Nonspecific low back pain in general practice: a delicate point

A descriptive study on the clinical relevance

of four regional pain syndromes

The study presented in this thesis was performed at the Department of General Practice, Erasmus University, Rotterdam, the Netherlands, and funded by a grant of the Programme for the Stimulation of Health Research (SGO).

Financial support for the publication of this thesis from the following institutions is greatfully acknowledged:

Rotterdam Medical Research Foundation (ROMERES), Rotterdam
SMS Cendata, Nieuwegein

Printing: Offsetdrukkerij Haveka BV, Alblasserdam.
Cover: Parijs 1985, KHN © .

Njoo, Khing Hua
Nonspecific low back pain in general practice: a delicate point. A descriptive study on the clinical relevance of four regional pain syndromes/ Khing Hua Njoo. - Thesis Rotterdam. - With ref. - With summary in Dutch.
ISBN 90-74494-07-2
Subject headings: low back pain/general practice

Nonspecific low back pain in general practice: a delicate point

A descriptive study on the clinical relevance

of four regional pain syndromes

Aspecifieke lage rugpijn in de huisartspraktijk: een teer punt.
Een descriptieve studie over de klinische relevantie van vier regionale pijn syndromen.

Proefschrift

ter verkrijging van de graad van doctor
aan de Erasmus Universiteit Rotterdam
op gezag van de rector magnificus
Prof. Dr. P.W.C. Akkermans MA

en volgens besluit van het College voor Promoties

De openbare verdediging zal plaatsvinden op
woensdag 12 juni 1996 om 11.45 uur

door

Khing Hua Njoo

geboren te Bandung

Promotie commissie

Promotores:	Prof. Dr. E. van der Does
	Prof. Dr. H.J. Stam
Overige leden:	Prof. Dr. L.M. Bouter
	Prof. Dr. H.F.J.M. Crebolder
	Prof. Dr. Ir. C.J. Snijders

Contents **page**

Chapter 1

The general practitioner and patients with nonspecific low back pain

This chapter introduces the problem of nonspecific low back pain (LBP), the usual management of the general practitioner (GP) and the level of clinical knowledge on nonspecific LBP.

Introduction

LBP is usually a self-limiting symptom. It generally has no impact on the average life expectancy and major medical interventions are not necessary. The major problem of LBP is that such a benign physical condition not only incurs high costs due to sickness leave and incapacity for work, but also demands much of health care resources (Van Tulder 1995). The expenditure on LBP includes the costs of the GP, radiodiagnostics, prescriptions, the (physio)therapist, and the medical specialist in an out-patient clinic (Liu 1995).

In the Netherlands, musculoskeletal disorders, with LBP as the main cause, are the most frequent reason for sickness leave (Moens 1993). Annually, 2.5 per 1,000 employees become incapacitated for work due to LBP: a total of approximately 18,000 persons per year. In most cases, a specific cause for LBP cannot be found (Verbeek 1993). Of all reasons for visiting a GP approximately 15% are for musculoskeletal disorders, of which LBP is the most frequently occurring reason (Lamberts 1991a).

LBP classification

LBP can be classified into two major categories:

A. Specific causes

Most textbooks and reviews give a long list of differential diagnoses for LBP. Specific causes of LBP include: lumbar disc disease; infections; inflammation (ankylosing spondylitis, rheumatoid arthritis); bone diseases (osteoporosis, Paget's disease); Scheuerman's disease; spondylolithesis; fractures and malignancies. Knowledge on the pathogenesis of these diseases and, therefore, a model for diagnostics and treatment is available in most cases.

Identification of these serious diseases without unnecessary and costly examinations is a challenge to the GP. A balance has to be found between adequate referral to the medical specialist, if there are signs of specific pathology, and prevention of iatrogenic disability (Van der Does 1989). Most serious specific causes generally require diagnostic procedures and treatment by a medical specialist. For this reason, the category of specific causes of LBP will not be discussed in this thesis.

B. Nonspecific causes

Nonspecific LBP is pain localized between the lower rib cage and gluteal folds in which no specific pathology can be detected by X-ray or laboratory tests, or in which the relationship between detectable pathology and pain experienced by the patient is a matter of controversy (Spitzer 1987, Frank 1993).

The majority (90%) of LBP patients in general practice is classified as nonspecific LBP (Bywaters 1982, Deyo 1991, Lamberts 1991a). Psychological, social and cultural aspects can play an important role in the persistence of LBP complaints and the resulting disability. Therefore the GP's management will be based on an integrated strategy (Van der Does 1975, Hoekstra 1985).

Although psychosocial factors are related to chronic LBP (Remerie 1992, Weiter 1992), they are not the subject of this study and are therefore not discussed. The primary focus of this thesis is at patients with nonspecific LBP of recent onset, because most of these patients can be managed solely by the GP and prevention of chronic outcome is still possible (Van der Does 1980, Chavannes 1992).

The GP and patients with nonspecific LBP

The incidence of nonspecific LBP in general practice is reported to be 26 (Van der Velden 1991) to 33 (Lamberts 1991b) per 1000 enlisted persons per year. The incidence of specific causes of LBP is much lower than the incidence of nonspecific LBP in general practice. For example: in the Netherlands the incidence of lumbar disc prolapse is 5 and spondylitis ankylopoetica 0.4 per 1000 enlisted persons per year (Van der Velden 1991).

In contrast to the equal male-female distribution of LBP in the open population (Haanen 1984), more men than women consult their GP for LBP (Lamberts 1991b, Van der Velden 1991). Before the age of 15, consultations for LBP are rare. The

highest incidence (38/1000 enlisted persons per year) is found in the age categories 25-44 and 45-64 years (Lamberts 1991b). Most episodes (80%) resolve within 4 weeks (Lamberts 1991b), irrespective of treatment (Roland 1983).
The National Guideline on nonspecific LBP compiled by the Dutch College of GPs (NHG) is very pragmatic with regard to the diagnostics and management of nonspecific LBP. Generally, diagnostics is directed to exclude serious causes of LBP; i.e. "specific" causes. It is not considered useful to distinguish subgroups in nonspecific LBP, because of the lack of evidence that their prognosis or response on specific therapy is different than in nonspecific LBP. Radiological diagnostics are not recommended, because there is little relation between the clinical and radiological findings. Based on the generally favourable outcome of nonspecific LBP, only symptomatic relief is suggested. Management is focused on education and analgesic medication. The LBP patients are encouraged to continue their routine physical activities (Faas 1996). Bedrest is reported to have a disabling effect (Deyo 1986, Malmivaara 1995) and is therefore not recommended.
Several studies have described the diagnostic and management strategies for LBP in general practice. Based on the results of a survey among GPs, Oliemans concluded that GPs do not appear to make a diagnosis, but rather limit themselves to exclude serious causes of LBP (Oliemans 1980). The most frequently used management strategies are pain medication and physiotherapy (Van Weel 1983). Education on the natural course of LBP and associated warning signs is understood and appreciated by most patients (Broekema 1994) and generally prevents referral to the physiotherapist and the medical specialist (Roland 1989). Exercise therapy does not influence prognosis or relapse of LBP (Chavannes 1992, Faas 1992). Only few cases of LBP are referred to a medical specialist (2%), mostly for radiological diagnostics (Lamberts 1991b).

Of all LBP patients in general practice, 6% (Lamberts 1991b) to 9% (Chavannes 1992) become chronic LBP patients, i.e. LBP persisting longer than 3 months. The outcome for an individual nonspecific LBP patient cannot be predicted. Several risk factors for chronic outcome have been recognized in epidemiological studies:
1. LBP history
- LBP in the medical history (Biering-Sörenson 1983, Haanen 1984);

- more than 3 LBP episodes in the preceding year (Pedersen 1981);
- slow onset of LBP (Pedersen 1981);
- extensive distribution of pain area (Murphy 1984);
- radiating pain in the leg (Chavannes 1983, Esser 1985).

