular cases has its dangers and the absence of a comparison group disallows hypothesis testing. Those who promote the concept of levels of evidence allocate case studies next to bottom level in the hierarchy of evidence[19]. Nevertheless, they still have important roles (box 1)[135] and have seen a revival of interest. In 1995, the Lancet invited the submission of case reports [10]. The British Medical Journal launched a series of evidence-based case reports, by which was meant a case report supported by a systematic search of the literature. At least three online resources devoted solely to case reports have been launched since 2007 [66] [1; 113].

Discovery begins with finding the unexpected and the stimulation of further research [135]. Evidence of cases and their occurrence is needed before evidence of aetiology or treatment effectiveness can be established[59]. Case reporting can, therefore, lead to more advanced research. At times, case reports have led to changes without further research though, most commonly to withdrawals for adverse drug reactions[88].

Adhering too rigidly and uncritically to the hierarchy of evidence overlooks 'lower levels' of evidence that have potential. Jenicek has pointed out: 'Case reports and case series may be the "lowest' or the "weakest" level of evidence but they often remain the "first line of evidence"'[59].

Structured reporting of case studies

The question, therefore, is not whether case study reports have a role in medical progress but whether they can fulfil that role effectively. Jenicek went on: 'Clinical case reports…should represent…a scientific endeavour comparable to other observational or experimental research projects'. Vandenbroucke argued that anything less is not acceptable. 'A certain type of case report will (or should) never make a come back: the droning recital of one case after the other as a lame excuse for an (unstructured) review of the literature'[135].

A survey of 249 journals, found that 162 had instructions for authors on case reports but that the information provided was limited and varied[117]. Aaronson has argued for guidelines for reporting of adverse drug reactions[3]. Jenicek has described the elements of a good case study report[59]. Carey and Boden have suggested criteria for good case reporting [18]. However, there is no consensus on the reporting of case series in contrast to other research designs: CONSORT for trials[84], MOOSE for observational studies[137][124], STARD for studies of diagnostic accuracy[11], PRISMA for systematic reviews[76] and even for guidelines, AGREE [129].

Systematic reviews of case studies

Case studies are suitable material for systematic reviews. Ernst conducted several narrative syntheses of the adverse effects of complementary and alternative treatments[36] [37] [38; 39]. Most of his reviews have listed adverse events with the aim of drawing attention to them. One went further and synthesised data from diverse study types, including case studies, to answer several questions, such as an assessment of the extent of under-reporting [39].

Case studies provide suitable material for meta-analyses too. Raney et al pooled the complication rates following the removal of orthopaedic implants in a paediatric population [100]. Cook et al reviewed cure rates for any intervention compared to no intervention for traumatic optic neuropathy [29]. Limongelli compared the rate of delayed postoperative hemorrhage with two techniques for pancreaticoduodenectomy [77]. West et al reported the cure rate following bronchoscopic approaches to post-pneumonectomy bronchopleural fistula to explore its feasibility as an alternative to thoracotomy in patients not fit for the latter[140]. Schlosser et al conducted a meta-analysis of prognostic factors following aortic aneurysm repair[110].

All the previous systematic reviews have been to do with interventions. The only systematic reviews relating to clinical features we were able to find were those of Soga et al who studied the clinical features, laboratory results, and prognosis of patients with carcinoid[114; 115].

Existing reviews of piriformis

The literature on PS consists largely of reviews and case studies. Most reviews of PS have been either narrative reviews [15] [50] [93] [102] [103] [106] sometimes with illustrative case reports[91] [141] or case studies accompanied by a review to place them in context.

Silver and Leadbetter[111] identified 26 cases in 12 studies [2] [4] [5] [13] [23] [45] [58] [61] [94] [109] [136] [141] and calculated frequencies for only three clinical features: ‘neurologic deficit’, the Freiberg sign and the Pace sign. The only systematic review of PS available at the time of our search was confined to non-surgical interventions[30]. Its two trials with positive outcomes were excluded from our review because they did not describe the clinical features sufficiently.

Filler, in a conventional review, claimed that three “large scale formal class A study design” publications have proven the existence of the syndrome [40]. Two of the studies were the ones by Fishman et al[41] and by Filler et al [40] mentioned above in relation to investigations. However, neither of these two studies were actually “class A”. By class A, Filler meant what Kent et al called “grade A” studies in the article he refers to[64]. The criteria in the paper by Kent et al were not as explicit as those on the web site of the CEBM [19] but did include the requirement that there be no serious flaws in the method. Both the Fishman and Filler studies had two serious flaws for studies of diagnostic accuracy: that all clinical features be described and that an adequate reference standard be used. The third study Filler summoned as proof was by Fishman et al, a study of botulinum toxin injection therapy for PS. This was an unblinded study so it too does not qualify as a grade A study.

Two more reviews have been published since the completion of our search and analysis. Hulbert and Deyle set out "to provide a review of the current literature from an evidence-based perspective for the conservative management of PS and to differentiate PS from classic sciatica or peripheral nerve entrapments"[55]. Other objectives were "to rank the levels of evidence" and "to identify areas for future research". Their main contribution was to highlight the paucity of evidence for differential diagnosis and treatment. However, their study was not, despite the desire to take an evidence-based perspective, a systematic review. The authors did not report essential features of a systematic review: the search strategy, the inclusion/exclusion criteria, the method of data extraction, or how the quality of studies contributed to the analysis. Scrutiny of their ranking of studies by quality reveals disconcerting clues that they may have misapplied the grading system. They reference the grading system as the “Sackett” system, meaning the hierarchy of levels of evidence published on the web site of the Centre of EBM, Oxford, England[19]. Discordance between the intended use of the hierarchy for primary studies and the actual use by Hulbert and Doyle (table 1 in their article) is illustrated by the following. First, the “Sackett system” and a paper by Guyatt and Rennie describing how to critique medical literature are given rankings although they are not primary studies about PS at all. Second, some case studies are given a level of 4 while others are given a level of 5 (expert opinion). Third, the very first study that was a primary study, Fishman et al 2002[41] and given a ranking of 2b, did not apply an independent standard test to all patients, a criterion needed for 2b status. In their conclusions for future research, Hulbert and Deyle called for more studies of conservative interventions and more studies of diagnostic tests but did not develop any clear research question.

Kirschner et al reviewed the evidence for botulinum toxin (BTX) and also discussed diagnosis[67]. The review of BTX was largely unsystematic. The search was confined to PubMed, there were no descriptions of inclusion/exclusion criteria, methods of extraction and analysis, or assessment of the quality of studies. The discussion on diagnosis (clinical features and investigations) did not describe the search strategy.