2. Life style
- smoking (Frymore 1983, Deyo 1989);
- obesity (Deyo 1989).

3. Psychosocial factors
- feelings of inadequacy (Haanen 1984);
- persons living alone (Biering-Sörenson 1986).

4. Occupation (Coste 1994)

Chronic LBP can be diagnosed in patients both with and without a specific cause of LBP. Except for distribution of the pain area and radiating pain in the leg, the risk factors for chronic outcome are not based on clinical symptoms. Ideally, patients at risk should be identified early in the course of an episode of LBP as a means of preventing disability and high costs associated with chronic LBP (Chavannes 1992, Faas 1996).

Diagnosis of nonspecific LBP

In medicine, diagnosis is not a goal in itself, but a tool for prognosis and, eventually, for (therapeutical) intervention (Wulff 1980). It is possible to distinguish between three types of diagnoses: 1) symptom diagnosis, 2) syndromes, and 3) anatomical or causal defined diagnosis.

Symptom diagnosis

is based on the presence of a single symptom or sign. A symptom diagnosis is subordinate to the other types of diagnoses, because of its temporary nature. If specific causes of the symptom are eliminated, the symptom becomes the diagnosis.

Syndrome

is defined by the presence of a fixed combination of symptoms and signs. The diagnosis is made by determining the presence or absence of specific symptoms and signs, which are matched with the syndrome definition. All syndrome definitions are arbitrary. Usually medical scientific societies have agreed on syndrome definition by means of a consensus procedure.

Anatomical or causal defined diagnosis

is usually based on pathophysiologic models. The definition incorporates knowledge of the fundamental physiological processes of the body.

(Adapted from Wulff 1980)

This classification of diagnoses reflects the growth of clinical knowledge and experience concerning a disease. For example, first there was the symptom of tachycardia. At close observation, a group of these patients exhibited a combination of goitre, exophthalmus and tachycardia (Merseburg trias). The simultaneous presence of these signs is called Basedow's disease, a syndrome. As science advanced, a hyperfunction of the thyroid gland was discovered as the underlying cause of the disease. The clinical signs which formed the basis of Basedow's definition have been replaced by paraclinical findings, including the serum thyroxin concentration. Generally, this is the historical course of knowledge in medicine (Wulff 1980). The knowledge on LBP for each level of diagnosis is explored in the following paragraphs.

Symptom diagnosis

LBP is not a disease, but a symptom (Miettinen 1989). Often, LBP eventually proves to remain a symptom (diagnosis). Symptom diagnoses are often made in general practice; other examples include "stomach ache" or "headache" (Wulff 1980, Lamberts 1991a). A symptom diagnosis is concluded if there is no indication of a specific disease. The diagnosis LBP e causa ignota (e.c.i.) or nonspecific LBP demonstrates the lack of knowledge on the underlying cause of LBP (Bywaters 1982, Deyo 1992).

Syndrome diagnosis

Nonspecific LBP comprises of several partially unknown subgroups. Attempts have been made to subdivide nonspecific LBP into syndromes. These syndromes were defined on the basis of a combination of symptoms and signs observed in clinical practice (McKenzie 1981, Nachemson 1982), pain patterns (Barker 1977), follow-up of the LBP episode (Roland 1983, Spitzer 1987), statistical clustering methods on a large number of variables (Heinrich 1985, Burton 1991, Coste 1992), or on psychological categories (McNeill 1986, Talo 1992).

The proposed classifications have low acceptance and poor implementation in medical practice, because no specific management or therapy advice followed the subdivision. In the absence of consensus as to the aetiology of nonspecific LBP, it is important to rely on clinical data to identify specific syndromes. Syndromes based on clinical observations, recognized and considered relevant by the GP, have a right to be investigated further (Malterud 1993). Several regional pain syndromes are examples of this and may be potential candidates for further investigation. Some regional pain syndromes in LBP will be reviewed in Chapter 2.

Anatomical or causal defined diagnosis

Anatomical or causal defined diagnoses have been proposed in LBP. Much depends on the training of the physician, as to which school of thought he adheres to (Allan 1989). The cause of the pain is generally ascribed to: the discus (Mooney 1987), the facet joints (Mooney 1976), the sacro-iliac joint (Koes 1991b), muscles and ligaments (Travell 1983) and the psychosocial system (Coste 1992, Weiter 1992). In contrast to the syndromes suggested above, these anatomical or causal diagnoses potentially make 'rational' therapies possible, such as: physiotherapy (Gilbert 1985, Koes 1991a, Koes 1992a) and exercise therapy (Chavannes 1992, Faas 1992, Koes 1992a), chiropractic therapy (Koes 1992a, Koes 1992b), McKenzie therapy (Ponte 1984, Stankovic 1990), local injections (Sonne 1985, Collee 1991), and psycho-therapy (Altmaier 1992). The association between complaint, lesion and therapy is, however, often hypothetical. Little evidence exists on the efficacy of the different therapies (Deyo 1983, Deyo 1991).

Conclusion

In clinical practice, it is not considered useful to distinguish subgroups for LBP, because there is no evidence that these subgroups have a prognosis or an outcome after therapy different from that of nonspecific LBP (Faas 1996). In clinical research, however, the identification of subgroups can be useful, because it may reduce the clinical heterogeneity of nonspecific LBP. Homogeneous groups with regard to symptoms, facilitate research on aetiology, clinical course and intervention effects. This implies that research on nonspecific LBP should focus primarily on diagnostics and not on therapy (Miettinen 1989).

Wulff (1980) states that the purpose of recognizing diagnostic categories is not a goal in itself, but a tool for prognosis and/or specific therapy. Chronic outcome for nonspecific LBP cannot be predicted. The GP needs to discriminate beforehand the patients with a favourable outcome from those with a chronic outcome. In the latter group, greater demand will eventually be made on the health care resources and social insurance (Chavannes 1992).

Clinical knowledge on nonspecific LBP has barely evolved from the stadium of symptom diagnosis. Research should aim to systematically distinguish subgroups based on the syndrome or the causal diagnosis level, and to establish the clinical relevance of these diagnoses. The main objective of the present study was to investigate the clinical relevance of regional pain syndromes, which will be reviewed in the next chapter, in nonspecific LBP.

References

Allan DB, Waddell G. A historical perspective on low back pain. Acta Orthop Scand 1989;60(suppl 234):1-23.

Altmaier EM, Lehman TR, Russell DW, Weinstein JN, Kao CF. The effectiveness of psychosocial interventions for the rehabilitation of low back pain: a randomized controlled trial evaluation. Pain 1992;49:329-35.

Barker ME. Pain in the back and leg: a general practice survey. Rheumatol Rehabil 1977;16:37-45.

Biering-Sörenson F. A prospective study of low back pain in a general population. I. Occurrence, recurrence and aetiology. Scand J Rehabil Med 1983; 15:71-9.

Biering-Sörenson F, Thomsen C. Medical, social and occupational history as risk indicators for low back trouble in a general population. Spine 1986;11:720-5.

Broekema F, Vingerhoets M, Balvert R, Blom J, Njoo KH. Voorlichting aan patiënten met acute aspecifieke lage-rugpijn. Huisarts Wet 1994;37:395-7.

Burton AK, Tillotson KM. Prediction of the clinical course of low back pain trouble using multivariable models. Spine 1991;16:7-14.

Bywaters EGL. The pathological anatomy of idiopathic low back pain. In: Symposium on idiopathic low back pain. American Academy of Orthopaedic Surgeons. Ed. White III AA, Gordon S. St. Louis, Mosby Company, 1982;144-77.

Chavannes AW, Gubbels JW, Post D, Rutten GEHM, Thomas S. Acute lage rugpijn in de praktijk. Huisarts Wet 1983;26:32-8.

Chavannes AW. Oefenen bij lage rugpijn. [Thesis] Rijksuniversiteit Utrecht, Utrecht 1992.

Collee G, Dijkmans BAC, Vandenbroucke JP, Cats A. Iliac Crest pain syndrome in low back pain: A double blind, randomized study of local injection therapy. J Rheumatol 1991;18:1060-4.

Coste J, Paolaggi JB, Spira A. Classification of nonspecific low back pain. II Clinical diversity of organic forms. Spine 1992;17:1038-42.