No review prior to ours had systematically searched for all reports, including case studies, nor extracted and analysed data according to pre-specified criteria, nor developed clear research questions.

Aims

We had two aims. First, to make the best use of existing evidence to estimate the frequencies of clinical features in patients reported as having PS. Our main research question was, in cases of PS reported in the literature, what is the frequency of the symptoms, signs specific to PS and signs looked for in sciatica in general? Second, to identify future research questions. We used any study types that reported data relevant to diagnosis.

Methods

The methods are in accord with the PRISMA statement on the conduct of systematic reviews [76].

Search

The search included all studies up to 1 March 2008. The Thomson Dialog NHS facility[1] was used to search four databases: Allied and Complementary Medicine (AMED), Cumulative Index to Nursing and Allied Health Literature (CINAHL), Embase, and Medline. The following search strings were used:

#1 (PIRIFORMIS OR PYRIFORMIS) ADJ SYNDROME.TI, AB

#2 (PIRIFORMIS OR PYRIFORMIS) AND SCIATIC$.TI, AB

1 OR 2

We chose this search strategy after comparing the results of alternatives with articles already known to us.

(1) The spelling pyriformis was included because a few authors have preferred this alternative.

(2) A search strategy with greater specificity for diagnosis missed several articles, so search tags for diagnosis such as sensitivity and likelihood ratio were dropped.

(3) Some authors did not use the term piriformis syndrome although they ascribed sciatica to piriformis pathology. The connector 'ADJ' (adjacent) was used to identify articles that used the term syndrome and the connector 'AND' was used to identify those that did not.

Additional studies were sought in the references of all retrieved articles.

[1] The Thomson Dialog NHS facility was available to all NHS staff but has since been withdrawn.

Inclusion/Exclusion

Two reviewers (KH and RR) independently judged all titles and abstracts for retrieval of the paper. Where a disagreement occurred, the study was retrieved. Studies were excluded if:

1. They were not about PS.
2. The language was not English, French, Chinese or Spanish
3. The publication was not a print or internet biomedical journal
4. The condition was a complication of hip surgery or fracture.

When a disagreement occurred, the article was retrieved for screening.

Two reviewers (KH and RR) independently screened the retrieved full text articles except for articles in French (KH only) and Chinese (FS only). Studies were included if they satisfied all three criteria.

1. The study had to be one of: a case studies report; a narrative review including a case studies report; a study of diagnostic test accuracy; or a study of a therapeutic intervention that described clinical features.
2. The cases matched the study definition of PS.
3. The clinical features were described sufficiently for data extraction.

Included French and Chinese articles were translated and then passed to a second reviewer.

Data extraction

Two reviewers (KH plus RR or SS or FJ) independently extracted data from included articles.

Studies were divided into 'individual data studies' (case reports and case series reporting data for each patient) and 'aggregated data studies' (case series reporting data aggregated for all patients). Articles were scrutinised for pre-specified features (box 2) chosen from prior knowledge of the literature with two (tonic external rotation and tenderness on rectal examination) added after reading retrieved articles.

Several reports of PS have differentiated between buttock pain and low back pain [2] [4] [5] [51] [68] [87] [109] [118] [132]. For this review, we accepted this demarcation although recent European guidelines define low back pain as localised below the ribs and above the inferior gluteal folds[134].

The rules for data extraction were:

1. Features stated as present or absent were recorded as positive or negative respectively.
2. If the absence of a feature was not explicitly stated, it was recorded as 'not reported'.
3. Ambiguous reports, arising from vague or summary phrases, for example, 'no signs of radiculopathy, were recorded as 'uncertain'.

Physical signs proposed as specific for PS were recorded as present or absent when authors used the eponymous name or described the manoeuvres sufficiently to identify them. Otherwise, the feature was recorded as not reported or uncertain. Where authors reported an idiosyncratic sign, this was recorded separately.

There is no consensus for the correct execution of the test or nomenclature for manoeuvres known variously as straight leg raising, passive straight leg raising, the Lasègue sign, the Lasègue test[101]. Therefore, they were treated as being the same.

Analysis

Managing biases in calculating frequencies in studies with individual patient data

Choosing the denominator to calculate frequencies from case studies is problematic. When a feature is not reported in a publication, it would be unsafe to conclude that the feature was absent. Brief clinical records are not written with future publications in mind[59] so there are alternative explanations. The clinician may not have sought the feature; the clinician may have sought the feature but not recorded its absence; the clinician may have recorded its absence but not included it in the report. A denominator that includes all cases might underestimate the frequency if a feature had been present but not sought. A denominator that is confined only to studies where a feature was reported explicitly as either present or absent, might overestimate the frequency particularly if the authors selectively report positive cases believing it to be pathognomic.

Another potential source of bias is the use of a clinical feature as a criterion for patient selection. This would tend to return a 100% frequency for the feature. Evidence for this bias was found in the aggregated data studies included in the review. Evidence was also found in two individual data studies Benzon et al [8] and Slipman et al[112]. Both were excluded on other grounds. We decided to calculate frequencies in four ways: all cases; only corroborated cases, only reported cases (i.e., feature explicitly reported as present or absent); only corroborated and reported cases. A feature recorded as uncertain, was treated as absent in the analysis. Denominators were calculated using only the sample relevant to a feature: only women for dyspareunia and only cases published after the first description of PS specific tests (table 2).

Numerators were calculated by adding the number of patients with positive features. Since the point estimates of percentages were often close to 100%, 95% confidence intervals were calculated by first transforming percentages to log odds[125].

Analysis of studies with aggregated patient data

Frequencies for diagnostic features were calculated for each study with the intention to pool data if appropriate.

Analysis of the quality of case studies

Although much has been written on the subject of reporting case studies, we could not locate an accepted tool for evaluating the quality of case reports in diagnosis. The Centre for Reviews and Dissemination's recent guidance on systematic reviews does not mention case studies at all[20], unlike an earlier version[65]. The section on case studies in the earlier version related only to therapy and prognosis. The US Agency for Healthcare Research and Quality's review rating systems did not include case series[139].

We found proposals from several individuals on structured case studies reports but none satisfied our needs. Aaronson has suggested guidelines specifically for the reporting of adverse drug reactions[3]. Young et al provided the only grading system we could discover[144] but it is quite specific for therapy so not suited for a review of clinical features. Terrasa et al. included studies of diagnostic features in their guide to the critical appraisal of case studies [128]. Their example related to a condition that was supported by laboratory diagnosis, unlike PS. Writing for a musculoskeletal readership, Carey and Boden[18] proposed a structure for reports that was based on the eight domains of reporting on observational studies taken from the Agency for Healthcare Research and Quality's recommendations[139] box 2. Their proposal was specifically for case series and concentrated on interventional studies.

We took therefore developed our own tool, taking as our starting point the core elements of a case study proposed by several authorities, Carey and Boden[18; 57], Jenicek[59] and Terrasa et al[128]:

1. The comprehensiveness of reporting.
2. Good case definition.
3. The minimisation of bias, such as recruitment of consecutive cases in cases series.

<u>Comprehensive reporting</u>

The items are shown in box 3. Both history and examination were considered in judging the comprehensiveness of a report. Age and sex are vital demographic data in medical records. It could be argued that occupation and social history are also important in musculoskeletal medicine but we decided to keep to a minimum standard. In judging the history, we included the basic components of history taking for pain. History includes past medical history, of both musculoskeletal disease and other disease, which may have a bearing on the present condition. Studies were categorised according to the number of items reported in the history: good if two or fewer items were missing; satisfactory if three or four items; and poor if more than four. For case series, the poorest report was used to categorise the study. For studies

with aggregated data, the history reporting was so poor that no attempt was made to categorise them.

Two sets of examinations can reasonably be expected in a report on PS: routine tests for sciatica, such as SLR; and specific tests for PS. The number of routine tests for sciatica in each case study was counted. For case series, the case with the lowest number of reports was taken to represent the quality of the study. For specific tests of PS, the presence of at least one specific sign was sought since the number of signs available has changed over time.

Case definition

The absence of a reference standard test means uncertainty over whether cases truly represent the condition. However, certainty is on a continuum. Some authors proffer evidence to support their diagnosis, such as response to surgery after a long duration of pain. Such evidence cannot be accepted uncritically but should be weighed and judged like all evidence. Evidence that supports a plausible cause and effect we have termed corroborating evidence, without implying incontrovertible case definition. Any potential corroborating evidence was recorded as free text comment. All comments were then scrutinised and compared to create categories of corroborating evidence.

Minimisation of bias

Case series are by definition more than one case but can be many more. It is important that the method of selection of the cases be described and also important that it minimise bias, for example, by including consecutive cases.

We decided against assigning quality scores and against performing sensitivity analysis. Sensitivity analysis would have been inappropriate because there is an overlap of certain items in the quality assessment and the calculation of frequencies, for example, reporting of routine signs for sciatica. We did, as already described, report different rates for corroborated and all studies but this was more to provide a range of frequencies rather than to perform sensitivity analysis.

Two reviewers (KH and RR) independently assessed the papers for study quality.

Results

Search results

The flow of records is shown in fig 2. Studies entered into the synthesis comprised 51 individual data studies (table 2), three aggregated data studies[34] [56] [78], and one combined[34]. Of the individual data studies, 31 were case reports (single case) and 24 case series (two or more cases). One of the case series was strictly speaking a cross-sectional study[21]. As a cross-sectional diagnostic study, it

was of poor quality since it compared cases to asymptomatic individuals and the sample size was small (38). We extracted the data on the group with PS.

Borderline cases

Two papers presented peculiar difficulties in the decision to include or exclude studies. Slipman et al [112] studied the accuracy of modified nerve conduction studies in patients with suspected PS. We excluded this study because the only clinical features reported were the ones used as inclusion criteria into the study. Other inclusion criteria were relief of symptoms with injection of local anaesthetic and exclusion of other causes by CT or MRI. These last two were similar to the inclusion criteria for the case definition by Filler et al. [40]. We did include Filler et al's results because, apart from the presence of buttock and leg pain, the clinical features were not the same as the inclusion criteria.

The report of a case series by Mullin [86] et al was ambiguous. Several features of PS were listed but it was not clear if patients had to have all features or just some in order to be recruited. This study was excluded. Some cases of PS such as Picco et al [99] were excluded because although they had leg pain they did not present with sciatica but some other symptom.

Quality assessments

Individual data studies

All reported age and sex. The quality of history reporting was good in only twenty four studies. Commonly missed items were onset of pain, past medical history and evolution of the symptoms. Forty six studies reported at least one sign of PS but six reported none. Twenty three studies reported three or more routine signs for sciatica, 14 reported one or two, and seven reported none. Reporting was uncertain in five. Thirty two studies were judged to have good or satisfactory descriptions of history and to have reported both sets of signs

Selection

Of the twenty four case series, only one reported its inclusion criteria[75]. It described a retrospective study of the records of patients with a mismatch between spinal MRI and their clinical condition referred for MRI neurography but failed to report how they were selected from such referrals.

Corroborating Evidence

Of the case studies with individual data, 79 cases had one or other form of corroborating evidence. The categories of corroborating data are shown in table 5 with examples. The types are not mutually exclusive so that many cases had more than one item of corroboration, illustrated by multiple entries in the examples column. There were reports of congenital anomalies of the PM and/or sciatic nerve, acquired

abnormalities of the PM and/or sciatic nerve but also of normal morphology with response to surgical division of the PM, for example, Barton, case 4[4].

Reporting of corroborative data was incomplete. Examples were: omission of the duration of the symptoms[87], operative findings[45] or of follow-up[6]. Even case series reports did not provide a consistent set of data for all cases in their series[68; 71].

Studies with aggregated patient data

Many items in history and examination were missed (table 3). Only Durrani and Winnie reported how patients were selected, how data were collected, the sex distribution, the mean age and age range and several features[34]. It was a prospective study of consecutive cases seen in a single clinic. Lu et al [78] reported only the range of ages and Indrekvam and Sudmann [56] reported only the mean age.

Filler et al[40] recruited from 239 patients with either failed disc surgery or no diagnosis after imaging, selecting those who obtained relief from MRI guided injection of steroid and local anaesthetic into the PM. They did not describe the sex and age distribution of the selected cases and reported only a few features.

All studies reported at least one sign specific to PS and one sign in the routine examination for sciatica.

Frequencies

Data were useable from a total of 126 patients, 100 in individual data studies and 26 from Durrani and Winnie.

Individual data studies

There were 52 women and 48 men with a mean age of 43 (95% CI 14, 72). Figure 3 shows the frequencies of the clinical features (with 95% CI) for each of the four denominators. Frequencies calculated from all cases (first plot on left) and corroborated cases (second plot from left) were similar (fig 3). However, frequencies calculated from reported studies (third plot from the left) were higher than in all studies and corroborated studies. Frequencies calculated from reported studies and reported corroborated studies (plot on furthest right) were similar. Corroboration made little difference to frequency estimates whereas reporting made a big difference.