Coste J, Delecoeuillerie G, Cohen G, De Lara A, Le Parc JM, Paolaggi JB. Clinical course and prognostic factors in acute low back pain: an inception cohort study in primary care practice. BMJ 1994;308:577-80.

Deyo RA. Conservative therapy for low back pain. Distinguishing useful from useless therapy. JAMA 1983;250:1057-62.

Deyo RA, Diehl AK, Rosenthal M. How many days of bed rest for acute low back pain? N Engl J Med 1986;315:1064-70.

Deyo RA, Bass JE. Lifestyle and low back pain: the influence of smoking and obesity. Spine 1989;14:501-6.

Deyo RA. Fads in the treatment of low back pain. N Engl J Med 1991;325:1039-40.

Deyo RA, Rainville J, Kent DL. What can the history and physical examination tell us about low back pain? JAMA 1992;268:760-5.

Esser CJ, Van Weel C. Het beloop van uitstralende rugpijn. Huisarts Wet 1985;28:335-7.

Faas A. Oefentherapie bij acute lage rugpijn, een interventie-onderzoek in 40 huisartspraktijken. [Thesis] Oldenzaal 1992.

Faas A, Chavannes AW, Koes BW, Van den Hoogen JMM, Mens JMA, Smeele LJM, et al. NHG-standaard Lage-Rugpijn. Huisarts Wet 1996;39:18-31.

Frank A. Low back pain. BMJ 1993;306:901-9.

Frymore JW, Pope MH, Clements JH, Wilder DG, MacPherson B, Ashikaga T. Risk factors in low back pain: An epidemiological survey. J Bone Joint Surg 1983;65:213-8.

Gilbert JR, Taylor DW, Hildebrand A, Evans C. Clinical trial of common treatments for LBP in family practice. BMJ 1985;291:791-4.

Haanen HCM. Een epidemiologisch onderzoek naar lage rugpijn. [Thesis] Erasmus University, Rotterdam 1984.

Heinrich I, O'Hare H, Sweetman B, Anderson JAD. Validation aspects of an empirically derived classification for 'non-specific' low back pain. Statistician 1985;34:215-30.

Hirschberg CG, Froetscher L, Naeim F. Iliolumbar syndrome as a common cause of LBP: diagnosis and prognosis. Arch Phys Med Rehabil 1979;60:415-9.

Hoekstra GR. Patiënten met lage rugklachten in een huisartspraktijk. [Thesis] Van Denderen, Groningen 1982.

Koes BW, Bouter LM, Beckerman H, van der Heijden GJ, Knipschild PG. Physiotherapy exercises and back pain: a blinded review. BMJ 1991a;302:1572-6.

Koes BW, Assendelft WJ, van der Heijden GJ, Bouter LM, Knipschild PG. Spinal manipulation and mobilisation for back and neck pain: a blinded review. BMJ 1991b;303:1298-303.

Koes BW, Bouter LM, van Mameren H, Essers AH, Verstegen GM, Hofhuizen DM, Houben JP, Knipschild PG. The effectiveness of manual therapy, physiotherapy, and treatment by the general practitioner for nonspecific back and neck complaints. A randomized clinical trial. Spine 1992a;17:28-35.

Koes BW, Bouter LM, van Mameren H, Essers AH, Verstegen GM, Hofhuizen DM, Houben JP, Knipschild PG. Randomised clinical trial of manipulative therapy and physiotherapy for persistent back and neck complaints: results of one year follow up. BMJ 1992b;304:601-5.

Lamberts H. In het huis van de huisarts. Verslag van het Transitieproject. Lelystad: Meditekst 1991a.

Lamberts H, Brouwer HJ, Mohrs J. Reason for encounter- & episode- & process oriented standard output from the Transition Project. Part I. Amsterdam: Department of General Practice / Family medicine, University of Amsterdam, 1991b:262.

Liu AC, Byrne E. Cost of care for ambulatory patients with low back pain. J Fam Pract 1995;40:449-55.

Malmivaara A, Häkkinen U, Aro T, Heinrichs ML, Koskenniemi L, Kuosma E, Lappi S, Paloheimo R, Servo C, Vaaranen V, Hernberg S. The treatment of acute low back pain. Bedrest, exercises, or ordinary activity? N Engl J Med 1995;332 :351-5.

Malterud K. Medical theories derived from clinical practice. Scand J Prim Health Care 1993;11:81-2.

McKenzie RA. The lumbar spine: mechanical diagnosis and therapy. Waikanae, New Zealand, Spinal Publication, 1981.

McNeill TW, Sinkora G, Leavitt F. Psychologic classification of low back pain patients: a prognostic tool. Spine 1986;11:955-9.

Miettinen OS, Caro JJ. Medical research on a complaint: orientation and priorities. Ann Med 1989;21:399-401.

Moens GF, Dohogne T, Jacques PJ. Rugklachten bij werknemers in de gezondheidszorg. Tijdschr Soc Geneesk 1993;71:333-8.

Mooney V, Robertson J. The facet syndrome. Clin Orthop 1976;115:149-56.

Mooney V. Where is the pain coming from? Spine 1987;12:754-9.

Mooney V. The classification of low back pain. Ann Med 1989;21:321-5.

Murphy KA, Cornish RD. Prediction of chronicity in acute low back pain. Arch Phys Med Rehabil 1984;65:334-7.

Nachemson AL, Andersson GBJ. Classification of low back pain. Scand J Work Environ Health 1982;8:-134-6.

Oliemans AP. Lage rugpijn in de huisartsenpraktijk. Ned Tijdschr Fysiother 1980;3:91-3.

Pedersen PA. Prognostic indicators in low back pain. J Royal Coll Gen Pract 1981;31:209-16.

Ponte DJ, Jensen GJ, Kent BE. A preliminary report on the use of the McKenzie protocol versus Williams protocol in the treatment of low back pain. J Orthop Sports Phys Ther 1984;6:130-9.

Remerie S. Ruggespraak als ruggesteun? [Thesis] Thesis publishers, Amsterdam 1992.

Roland M, Morris R. A study of the natural history of Low Back Pain. Part II: Developments of Guidelines for trials of treatment in Primary Care. Spine 1983;8:145-50.

Roland M, Dixon M. Randomized controlled trial of an educational booklet for patients presenting with back pain in general practice. J R Coll Gen Pract 1989;39:244-6.

Sonne M, Christensen K, Hansen SE, Jensen EM. Injection of steroids and local anaesthetics as therapy for low back pain. Scand J Rheumatol 1985;14:343-5.

Spitzer WO, LeBlanc FE, Dupuis M. Scientific approach to the assessment and management of activity-related spinal disorders. A monograph for clinicians. Report of the Quebec Task Force on spinal disorders. Spine 1987; 12(suppl 7):1-59.

Stankovic R, Johnell O. Conservative treatment of acute low-back pain. A prospective randomized trial: McKenzie method of treatment versus patient education in "mini back school". Spine 1990;15;120-3.

Talo S, Rytokoski U, Puukka P. Patient classification, a key to evaluate pain treatment: a psychological study in chronic low back pain patients. Spine 1992;17:998-1011.

Travell JG, Simons DG: Myofascial pain and dysfunction. The trigger point manual. Baltimore: Williams and Wilkins. 1983.

Van der Does E. Scheiding van behandeling en kontrole: een achterhaalde zaak? Medisch Contact 1975:83-4.

Van der Does E. Huisarts en epidemioloog. Medisch Contact 1980:485-90.

Van der Does E. Lage rugpijn in de huisartspraktijk. Collegesyllabus Huisartsgeneeskunde 1989-1990, Erasmus Universiteit, Rotterdam 1989:188-218.

Van der Velden J, De Bakker DH, Claessens AAMC, Schellevis FG. Een nationale studie naar ziekten en verrichtingen in de huisartspraktijk. Basisrapport: Morbiditeit in de huisartspraktijk. Utrecht, NIVEL 1991. [Morbidity in general practice. Dutch national survey of general practice. Utrecht: Netherlands Institute of Primary Health Care (NIVEL), 1992.]

Van Tulder MW, Koes BW, Bouter LM. A cost-of-illness study of back pain in the Netherlands. Pain 1995;62:233-240.