Symptoms

Buttock pain was common and more common than low back pain for all denominators used. The estimates for buttock pain ranged from 50% (corroborated) to 95% (reported) and for low back pain from 14% (corroborated) to 63% (reported). Aggravation of sciatica through sitting was as common as buttock pain, with estimates ranging from 39% (all) to 97% (corroborated and reported). Dyspareunia showed the greatest discrepancy between all cases and reported cases (13% to 100%% respectively), reflecting the very large proportion of under-reporting in the all cases studies. Therefore, none of the estimates for dyspareunia are reliable.

PS specific signs

Frequencies were similar for the Freiberg sign, range 32% (all studies) to 63% (reported studies), and the Pace sign, 30% (corroborated) to 74% (reported). The numbers reported for tonic external rotation, FAIR and Beatty signs were small and the proportions of unreported cases high, so estimates are not reliable. External tenderness was common, with a range of 59% (corroborated) to 92% (corroborated and reported). Internal tenderness was frequently unreported, probably because this examination is seldom performed in orthopaedic or neurological practice. The range of estimates was 24% (corroborated studies) to 83% (reported).

Routine signs in sciatica

Limited SLR appeared to be the commonest finding, range 42% (all) to 62% (corroborated and reported), with diminished reflex, sensation and power reaching a maximum of 26%, 39% and 37% respectively.

Combinations of features

The commonest features were further analysed. Three features, pain in the buttock, pain aggravated by sitting and external tenderness were reported together in 22 cases, a frequency of 22% (CI 15-31) for all cases and 31% (CI 21-42) for reported cases. Of these 22, 12 were positive for at least one manoeuvre increasing PM tension.

Aggregated data studies

All four reported 100% frequency for buttock pain, suggesting this was part of their case definition (table 4). Two reported very few features[78] [56] and whose frequencies were close to or equal to 100%, suggesting case selection on the basis of these features. Filler[40] reported only frequencies rather than raw data. Pooling was therefore considered inappropriate. Only Durrani and Winnie reported several features (table 4).

In three studies, women comprised 39-73% of the series. In Durrani and Winnie's series, the features present in half or more than half the cases were: buttock pain, low back pain, pain aggravated by sitting, external tenderness, and internal tenderness. Only two specific signs were tested, Pace and tonic external rotation which were about as frequent as limited SLR.

Discussion

Strengths and limitations

The main strength of this is study is that it is the first review of the diagnostic features of PS to use systematic methods to synthesise existing evidence. It is the most comprehensive review of diagnosis, incorporating data from 100 individual cases and aggregated data from another 26. We have extracted data according to pre-specified criteria to cover three important diagnostic areas: symptoms, physical signs specific to PS and signs routinely tested in sciatica.

The limitations of the study arise from the nature of the literature reviewed. A synthesis of case studies may suffer from either under-reporting or over-reporting. Under-reporting is most likely to be problem for the absence of features. Over-reporting of signs may be a particular problem when the authors are promoting them as pathognomic. We have tackled this problem by providing a range of estimates through alternative methods of calculating frequencies. The ranges enable comparison of the features with each other. The absence of a reference standard does not diminish the value of these ranges since we found them to be similar in both corroborated and non-corroborated studies. Of the aggregated data studies, the one with the highest quality, Durrani and Winnie, reported frequencies close to those calculated from individual data studies, adding credibility to the findings.

The majority of cases were reported from secondary and tertiary centres, which are more likely to encounter severe or more chronic cases. Therefore, the generalizability to primary care is limited.

An important aspect of a case study is case definition: other causes for the condition should have been considered and reasons given for excluding them or for suggesting that the chosen diagnosis was the most plausible. In practice, many case studies typically present the outcome of treatment as *implicit* evidence of proof of the diagnosis. However, there are alternative explanations for such improvement, such as natural history, placebo response and observer bias. One strength of our review is that we have made the process *explicit* and assigned a lesser weight of evidence, support rather than proof. We have referred to this evidence as corroboration. However, what counts as corroboration is itself open to interpretation and the degree of certainty it can claim is variable. For example, does response to local anaesthetic and steroid into the PM count as evidence of PS or can it, as Tiel [131] has argued, also be expected in cases of more distal nerve impingement? There are instances where evidence even in the absence of a comparison group makes cause and effect seem so probable that a causal relationship is credible[42]. One example, is the case series of Lewis et al in which several clinical signs, MRI findings and findings at operation were all concordant and where surgery was followed by relief of symptoms[75]. Not all corroborating evidence was equally cogent. It is possible to rate the studies according to the strength of the corroboration but we did not attempt to do so because it would have been a post hoc analysis. Furthermore, we concluded that doing so would not settle the controversy over the status of PS but synthesizing and making transparent the data would enable judgement on how much weight to give them when considering the implications for practice and research.

Implications for practice

The concurrence of several clinical features and the numerous cases with corroborating data lend strong support for the existence of the syndrome. Practitioners may consider entertaining the diagnosis in patients with atypical histories[53] or a "negative MRI". Patients without a diagnosis after imaging still deserve an explanation for their symptoms and hope for their relief. Discussing the possibility of PS with patients in these situations is an option.

Four features appear to be most common: buttock pain, aggravation of sciatica through sitting, external tenderness over the greater sciatic notch, and augmentation of the pain with manoeuvres that increase PM tension. These tests are easy to perform within the usual clinical examination. Most practitioners, however, may be less inclined to perform routine internal examination without stronger proof of its accuracy.

This synthesis provides empirical data that challenge the received wisdom that neurologic deficits and limited SLR are rare in PS[91] [121]. It also challenges the belief that the prevalence in women is very much greater[91] [105].

It could be argued that there is no value in making a diagnosis where there is no proven treatment. However, the paucity of effective treatment is true of low back

pain and sciatica in general. The relief of pain with surgery in carefully selected cases of PS identified in this review has its parallel in the early history of disc decompression by Mixter and Barr. Nevertheless, the high success rates for surgery have been reported only in small series[43] [75] [97]. There is limited evidence for non-surgical therapy[30]. Whilst uncertainty about therapy remains, what is certain is that research into therapy is more likely to proceed when the syndrome has been systematically studied.

Implications for research

Filler marshaled imaging and outcome data to argue for the importance of PS in the aetiology and management of sciatica[40]. While the volume of empirical data he presented deserves attention, we have argued it does not amount to the highest level of evidence as he claims. Tiel has argued that there are alternative explanations for Filler's observations: that MR neurography changes are artifacts, that PM injections act by non-specific means and that placebo response may explain treatment success [130]. While Tiel may be correct in his line of reasoning with specific reference to Filler's arguments, it would be wrong to explain all successes as placebo. Many patients in our review had not had a placebo response to previous therapies, including disc surgery, but did improve after PM resection. The results of our study will not settle the debate on the existence or rarity of PS but they do lead to the formulation of specific research questions.

The significant minority of people with sciatica but no spinal cause (whether HIVD or spinal stenosis) points to the need to research extraspinal causes of sciatica. Our review raises five questions for research that would progress our understanding of the role of PS in these cases, starting with the frequency of PS specific features in sciatica in general, data which are not available because these tests are not routinely conducted.

1. How commonly do PS specific features occur in patients presenting with sciatica?
2. How do these frequencies compare with the conservative estimate of prevalence of ≤1% all sciatica cases?
3. Do PS specific features occur significantly more often in patients without a spinal cause than in patients with a proven spinal cause? This would provide stronger evidence that these features represent a condition distinct from sciatica from spinal causes.
4. Do the four features, buttock pain, pain on sitting, external tenderness and pain with increased PM tension occur significantly together and significantly more commonly in patients without spinal causes than in patients with spinal causes?
5. Is the quartet accompanied by objective tests of nerve trunk compression, such as imaging or NCS?

The first two questions could be answered by a prospective, structured documentation of patients presenting in primary care. The other three questions are best answered by cross-sectional studies of patients with sciatica.

A further implication is that single case reports or small series are unlikely to improve our understanding of PS unless they reveal previously undiscovered aspects of the condition. But future case studies as well as cross-sectional studies must be more informative. The quality of most case studies reviewed was disappointing. Future studies should report clinical features both comprehensively and explicitly. The items we used for quality assessment provide a framework for such reporting.

Appendices

Appendix 1 Figures

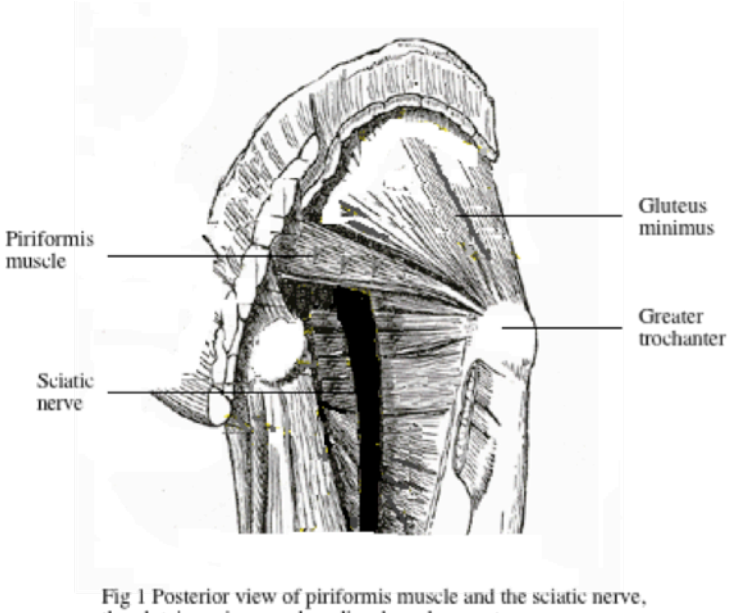
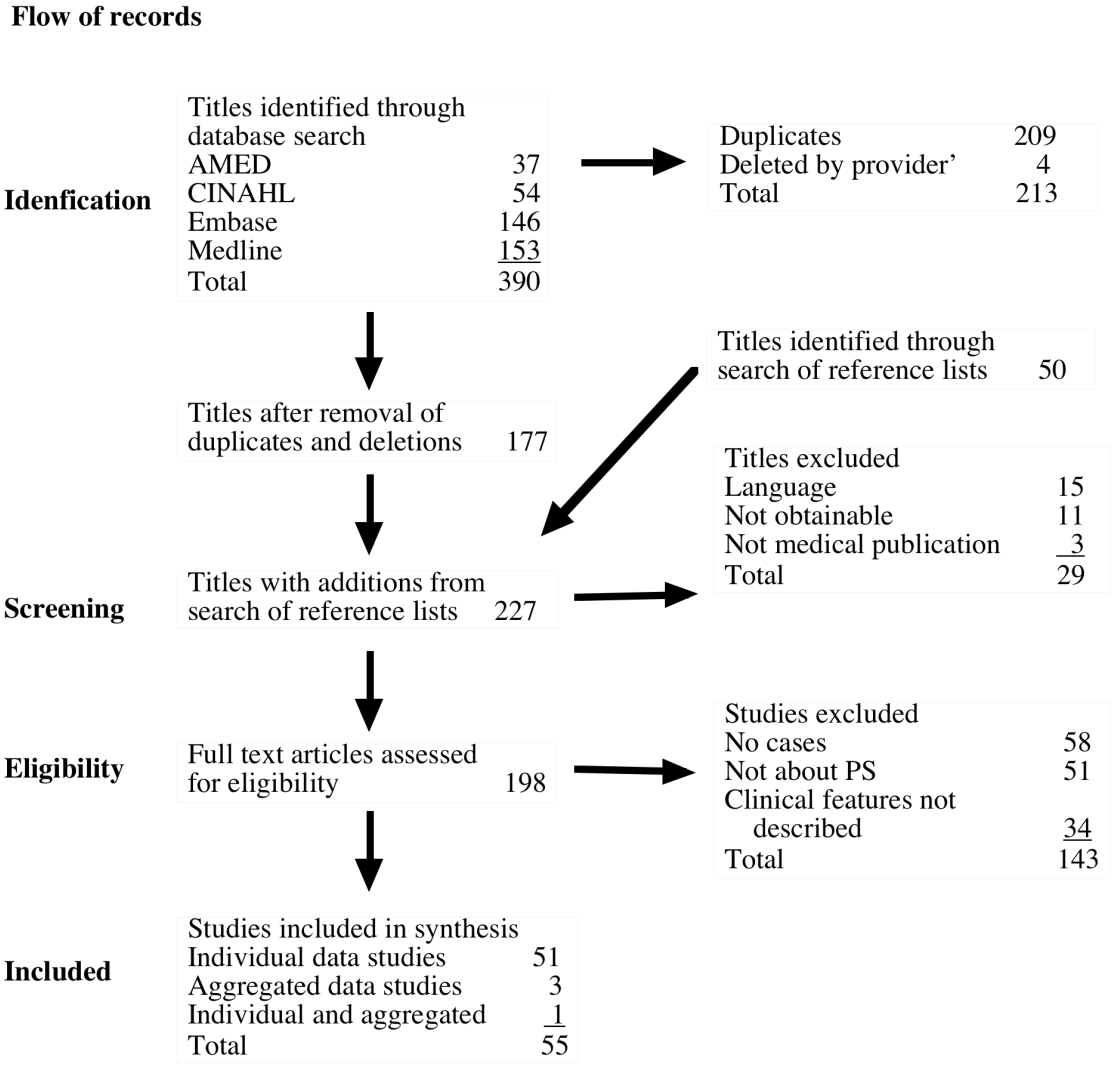


Fig 1 Posterior view of piriformis muscle and the sciatic nerve, the glutei maximus and medius have been cut away.