Van Weel C. Rugklachten in de huisartspraktijk. Huisarts Wet 1983;26(suppl.H&P7):5-8.

Verbeek JHAM. Rugpijn, ook werk voor de huisarts. Tijdschr Huisartsgeneesk 1993;10:109-12.

Weiter S, Cedraschi C. Psychosocial issues in the prevention of chronic low back pain - a literature review. Baillière Clinical Rheumatology 1992;6:657-84.

Wulff H. Principes van klinisch denken en handelen. Bohn, Scheltema en Holkema, Utrecht, 1980. [Rational diagnosis and treatment. Blackwell Scientific Publications, Oxford, 1976.]

Chapter 2

Literature review of four regional pain syndromes in nonspecific LBP

This chapter presents a review of the medical literature on four regional pain syndromes in nonspecific LBP.

Introduction

Several regional pain syndromes have been described in relation to nonspecific LBP. They are usually classified in the category of fibrositis (Bernard 1987, Frank 1993). Examples of regional pain syndromes include: Myofascial Pain Syndrome (MFPS), Fibromyalgia Syndrome (FS), Iliac Crest Pain Syndrome (ICPS), and Long Ligament Pain Syndrome (LLPS). The definition of all these pain syndromes includes a 'tender point' or a 'trigger point'. The distinction between 'tender' or 'trigger' point is arbitrary, both terms indicate a site of maximal tenderness in a muscle or ligament (Travell 1983, Wolfe 1990).

In LBP literature most attention focuses on spinal pathology and nerve root compression, i.e. the research areas of orthopaedic and neuro-surgeons. Little attention is paid to musculo-ligamental structures. The main problems concerning the reports on musculo-ligamental pathology of LBP are due to confusing nomenclature, hypothetic speculations and flaws in research methodology. A few examples of confusing terms are: tender points, trigger points, myogelosis, tenoperiostosis, myofibrositis, and muscular rheumatism (Simons 1990). The syndromes are not well defined. The relationship between symptom and aetiology is merely speculative. No supportive evidence is offered for the successful treatment results claimed (Waddell 1982).

The reasons why regional pain syndromes are not diagnosed in daily practice in the Netherlands may stem from a lack of obvious organic findings and a lack of consensus concerning the definition. Nevertheless, there are several reasons to investigate regional pain syndromes. Firstly, although imprecise in definition and lacking evidence, regional pain syndromes have the advantage of being rooted in

clinical observations. In medical history, knowledge on diseases usually starts with the clinical observation of patients who show similar symptoms and signs. The hypothesis being that this similarity stems from having the same pathophysiological source, i.e. the same diagnosis (Wulff 1980). Therefore, regional pain syndromes make diagnosis possible on a syndrome level and provide for a subdivision of nonspecific LBP. This subdivision is clinically relevant, if the subgroups form a homogeneous group with regard to prognosis.
Secondly, diagnostic procedures of these syndromes, as well as treatment, can be managed by the GP. Therefore, regional pain syndromes typically belong to the domain of general practice.
In this thesis, four regional pain syndromes will be investigated in nonspecific LBP patients, including: Quadratus Lumborum Pain Syndrome (QLPS), Gluteus Medius Pain Syndrome (GMPS), Iliac Crest Pain Syndrome (ICPS), and Long Ligament Pain Syndrome (LLPS). The choice of these four regional pain syndromes is arbitrary. QLPS and GMPS are regional pain syndromes in muscles. ICPS and LLPS are regional pain syndromes in ligaments.
QLPS and GMPS, which belong to the category of MFPS, have a history of reports from internal and rehabilitation medicine (Travell 1983, 1992). ICPS has been investigated in a rheumatology clinic (University Hospital Leiden) and in four general practices in the Netherlands (Collee 1991c). The present study is a continuation of Collee's work on ICPS. There are few publications on LLPS; knowledge is based mainly on clinical observations, experience and personal reports of GPs.

Method

A Medline search of publications was conducted at regular intervals between 1988 and 1995. The index-words myofascial pain syndromes and fibromyalgia were added to the textwords trigger points and tender points. In addition, the index-word back pain were combined with the textwords iliac crest, iliolumbar ligament and sacroiliac joint. The trigger point manuals (Travell 1983, 1992) were screened for missing references.
To be included in the sections on epidemiology, aetiology and therapy of this review, only publications indexed as journal articles on humans or human material were selected.

Results

1. Quadratus Lumborum Pain Syndrome and Gluteus Medius Pain Syndrome

QLPS and GMPS are two different types of MFPS. This study will examine QLPS and GMPS only; the reason for this will be explained in the paragraph on localization.

Definition

MFPS is defined as regional pain referred from trigger points with associated dysfunction (Travell 1983).

Diagnostic criteria

The presence of a trigger point is one of the diagnostic criteria for the MFPS. A myofascial trigger point is a hyperirritable spot, usually within a taut band of skeletal muscle or in the muscle fascia, that is painful on compression (Travell 1983). Localized tenderness is considered a prerequisite of a trigger point; without localized tenderness there is no trigger point. The identification of a trigger point depends on clinical judgement. Over time Simons and Travell have published several sets of criteria (Table 2.1) to establish the presence of MFPS and trigger points (Simons 1983, Travell 1983, Simons 1990). The reason for the changes in criteria sets remains unclear; it was evidently not on the basis of epidemiological reports.

MFPS continues to be regarded by some researchers and clinicians as subjective, or simply nonexistent. To add to the confusion, the terminology used is inconsistent and sometimes confusing (Fricton 1990). For example, referred pain is used as a diagnostic criterium of

the MFPS and for the presence of a trigger point. It is also unclear how many or which criteria are necessary to diagnose the presence of the syndrome.

No international criteria, based on consensus of clinical experts in the field of MFPS, have been defined. In 1990, Simons proposed the following criteria: 1) localized tenderness and 2) referred pain; and when applicable: 3) a taut, palpable band in the muscle concerned; 4) limited stretch range of the muscle concerned, and/or 5) a 'twitch response' on needling (Simons 1990). The first two characteris

Table 2.1 Criteria for the identification of Myofascial Pain Syndrome (MFPS) and trigger points (TP).

	Travell 1983	*Simons 1983*	*Simons 1990*
Criteria			
Localized tenderness	Exquisite, focal tenderness to digital pressure, i.e. a TP in the band of taut muscle fibres.	A TP is consistently located at the spot of maximum tenderness along the length of a taut band of muscle fibres.	Exquisitly tender spot (located at one point along a taut band if it is palpable) in the muscle belly.
Referred pain	Characteristic patterns of pain that are referred from myofascial TPs, patterns that are specific to individual muscles.	Active TPs cause referred pain, usually projected to a distance, in predictable patterns specific for each muscle. These distinctive patterns are the key to recognizing which muscles are likely to be causing the pain. The TP is rarely located where the patient reports pain.	Tender spot must cause referral of pain (or change of sensation) at a distance of at least 2 cm beyond the spot of local tenderness. Pain referral is elicited in response to needle penetration of the TP or to pressure held for 10 seconds on the tender spot.
Palpable band	A taut, palpable band in the affected muscle.	The muscle that harbors the TP contains taut fibres in the form of a palpable band that tighten and shorten the muscle.	Identification of a taut band by palpation in an accessible muscle.
Limited stretch range	Weakness and restriction in the stretch range of motion of the affected muscle.	This 'ropiness' or 'nodularity' of the muscle is highly indicative of TPs, ...and is associated with some reduction in both range of motion and strength of the involved muscle.	Restricted stretch range of motion for the primary function of that muscle, if measurable.
Twitch response	A local twitch response elicited through snapping palpation or needling of the tender spot (TP).	The local twitch ... is a brief contraction of the fibres within a taut band in response to a sudden change in pressure on the TP produced by brisk rolling of the band under the finger (snapping palpation).	If the tender spot is penetrated with a needle, it must respond with a local twitch response.

	Travell 1983	*Simons 1983*	*Simons 1990*
Other criteria	**Reproduction of patient's pain** The reproduction of the patient's pain complaint by pressure on, or needling of the tender spot (TP).	**Jump sign** Is the patient's movement and vocalisation in response to pressure exerted on a TP.	
Miscellaneous			
History	A history of sudden onset during or shortly following acute overload stress, or a history of gradual onset with chronic overload of the affected muscle.		
Therapy	The elimination of symptoms by therapy directed specifically to the affected muscle.		
Major and minor criteria	Finding a site of *local tenderness* is essential to the diagnosis, but nonspecific. A *local twitch response* and *pain reproduction*, when present, are specific and strongly diagnostic of a myofascial TP. The more of the remaining findings present, the more certain is the diagnosis.	*Referred pain* and the *local twitch response* are the two unique characteristics of a TP. A *jump sign* is strongly indicative of a TP.	An active TP must have the presence of a *tender spot* and *referred pain*; and the presence of a *taut band* and *restricted stretch range* if the muscle is accessible to these examinations. At least part of the clinical pain pattern (or change in sensation of the complaint) and twitch sign must be produced either by pressure applied transcutaneously to the tender spot or by needle penetration of the TP.

References

Simons DG, Travell JG. Myofascial origins of low back pain. 1. Principles of diagnosis and treatment. Postgrad Med 1983;73:66-77.
Simons DG. Muscular pain syndromes. In: Frieton JR, Awad E ed. Advances in pain research and therapy, vol. 17. New York: Raven Press,1990:1-41.
Travell JG, Simons DG. Myofascial pain and dysfunction. The trigger point manual. Baltimore: Williams and Wilkins,1983:18.

tics are considered to be the major characteristics of MFPS. The other characteristics palpable band, limited stretch range, and twitch response, are present in the two other lists of criteria (Simons 1983, Travell 1983). Reproduction of the patient's pain and jump sign are present in only one of the different criteria sets (Simons 1983, Travell 1983).

Localization

MFPS and trigger points can be found in other parts of the body, not only in the low back region. Localizations of the trigger points and their zone of reference are described in detail in two manuals (Travell 1983, 1992). When a patient complains about pain in a certain area, the manual indicates where the corresponding trigger point(s) can be found.

In LBP patients MFPS has been described in the M. Iliocostalis Lumborum, M. Longissimus Thoracis, M. Multifidus, M. Quadratus Lumborum and M. Gluteus Medius (also called the lumbago muscle). Simons and Travell suggest that MFPS in especially the M. Quadratus Lumborum and M. Gluteus Medius is frequently found in LBP patients (Simons 1983, Travell 1992). These two muscles are highly suitable for research, because there is a clear anatomical distinction between them and the trigger point localizations are accessible without the necessity of palpation through layers of other muscles or a thick fascia. For these reasons, the present study will examine QLPS and GMPS.

History

There are many early descriptions about hardening inside the muscles; sometimes called muskelhärten (Lange 1921, Kraus 1937) or myogelosis (Jordan 1942). In Anglo-Saxon medical literature the term fibrositis is usually applied to this condition (Simons 1975). Travell's first report on trigger points in the MFPS appeared in 1942. The report dealt with 58 cases of shoulder pain, findings on physical examination, and the results of local procaine injections (Travell 1942). Some years earlier, Steindler and Luck reported the same clinical findings in the low back. They found referred pain in the leg in 451 LBP patients when a certain point in the low back was palpated. In 50% of these patients insertion of a needle in that particular point provoked both the local pain complaint and the referred pain. Both

types of pain disappeared after a single injection of procaine. The specific point was found in a ligament around the sacrum and in a muscle; which ligament or muscle was not specified (Steindler 1938).
The major criticism on the work of Simons and Travell is that MFPS symptoms are regarded as too subjective to withstand a controlled study or a study on interobserver agreement (Bennett 1990, Nice 1992, Wolfe 1992). A controlled study can prove the association between the MPFS findings and LBP when the occurrence of these findings in patients with LBP exceeds that in controls.

Epidemiology

Epidemiological studies on MFPS are rare. Studies usually deal with very specific populations. Sootsky found that in 54 pain patients of an outpatient clinic, 30% fulfilled the criteria for MFPS, of whom 75% was female (Sootsky 1989). In a study on 208 chronic pain patients, MFPS was diagnosed in 85% of the cases (Fishbain 1986). From a group of patients (n=164) with chronic pain in the head-neck area 55% had MFPS (Fricton 1985b).
Only one epidemiological study on LBP patients is available. In a retrospective study on LBP patients (n=1293) in a pain clinic, the most common muscle pain syndromes were Gluteus medius (2%), Gluteus maximus (2%), and Quadratus lumborum (1%) (Bernard 1987).
Studies on the prevalence of MFPS in general practice are unknown. It is unclear whether the epidemiological findings in these selective populations apply to patients in general practice, because the patient population in pain clinics and hospital outpatient departments is not comparable to the patient population in general practice concerning presentation of symptoms and prognosis (Wulff 1980).

Aetiology

A gold standard, i.e. an objective underlying cause for trigger points, is still not available. Studies that have attempted to establish pathophysiological evidence for trigger points are not sufficiently sound (Klemp 1982, Hagberg 1984, Fricton 1985a). Therefore clinical validation is currently not possible.
Assuming the cause to be in muscles and fascia, many studies have focused on processes such as local spasm, ischaemia or inflammation of the muscle.

An injection of 3.5% Na-lactate in the M. Trapezius of healthy volunteers provoked a local hardening in the muscle within minutes, which lasted about 30 minutes (Ruhmann 1932). Microscopy of muscle biopsies at trigger/tender point sites shows a "rubber band" morphology, i.e. regular notches along the fibre. The genesis of the "rubber band" morphology is unknown (Jacobsen 1991). A "moth eaten appearance" of the myofibres is seen in histochemical preparations, indicating focal loss of enzyme (NADH-diaphorase) activity (Hendriksson 1982).
Blood flow measurements of the afflicted muscles show no difference compared with healthy muscles (Klemp 1982). EMG studies show contradicting results: some investigators found a higher motor unit potential (Awad 1973, Dexter 1981, Fricton 1985a, Hubbard 1993), others found no activity at all (Hagberg 1984, Durette 1991).

Therapy

A diversity of therapies is available for MFPS. The point of application is the trigger point in the muscles. The therapy promoted by Travell (1983) is the "stretch and spray" therapy. By means of a vapocoolant spray the skin above the afflicted muscle is treated, whereafter the muscle will be stretched passively. After such a session the pain is reported to be diminished and the range of movement increased (Travell 1983). In a later study (n=244), even without prior application of a vapocoolant spray, the technique of post-isometric relaxation achieved long-lasting pain relief in 63% of the cases. According to this technique, the afflicted muscle was first passively stretched to a point just short of pain or to the onset of resistance to further movement. From this position, the patient carried out a prolonged gentle isometric contraction against minimal resistance by the therapist for about 10 seconds, then was told to "let go" ,i.e. to relax. A set of 3 to 5 repetitions in one session was sufficient to achieve long-lasting pain relief (Lewitt 1984). In a prospective, randomized, double-blind study "stretch and spray" proved to be superior to injection therapy. After 2 weeks, 67% of LBP patients treated with "stretch and spray" experience less pain compared with 42% treated with injection therapy (Garvey 1989).
Injecting a local anaesthetic in myofascial trigger points, sometimes combined with a steroid (Hameroff 1981) or a NSAID (Frost 1986), does not always prove to be

superior to saline injections (Frost 1980) or dry needling (Lewitt 1979, Gunn 1980). This suggests that the critical factor in pain relief is not the injected substance (Garvey 1989). The pain relief achieved with a local anaesthetic is naloxone reversible, suggesting an endogenous opioid system as mediator for the improvement (Fine 1988).
Clinical trials with laser therapy (Waylonis 1988, Ceccherelli 1989, Olavi 1989, Snijder-Mackler 1989, Thorsen 1992) and Transcutaneous Electrical Nerve Stimulation (Chee 1986, Graff-Radford 1989) give conflicting results. Unfortunately, the studies cannot be compared due to use of different equipment, different selection procedures and treatment, and the small numbers in the treatment groups.