The PM originates from the pelvic surface of the sacral segments S2-S4, the sacro-iliac joint, the anterior sacro-spinous ligament, and the sacro-tuberous ligament. It passes through the greater sciatic notch to insert onto the greater trochanter of the femur. The sciatic nerve exits the pelvis below the belly of the muscle. Many congenital variations exist: the nerve may divide proximally, the nerve or a division of the nerve may pass through the belly of the muscle, through its tendons or between the part of a congenitally bifid muscle [85; 86]. The PM externally rotates, abducts and partially extends the hip.

Figure 1 Functional anatomy



Numbers refer to number of studies.

Figure 2 Flow of records*

* See note at end, Corrections

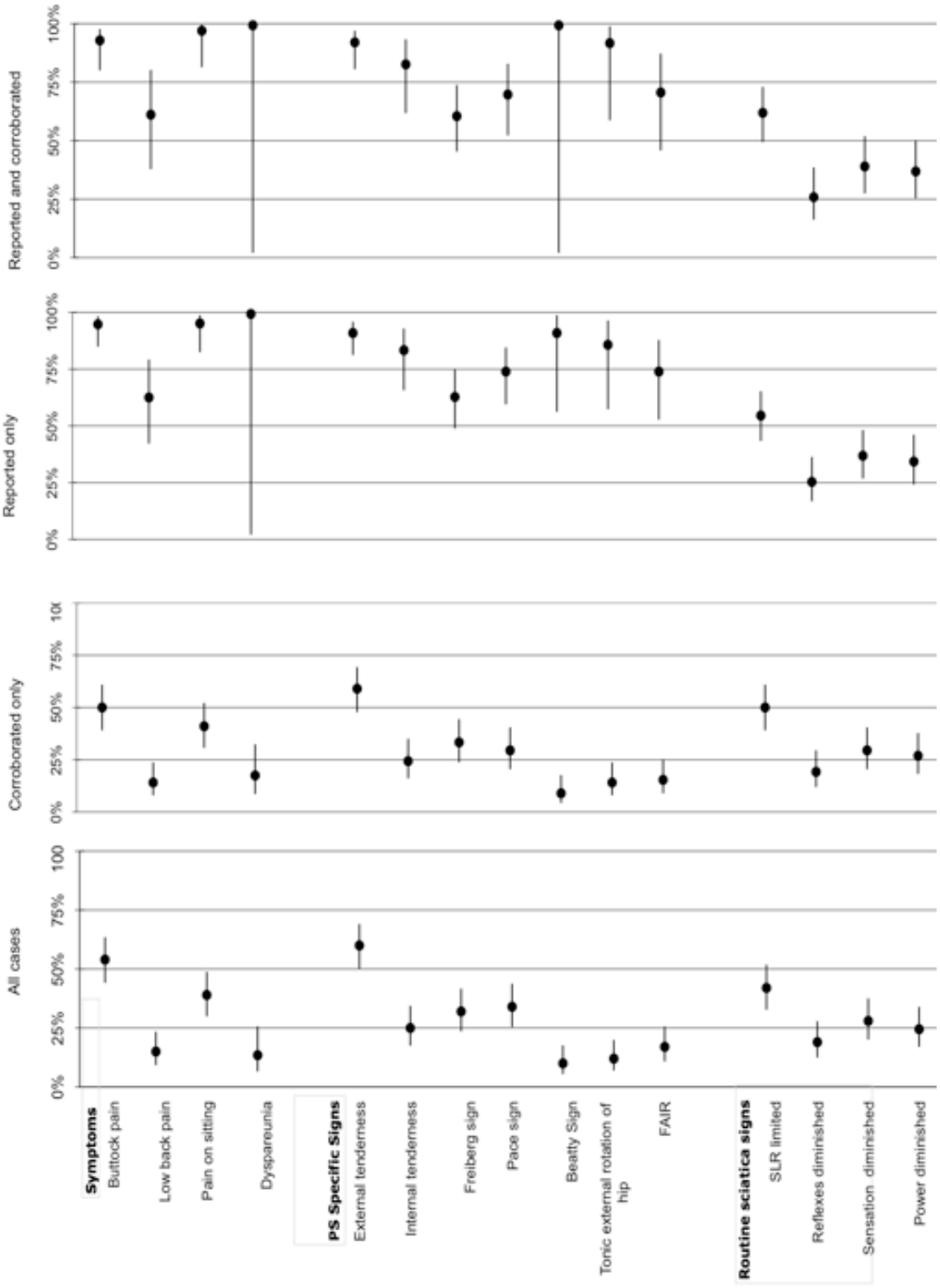


Figure 3 Frequencies from individual data studies

Frequencies are shown as the calculated value (circle) with 95% CI (horizontal bar) over the 25%, 50% and 75% centiles (vertical bars).

Appendix 2 Boxes

Box 1 Case reports: Limitations in knowledge acquisition and potential roles

Limitations Logical: danger of generalising from the particular Event rates not calculable or unreliable Lack of control group militates against hypothesis testing (risk factors, diagnostic accuracy, therapeutic effectiveness). Bias potential great
Potential Roles Recognition and description of new diseases Detection of drug side effects (adverse or beneficial) Study of mechanisms of disease Medical education and audit Recognition of rare manifestations of disease Generation of hypotheses for research

Box 2 Items for data extraction

Citation Type of study Patient identification number *Symptoms* Buttock Pain Low back pain Difficulty sitting or pain aggravated by sitting Dyspareunia *Signs specific for PS* External tenderness over the greater sciatic notch Internal tenderness of the PM on vaginal or rectal examination Freiberg test Pace test Beatty test Tonic external rotation of hip Flexion-Adduction-Internal Rotation (FAIR) painful *Routine sciatica signs* Limited SLR or positive Lasegue Knee or ankle tendon reflex diminished Sensation along dermatomes L4 L5 and S1 diminished Power in myotomes L3/L4 and L5/S1 diminished

Box 3 Items in quality assessment of studies

Description
1 Were all relevant demographic features, namely, age and sex, described?
2 Were key features, in the history reported? These are: onset whether acute or gradual, site of pain, radiation, relieving and aggravating factors, duration, evolution of the condition, past medical history.
3 Were routine sciatica examinations reported: sensation, power, tendon reflexes, straight leg raising/Lasegue?
4 Was at least one examination specific for PS reported: tonic external rotation of foot, Freiberg sign, Pace sign, Beatty sign, Flexion-Adduction-Internal Rotation (FAIR) test?
Case definition
5 Was there corroborating evidence?
Selection
6 Applies only for case series
Was the method of selection free of bias, for example, through recruitment of consecutive cases?