2. Fibromyalgia Syndrome (FS)

FS is usually discussed together with MFPS, but there are some differences. The American College of Rheumatology (ACR) 1990 criteria for the classification of FS are: 1) history of widespread pain, i.e. pain complaints at left and right side of the body, and above and below the waist, and 2) pain in 11 of the 18 tender point sites on digital palpation. The 9 bilateral tender point sites are:
- the suboccipital muscle insertions,
- the anterior aspects of the intertransverse spaces at C5-C7,
- the midpoint of the upper border of M. Trapezius,
- the origins of M. supraspinatus, above the scapula spine near the medial border,
- second costochondral junctions, just lateral to the junctions on upper surfaces,
- 2 cm distal to lateral epicondyles of the elbow,
- the upper outer quadrants of the buttocks in the anterior fold of gluteal muscle,
- posterior to the greater trochanter prominence,
- the medial fat pad proximal to the joint line of the knee.

Beside these ACR criteria, sleep problems and chronic fatigue are accompanying symptoms of FS (Wolfe 1990).
The main difference between FS and MFPS is the distribution and duration of the pain. In FS, pain is widespread, whereas in MFPS it is regional. FS pain must persist for more than 3 months, whereas MFPS pain can be either acute or chronic.

Both syndromes include a 'tender point' (FS) or a 'trigger point' (MFPS) as a

diagnostic criterium. Tender points are painful spots at predefined sites. Trigger points also include painful spots by digital palpation, can be found all over the body, and must be accompanied by phenomena such as referred pain, twitch and jump sign.
Most reports deal separately with the two syndromes; but are they so distinct? In fibromyalgia patients, myofascial trigger points are present and vice versa. Also the similarity of a painful spot by digital palpation suggests a similar cause of pathology. It is even postulated that the pain starts as a regional MFPS which in time develops into a widespread pain of FS (Bennett 1990, Goldman 1991, Wolfe 1992). Literature on FS is abundant. Several reviews give good insight in the accumulation of knowledge and concepts from several clinical studies (Boissevain 1991, Cohen 1993).

3. Iliac Crest Pain Syndrome

Definition
ICPS is defined by LBP and findings of typical local tenderness over the medial part of the iliac crest, near the attachment of the iliolumbar ligament (Collee 1991c).

Diagnostic criteria
By systematic palpation of the iliac crest from lateral to medial at the dorsal aspect:
1. Local maximal tenderness is found at the medial part of the crista iliaca near the attachment of the iliolumbar ligament.
2. Tenderness is recognized by the patient as their own "typical" characteristic pain (Collee 1991c).

Localization
At the medial part of the crista iliaca near the attachment of the iliolumbar ligament.

History

ICPS has been described in earlier reports under the names of the Iliolumbar Syndrome, the Iliolumbar Ligament Syndrome, or Maigne's Syndrome (Hirschberg 1979, Broudeur 1981, Naeim 1982, Bernard 1987). In 1979 a clinical note describing two case histories of the Iliolumbar Syndrome was published (Hirschberg 1979). In 1983 a report on seven case histories described that by digital palpation the site of maximal pain could be found at the crista iliaca. These pain complaints were successfully treated by injecting a local anaesthetic at the painful spot; the authors name it the ICPS (Fairbank 1983). In the Netherlands, Collee et al. investigated this syndrome in an epidemiological study (Collee 1991a), and in a clinical trial using injection therapy (Collee 1991b).

Epidemiology

Hirschberg observed the Iliolumbar Syndrome in 50% of the LBP patients (Hirschberg 1979). In the Netherlands, the prevalence of ICPS in LBP was reported to be 53% in general practice (n=40), 33% in an occupational health service (n=124), and 58% in a university rheumatology outpatient clinic (n=40) (Collee 1991a). In general practice the prevalence of the syndrome was equally distributed between male and female LBP patients. In another Dutch study in LBP patients (n=170), however, GPs found a prevalence of 27% for ICPS (Collee 1991c). The discrepancy concerning the prevalence in the general practice settings may be attributed to selection bias or poor interobserver agreement on the presence of the ICPS.

Aetiology

ICPS is a syndrome diagnosis and the underlying cause is unknown. Various hypotheses on the local cause of ICPS indicate the involvement of bursitis, periostosis, enthesopathy or nerve entrapment. In a report on 440 patients with Iliolumbar Syndrome, most (91%) showed radiological anomalies; 'Rose-thorn' calcification or diffuse opacities were seen in the ligament, suggesting a process of inflammation and calcification (Broudeur 1981). In Collee's study (n=96), however, the presence of ICPS was not correlated with calcification on the radiographs (Collee 1990).

A postmortem study of 37 adult cadavers (Maigne 1991) showed that the insertion of the iliolumbar ligament was always located on the ventral aspect (abdominal

surface) of the iliac crest. Therefore, it may be concluded that the insertion of the iliolumbar ligament is inaccessible to palpation, because it is shielded by the iliac crest.
Moreover, this site is where the medial dorsal rami (L1 or L2) that innervates the cutaneous layers of the buttock pass the iliac crest, which make them easily accessible to palpation. In 2 of the 37 dissections performed, some rami were found to cross over the crest through a narrowed osteofibrous orifice, thus being susceptible to an entrapment neuropathy. However, based on this finding from a postmortem study the cause of ICPS may not be inferred (Maigne 1991).

Therapy
A few reports describe good results from local injections with steroids (Sonne 1985), or NSAID (Broudeur 1981), or with a local anaesthetic (Fairbank 1983). In a double-blind, randomized trial, a beneficial effect was shown on the pain score and pain severity after a local lignocaine injection compared to isotonic saline. The effect is reported to be evident in LBP patients in a rheumatology clinic (total n=24), but not in general practice (total n=17) (Collee 1991b).

4. Long Ligament Pain Syndrome
In the present study the definition and diagnostic criteria of LLPS are considered to be analogous to those of ICPS, which is also a regional pain syndrome in a ligament.

Definition
The patient complains of pain in the low back region. Maximal tenderness is found on palpation of the long dorsal sacroiliac ligament, i.e. long ligament, which lies directly caudal to the posterior superior iliac spine (PSIS).

Diagnostic criteria
1. Maximal tenderness is found on palpation of the long dorsal sacroiliac ligament.
2. Tenderness is recognized by the patient as their own "typical", characteristic pain.

Localization

Fibres of the long dorsal sacroiliac ligament connect the PSIS (and a small part of the iliac crest) with the lateral crest of the third and fourth segments of the sacrum. The ligament can be palpated directly caudal to the PSIS underneath the skin. It is covered by the fascia of the gluteus maximus muscle and feels solid on palpation, giving the impression of a bony structure (Vleeming 1994).

History

LLPS is known primarily from case histories written by GPs (Broadhurst 1989, Leblanc 1992). It has been named: Sacroiliac Joint Syndrome or Sacroiliac Dysfunction Syndrome. Generally, patients with these syndromes present with pain in the low back, slightly to one side. They invariably point to the dimple in the region of the posterior superior iliac spine as the source of their pain. Relief of the symptoms is achieved by infiltrating the point with a local anaesthetic (Broadhurst 1989, Leblanc 1992).

Personal reports from Dutch GPs indicate that LLPS is also "common knowledge" in the Netherlands. Some GPs claim good results with local injection therapy. Unfortunately, no research data have been published.

Epidemiology

In a retrospective study of 1293 LBP patients referred to a pain centre, Sacroiliac Joint Syndrome was found in 23% (Bernard 1987). From a survey in 394 women with peripartum pelvic pain, 42% of the women indicated that their pain was located in the area of the long ligament (Mens 1992).

Aetiology

Aetiology of LLPS is hypothetical; much is inferred from the function of the long ligament. Until recently, LBP in the sacroiliac region was attributed to a blockade or hyper/hypomobility of the sacroiliac joint; the role of the sacroiliac ligaments is also acknowledged. The stability of the sacroiliac joint arises primarily from the surrounding ligaments. The sacrotuberous ligament (Vleeming 1989) and the long dorsal sacroiliac ligament (Vleeming 1996) seem important in this respect. The surrounding muscles: quadratus lumborum, erector spinae, gluteus maximus,

gluteus minimus, piriformis en latissimus dorsi, only contribute by means of their fascia to the anterior and posterior ligaments of the sacroiliac joint. In a postmortem study, it was found that displacement of the sacroiliac joint altered the tension in the long ligament. Pain in the long ligament is possibly caused by an enthesopathy (Vleeming 1994).
The second hypothetical cause of LLPS is attributed to episacroiliac lipoma. Reports on episacroiliac lipoma were published for the first time in 1937 (Reis 1937). In fact, the term lipoma is slightly inappropriate, since the pain is caused by herniation of the fat through the fascia (Pace 1975). Thirty-five years after the Reis report, four case histories of patients with episacroiliac lipomas were published in the American Family Physician journal; these patients were cured by surgical excision of the fat mass (Pace 1972).

Therapy
In patients with Sacroiliac Joint Syndrome or Sacroiliac Dysfunction Syndrome, good results are reported with manipulation techniques, local injections, oral NSAID or simply rest (Bernard 1987, Leblanc 1992). Unfortunately, none of the reported studies are comparative studies.

2.5 Separate entities

Regional pain syndromes are diagnoses, based on observations from clinical practice. Publications on regional pain syndromes deal with them separately. Whether or not regional pain syndromes occur simultaneously has not been investigated, but there are some indications that they can occur together. In a review on sacroiliac pain syndromes it was explained that a limitation in the movement of the sacroiliac joint causes a cascade of effects in the surrounding structures to an ultimate contracture of the quadratus lumborum muscle (Bayens 1986). A retrospective study including 1293 LBP patients reported a combination of regional pain syndromes in 31 (8%) out of 387 patients with the Sacroiliac Joint Syndrome (SJS), Maigne's Syndrome (MsS) and MFPS in the gluteus maximus, medius and quadratus lumborum muscles. Although different names were used, the description of Sacroiliac Joint Syndrome (SJS) resembles that of LLPS, and Maigne's Syndrome (MsS) that of ICPS (Bernard 1987).

Conclusion

Each regional pain syndrome has been described at various times under different names. There is no consensus concerning the diagnostic criteria or syndrome definition. To add to the confusion the definitions tend to change over time. No evidence of a sound basis for aetiology or pathogenesis is known. Studies on the application of therapeutic modalities show conflicting results. Therefore, the syndromes are generally regarded as subjective and subsequently have poor interobserver agreement.

Although imprecise in definition and lacking evidence, regional pain syndromes have the advantage of being rooted in clinical observations and can be managed by the GP.

Furthermore, they provide for a diagnosis on a syndrome level and, consequently, a subdivision of nonspecific LBP.

Since no follow-up studies have been performed, prognosis of these four regional pain syndromes is unknown. Furthermore, there are some indications that regional pain syndromes occur simultaneously in LBP patients, but this aspect remains to be investigated.

References

Awad EA. Interstitial myofibrositis: hypothesis of the mechanism. Arch Phys Med Rehabil 1973;54:449-53.

Bayens H. Diagnoseschema voor lage rugpijn. Nieuwe Kompas voor de huisarts 1986; III/5:1-21.

Bennett RM. Myofascial pain syndromes and the Fibromyalgia Syndrome: a comparative analysis. In: Fricton JR, Awad E ed. Advances in pain research and therapy, vol. 17. New York: Raven Press, Ltd. 1990:43-65.

Bernard TN, Kirkaldy-Willis WH. Recognizing specific characteristics of nonspecific low back pain. Clin Orthop 1987;217:266-80.

Boissevain MD, McCain GA. Toward an integrated understanding of Fibromyalgia Syndrome. I. Medical and pathophysiological aspects. II. Psychological and phenomenological aspects. Pain 1991;45:-227-38 and 239-48.

Broadhurst NA. Sacroiliac dysfunction as cause of low back pain. Aust Fam Physician 1989;18:623-8.

Broudeur P, Larroque CH, Passeron R, Pellegrino J. Les aspects nouveaux du syndrome ilio-lombaire (SIL). Lyon Mediterraneé Medicine du Sud-Est 1981;17:4708-33.

Ceccherelli F, Altafini L, Lo Castro G, Avila A, Ambrosio F, Giron GP. Diode laser in cervical myofascial pain: a double blind study versus placebo. Clin J Pain 1989;5:301-4.

Chee EK, Walton H. Treatment of trigger points with microamperage TENS. J Manipul Phys Ther 1986;9:131-4.

Cohen ML, Quinter JL. Fibromyalgia Syndrome, a problem of tautology. Lancet 1993;342:906-9

Collee G, Kroon HM, Dijkmans BAC, Vandenbroucke JP, Cats A. Radiological findings in relation to clinical features in low back pain. J Orthop Rheumatol 1990;3:147-54.

Collee G, Dijkmans BAC, Vandenbroucke JP, Cats A. Iliac Crest pain syndrome in low back pain: Frequency and features. J Rheumatol 1991a;18:1064-7.

Collee G, Dijkmans BAC, Vandenbroucke JP, Cats A. Iliac Crest pain syndrome in low back pain: A double blind, randomized study of local injection therapy. J Rheumatol 1991b;18:1060-4.

Collee G. Clinical syndromes in low back pain. [Thesis] University of Leiden, Leiden 1991c.

Dexter JR, Simons DS. Local twitch response in human muscle evoked by palpation and needle penetration of a trigger point. Arch Phys Med Rehabil 1981;62:521-2.

Durette MR, Rodriquz AA, Agre JC, Silverman JL. Needle electromyographic evaluations of patients with myofascial or fibromyalgic pain. Am J Phys Med Rehabil 1991;70:154-6.

Fairbank JCT, O'Brien JP. The iliac crest syndrome. A treatable cause of low back pain. Spine 1983;8:220-4.

Fine PG, Milano R, Hare BD. The effects of myofascial trigger point injections are naloxone reversible. Pain 1988;32:15-20.

Fishbain DA, Goldberg M, Meather BR, Steele R, Rosomoff H. Male and female chronic pain patients categorized by DSM-III psychiatric diagnostic criteria. Pain 1986;26:181-97.

Frank A. Low back pain. BMJ 1993;306:901-9.

Fricton JR, Auvinen MD, Dykstra D, Schiffman E. Myofascial pain syndrome: electromyographic changes associated with local twitch response. Arch Phys Med Rehabil 1985a;66:314-7.

Fricton JR, Kroening R, Haley D, Siegert R. Myofascial pain syndrome of the head and neck: a review of clinical characteristics of 164 patients. Oral Surg 1985b;60:615-23.

Fricton JR. Myofascial Pain Syndrome. Characteristics and epidemiology. In: Fricton JR, Awad E ed. Advances in pain research and therapy, vol. 17. New York: Raven Press,1990:107-25.

Frost FA, Jessen B, Siggaard-Andersen J. A control, double blind comparison of mepivacaine injection versus saline injection for myofascial pain. Lancet 1980;1(8167):499-501.

Frost A. Diclofenac versus lidocaine as injection therapy in myofascial pain. Scand J Rheumatol 1986;15:-153-6.

Garvey TA, Marks MR, Wiesel SW. A prospective, randomized, double-blind evaluation of trigger-point injection therapy for low back pain. Spine 1989;14:962-4

Goldman LB, Rosenberg NL. Myofascial pain syndrome and fibromyalgia. Seminars in neurology 1991;11:-274-80.

Graff-Radford SB, Reeves JL, Baker RL, Chiu D. Effects of transcutaneous electrical nerve stimulation on myofascial pain and trigger point sensitivity. Pain 1989;37:1-5.

Gunn CC, Milbrandt WE, Little AS, Mason KE. Dry needling of muscle motor points for chronic low back pain. A randomized clinical trial with long term follow-up. Spine 1980;5:279-91.

Hagberg M, Kvarnström S. Muscular endurance and electromygraphic fatigue in myofascial shoulder pain. Arch Phys Med Rehabil 1984;65:522-5.

Hameroff SR, Crago BR, Blitt CD, Womble J, Kanel J. Comparison of bupivacaine, etidocaine and saline for trigger point therapy. Anesth Analg 1981;60:752-5.