Appendix 3 Tables

Table 1 Specific tests for sciatica

Name of test	Date first described	Description	Attributed to
Freiberg	1934	Passive internal rotation of the hip in extension reproduces pain.	Freiberg and Vinke [44]
Pace	1976	The clinician provides resistance to hip abduction by holding the sitting patient's knee, reproduces pain.	Pace and Nagle [91]
Tonic external rotation of hip	1981	Visible sign in patient at rest	Solheim[116]
FAIR = flexion abduction internal rotation of hip	1981	Maintaining the hip in *f*lexion *a*bduction and *i*nternal *r*otation reproduces pain.	Solheim [116]
Beatty	1994	The patient holds the flexed hip in abduction against gravity while lying on the unaffected side, reproduces pain.	Beatty [5]

Table 2 Included studies with individual data

Study: first author and year. (Language if not English)	No. in study	No. included in review	No of. routine sciatica signs reported	If signs specific to PS reported	Selection method
Adams 1980[2]	4	4	4	Yes	Not described
Barton 1991 [4]	4	4	Uncertain	Yes	Not described
Beatty[5]1994	3	3	0	Yes	Not described
Beauchesne 1997 [6]	1	1	4	No	Not applicable
Brown 1988 [13]	1	1	3	Yes	Not applicable
Bustamante 2001 [14]	2	1	3	Yes	Not described
Chantraine 1990(French) [22]	2	1	0	Yes	Not described
Chen and Wan 1992 [25]	2	2	4	Yes	Not applicable
Chen 1992 [23]	1	1	4	Yes	Not applicable
Chen 1994 [24]	1	1	4	Yes	Not applicable
Chong 2004 [26]	1	1	3	Yes	Not applicable
Colmegna 2007 [28]	1	1	1	yes	Not applicable
Dalmau 2005 [31]	1	1	0	Yes	Not applicable
Durrani and Winnie 1991 [34]	1	1	4	Yes	Not applicable
El-Rubaidi 2003(Spanish) [35]	1	1	2	No	Not described
Foster 2002 [43]	7	7	0	Yes	Not described
Freiberg 1937 [45]	2	2	1	Yes	Not described
Gandhavadi 1990 [46]	1	1	1	Yes	Not applicable
Guyomarc'h 2004 (French) [49]	3	3	Uncertain	Yes	Not described
Hanania 1998 [51]	6	6	0	No	Not described

Hopayian 1999 [53]	3	1	2	Yes	Not described
Hughes 1992 [54]	5	5	1	Yes	Not applicable
Jankiewicz 1991 [58]	1	1	1	Yes	Not applicable
Jroundi 2003(French) [60]	1	1	0	Yes	Not applicable
Julsrud 1989 [61]	1	1	Uncertain	Yes	Not applicable
Karl 1985 [62]	1	1	1	Yes	Not described
Kobbe 2008 [68]	2	2	1	Yes	Not described
Kosukegawa 2006 [70]	1	1	4	No	Not applicable
Kouvalchouk 1996 (French) [71]	4	4	Uncertain	Yes	Not described
Ku 1995 [72]	1	1	4	Yes	Not applicable
Lee 2004 [74]	1	1	Uncertain	Yes	Not applicable
Lewis 2006 [75]	14	14	3	No	Not described
Mayrand 2006 [80]	1	1	3	Yes	Not applicable
Molina 2003 [85]	1	1	4	Yes	Not applicable
Nakamura 2003 [87]	2	2	0	Yes	Not described
Ozaki [90]	1	1	4	Yes	Not applicable
Papadopoulos 1990 [94]	1	1	4	Yes	Not applicable
Park 1991 [96]	1	1	3	Yes	Not applicable
Richardson 1992 [104]	1	1	1	Yes	Not applicable
Robinson 1947 [105]	2	2	4	Yes	Not applicable
Rossi 2001 [107]	1	1	1	Yes	Not described
Sayson 1994 [109]	1	1	3	Yes	Not applicable
Solheim 1981 [116]	2	2	2	Yes	Not applicable
Spinner 2001 [118]	1	1	3	Yes	Not described
Stegbauer 1997 [119]	1	1	4	Yes	Not applicable
Synek 1987 [127]	1	1	3	Yes	Not applicable

Synek 1987 [126]	4	1	4	No	Not described
Stein [120]1983	2	1	4	1	Not described
Turtas 2006 [132]	1	1	3	Yes	Not applicable
Vallejo 2004 [133]	1	1	1	Yes	Not applicable
Vandertop 1991 [136]	1	1	4	Yes	Not applicable
Wyant 1979 [141]	2	2	4	Yes	Not applicable

Table 3 Summary of history and reported signs in studies with individual data

		History*		
		Poor	Satisfactory	Good
Signs	None	1	0	0
	Routine sciatica signs only	2	1	2
	PS signs only	2	4	0
	Sciatica and PS signs	8	10	22

* The quality of history is graded according to the number of items missing in the report: Good ≤2; Satisfactory = 3 or 4; Poor ≥5. The four shades within the cells represent overall quality (history and signs) ranging from poor (no shade) to maximum achievable (darkest shade).

Table 4 Clinical features in aggregated data studies, Number (%)

Study: First author and year (Language if other than English)	Lu et a 1985 (Chinese) [78]	Durrani and Winnie 91 [34]	Indrekvam and Sudmann 02 [56]	Filler et al 2005 [40]
No. of cases	60	26	19	162
Female	21 (35)	11(42)	15 (79)	Not reported
Age range	17-70	25-62	Not reported	Not reported
Age mean	Not reported	35.5	43	Not reported
Buttock pain	60 (100)	26 (100)	19(100)	(100)
Low back pain	Not reported	13 (50)	Not reported	(42.4)
Pain on sitting	Not reported	15 (58)	Not reported	Not reported
Dyspareunia	Not reported	6 (23)	Not reported	Not reported
External tenderness	54 (90)	24 (92)	19 (100)	(70.8)
Internal tenderness	Not reported	26 (100)	Not reported	Not reported
Freiberg sign positive	60 (100)	9 (35)	19 (100)	Not reported
Pace sign positive	Not reported	8 (31)	19 (100)	Not reported
Beatty sign positive	Not reported	Not reported	Not reported	Not reported
Tonic external rotation of hip	Not reported	10 (38)	Not reported	Not reported
FAIR sign positive	Not reported	Not reported	Not reported	Not reported
SLR limited	Not reported	12 (46)	5 (23)	(40.7)
Reflexes diminished	Not reported	Not reported	Not reported	Not reported
Sensation diminished	24 (40)	Not reported	10 (53)	Not reported
Power diminished	Not reported	Not reported	3 (16)	Not reported

Table 5 Types of corroborating data with examples

Corroborating item	Examples: description and study (first author and year)
Nerve conduction studies or electromyography show extraspinal delay	EMG findings suggestive of involvement of the inferior gluteal and peroneal branches of the sciatic nerve. Case 3. Hughes et al 1992[54] Delayed responses when hip held in FAIR position. Two out of two cases. Nakamura 2003[87]
Imaging shows structural abnormality:	Hypertrophy of PM. Two cases out of two. Chen & Wan 92 [25] Hypertrophy of PM. Jankiewicz et al 91[58] T2 hypersignal at level of PM and sciatic nerve. Jroundi et al 2003 [60] Abnormal MRI neurography, suggesting entrapment at the level of the PM. 12 out of 14 cases. Lewis et al 2006 [75]
Operative findings of abnormalities of PM and/or of sciatic nerve and/or of sciatic nerve impingement	Calcified PM. Beauchesne & Schutzer 1997 [6] Sciatic nerve impinged between PM and short external rotators. Case 1 out of two. Chen & Wan 1992[25] Tendinous band of PM indenting peroneal branch of sciatic nerve. Case 3 out of 5. Hughes et al 1992[54] Impingement of the sciatic nerve by the PM. Six out of seven cases. Foster 2002 [43] Impingement by the PM or by an associated fibrous band. All 4 cases that had surgery. Lewis et al 2006 [75] Anomalous division of the sciatic nerve with its superior branch passing through the PM. Case 2 out of four. Kouvalchouk 1996[71] Bifurcated Sciatic nerve with posterior cutaneous femoral nerve squeezed between the PM and the greater sciatic notch. Ozaki & Muro 1999[90]

Table 6 Excluded studies and reason for exclusion

Some details are incomplete for the following reasons:

1. Secondary titles discovered in the references of much older papers lack the bibliographic detail of modern papers.
2. The formatting of some titles retrieved from searching through Thomson Dialog was corrupted on import, particularly foreign names with accented letters. The first author is always present but in these cases subsequent authors have been curtailed to et al.

Study	Reason for exclusion
Anonymous. The piriformis syndrome. *Zeitschrift fur Orthopadie und Ihre Grenzgebiete*. 1989. **9(3):** 7	Foreign language
Anonymous. Piriformis syndrome treated with acupuncture. *California Journal of Oriental Medicine*. 2007. **20(4):** 199-200	No cases
Anonymous. Piriformis syndrome -- a 10-year study (n=918). *Acupuncture in Medicine*. 2002. **6(3):** 6	No cases
Anonymous. Steroidantihistamine injection: court finds no nursing negligence, accepts nurse as expert witness. *Legal Eagle Eye Newsletter for the Nursing Profession*. 2001. **127(6):** 691-694	Not medical publication
Anonymous. Piriformis syndrome. *Mayo Clinic women's healthsource*. 2002. **5(1):** 9	Not medical publication
Anonymous. No consensus on piriformis syndrome. *Not known*. 1999. **18(2):** Not known	Not obtainable
Anson H. The pyriformis muscle and sciatica. *J Bone Joint Surg*. 1938. **20(A):** 212-4	Not obtainable
Arifoglu Y, Surucu HS Sargon MF, Tanyeli E, Yazar F. Double superior gemellus together with double piriformis and high division of the sciatic nerve. *Surgical and Radiologic Anatomy*. 1997. **19(6):** 407-408	No cases
Babinski MA, Machado FA, Costa WS. A Rare Variation in the High Division of the Sciatic Nerve Surrounding the Superior Gemellus Muscle. *European Journal of Morphology*. 2003. **41(1):** 41-42	Not about PS
Balter K, Roayrao VM de. Piriformis syndrome: Management with caudal injections. *Pain Management*. 1991. **4(4):** 12–32	Clinical features not described
Banerjee T, Hall CD. Sciatic entrapment neuropathy Case report. *J Neurosurg*. 1976. **:** 216-7	Not about PS
Bauer P. Chronic anoperineal pain: diagnosis and strategy for evaluation. *Journal de chirurgie*. 2004. **141(4):** 225-31	No cases
Beaton L, Anson B. The sciatic nerve and the piriformis muscle: their interrelation a possible cause of coccygodynia. *J Bone Joint Surg*. 1938. **20:** 686-8	No cases
Beatty RA. Piriformis syndrome. *Journal of neurosurgery Spine*. 2006. **5(1):** 101	No cases

Bennett JD, Miller TA, Richards RS. The Use of Botox in Interventional Radiology. *Techniques in Vascular and Interventional Radiology*. 2006. **9(1):** 36-39	Not about PS
Benson ER, Schutzer SF. Posttraumatic piriformis syndrome: Diagnosis and results of operative treatment. *Journal of Bone and Joint Surgery - Series A*. 1999. **81(7):** 941-949	Clinical features not described
Benzon HT, Katz JA, Benzon HA, Iqbal MS. Piriformis syndrome: anatomic considerations a new injection technique, and a review of the literature. *Anesthesiology*. 2003. **98(6):** 1442-8	Clinical features not described
Bernard TM, Kirkaldy-Willis WH. Recognizing specific characteristics of nonspecific low back pain. *Clinical Orthopedics and related research*. 1987. **217:** 266-80	Clinical features not described
Betts A. Combined fluoroscopic and nerve stimulator technique for injection of the piriformis muscle. *Pain Physician*. 2004. **7(2):** 279-281	No cases
Bickels J, Kahanovitz N Rubert CK, Henshaw RM, Moss DP, Meller I, Malawer MM. Extraspinal bone and soft-tissue tumors as a cause of sciatica Clinical diagnosis and recommendations: analysis of 32 cases. *Spine*. 1999 Aug 1. **24(15):** 1611-6	Clinical features not described
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Corrections

The numbers in figure 2 differ slightly from an earlier version published in the European Spine Journal[52]. There were 176 excluded studies in the earlier version rather than the 172 here. The error arose in the earlier version in the following manner. The titles of excluded studies were exported by each category of exclusion from the database into a spreadsheet. The subtotals for each category were counted in the spreasheeet. Some titles had been exported under two categories in error, leading to double counting. This came to light when preparing a table of excluded studies and reasons for exclusion for the current report.

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Contributions

KH conceived and designed the project. KH and FJ developed the analytic tools. KH and RR extracted most of the data with additional contributions from FJ and SS. KH and FJ analysed the data. KH wrote the drafts with assistance from FJ. All have seen and approved this document and shorter report published in the European Spine Journal[52].

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