Hendriksson KG, Bengtsson A, Larsson J, Lindström F, Thornell LE. Muscle biopsy findings of possible diagnostic importance in primary fibromyalgia (fibrositis, myofascial syndrome). Lancet 1982;2(8312):1395.

Hirschberg CG, Froetscher L, Naeim F. Iliolumbar syndrome as a common cause of LBP: diagnosis and prognosis. Arch Phys Med Rehabil 1979;60:415-9.

Hubbard DR, Berkoff GM. Myofascial trigger points showing spontaneous needle EMG activity. Spine 1993;18:1803-7.

Jacobsen S, Bartels EM, Danneskiold-Samsoe B. Single cell morphology of muscle in patients with chronic muscle pain. Scand J Rheumatol 1991;20:336-43.

Jordan HH. Myogelosen: the significance of pathologic conditions of the musculature in disorders of posture and locomotion. Arch Phys Ther 1942;23:36-54.

Klemp P, Nielsen HV, Korsgard J, Crone P. Blood flow in fibromyotic muscles. Scand J Rehabil Med 1982;14:81-2.

Kraus H. Behandelung akuter Muskelhärten. Wien Klin Wochenschr 1937;50:1356-7.

Lange F, Eversbusch G. Die Bedeutung der Muskelhärten fur die allgemeine praxis. Munch Med Wochenschr 1921;68:418-20.

Leblanc KE. Sacroiliac sprain: an overlooked cause of back pain. Am Fam Phys 1992;46:1459-63.

Lewitt K. The needle effect in the relief of myofascial pain. Pain 1979;6:83-90.

Lewitt K, Simons DG. Myofascial pain: relief by post-isometric relaxation. Arch Phys Med Rehabil 1984;65:452-6.

Maigne JY, Maigne R. Trigger point of the posterior iliac crest: painful iliolumbar ligament insertion or cutaneous dorsal ramus pain? An anatomic study. Arch Phys Med Rehabil 1991;72:734-7.

Mens JAM, Stam HJ, Stoeckart R, Vleeming A, Snijders CJ. Peripartum pelvic pain; a report of analysis of an inquiry among patients of a Dutch patient society. Proceedings of the First International Congress on

Low Back Pain and the SI joint. San Diego, 1992.

Naeim F, Froetscher L, Hirschberg GG. Treatment of the chronic iliolumbar syndrome by infiltration of the iliolumbar ligament. West J Med 1982;136:372-4.

Nice DA, Riddle DL, Lamb RL, Mayhew TP, Rucker K. Intertester reliability of judgments of the presence of trigger points in patients with low back pain. Arch Phys Med Rehabil 1992;73:893-98.

Olavi A, Pekka R, Pertti K, Pekka P. Effects of the infrared laser therapy at treated and non-treated trigger points. Acupuncture Electro-therapeutics Res Int J 1989;14:9-14.

Pace JB, Henning C. Episacroiliac lipoma. Am Fam Physician 1972;6:70-3.

Pace JB. Commonly overlooked pain syndromes responsive to simple therapy. Postgrad Med 1975;58:107-13.

Reis E. Episacroiliac lipoma. Am J Obstet Gynecol 1937;34:490-4.

Ruhmann W. Uber das wesen der rheumatischen muskelhärte. Dtsch Arch Klin Med 1932;173:625-45.

Simons DG. Muscle pain syndromes. Part I. Am J Phys Med 1975:64:289-311.

Simons DG, Travell JG. Myofascial origins of low back pain. 1. Principles of diagnosis and treatment. Postgrad Med 1983; 73:66-77.

Simons DG. Muscular pain syndromes. In: Fricton JR, Awad E ed. Advances in pain research and therapy, vol. 17. New York: Raven Press, 1990:1-41.

Snijder-Mackler L, Barry AJ, Perkins AI, Soucek MD. Effects of helium-neon laser irradiation on skin resistance and pain in patients with trigger points in the neck or back. Phys Ther 1989;69:336-41.

Sonne M, Christensen K, Hansen SE, Jensen EM. Injection of steroids and local anaesthetics as therapy for low back pain. Scand J Rheumatol 1985;14:343-5.

Sootsky SA, Jaeger B, Oye RK. Prevalence of myofascial pain in general internal medicine practice. West J Med 1989;151:157-160.

Steindler A, Luck JV. Differential diagnosis of pain low in the back. JAMA 1938;110:106-13.

Thorsen H, Gam AN, Svensson BH, Jess M, Jensen MK, Piculell I, Schack LK, Skjott K. Low level laser therapy for myofascial pain in the neck and shoulder girdle. A double blind, cross over study. Scand J Rheumatol 1992;21:139-41.

Travell J, Rinzler S, Herman M. Pain and disability of the shoulder and arm. JAMA 1942;120:417-22.

Travell JG, Simons DG: Myofascial pain and dysfunction. The trigger point manual. Baltimore: Williams and Wilkins. 1983.

Travell JG, Simons DG: Myofascial pain and dysfunction. The trigger point manual. Baltimore: Williams and Wilkins. 1992.

Vleeming A. The sacrotuberous ligament: a conceptual approach to its dynamic role in stabilizing the sacroiliac joint. Clin Biomech 1989;4:201-3.

Vleeming A, Pool-Goudzwaard AL, Hammudoghlu D, Stoeckart R, Snijders CJ, Mens JMA. The function of the long dorsal sacroiliac ligament; its implication for understanding low back pain. Spine 1996 (in press).

Waddell G. An approach to backache. Br J Hosp Med 1982;28:187-219.

Waylonis GW, Wilke S, O'Toole D, Waylonis DA, Waylonis DB. Chronic myofascial pain: management by low-output helium-neon laser therapy. Arch Phys Med Rehabil 1988;69:1017-20.

Wolfe F, Smythe HA, Yunus MB, Bennett RM, Bombardier C, Goldenberg DL, et al. The American College of Rheumatology 1990 criteria for the classification of fibromyalgia. Report of the multicenter criteria committee. Arthritis Rheum 1990;33:160-72.

Wolfe F, Simons DG, Fricton J, Bennett RM, Goldenberg DL, Gerwin R, et al. The fibromyalgia and myofascial pain syndromes. J Rheumatol 1992;19:944-51.

Wulff H. Principes van klinisch denken en handelen. Bohn, Scheltema en Holkema, Utrecht, 1980. [Rational diagnosis and treatment. Blackwell Scientific Publications, Oxford, 1976.]

Chapter 3

Research on regional pain syndromes

This chapter introduces the aims of the present study and describes the material and methods used.

Introduction

When a certain pattern of symptoms occurs repeatedly in different patients, then the basic assumption is that there is probably a common cause of the medical problem in these patients. Patients with a similar pattern of symptoms, i.e. a syndrome, are looked upon as a subgroup. The main objective of the present study is to investigate the clinical relevance of regional pain syndromes in nonspecific LBP. Research on clinical syndromes, such as regional pain syndromes, is confronted with many problems.

Firstly, there is no gold standard, because aetiology is unknown. Therefore, it is not possible to perform a study on the sensitivity and specificity of the diagnostic criteria of the syndrome, although some attempts have been made. Cobb on rheumatoid arthritis (Cobb 1960) and Wolfe on FS (Wolfe 1992) have considered the clinical judgement of the medical specialist as the gold standard. After the medical specialist had given his opinion whether the syndrome was present or not, the sensitivity and specificity of the classification criteria, usually defined by international consensus procedures, were investigated. The interpretation of this kind of sensitivity and specificity is hard to assess. It is not sensitivity and specificity in the true sense, because no pathognomonic sign or test was involved (Cohen 1993).

Secondly, clinical descriptions are highly inconsistent. The definitions and diagnostic criteria are generally not stated clearly and no hierarchy in the diagnostic criteria is presented. This causes confusion in the discussion and evaluation of regional pain syndromes (Nachemson 1982). A clear description and international consensus on the diagnostic criteria of regional pain syndromes could serve to address the problem of subjective and biased observations (Wolfe 1990).

However, some aspects of regional pain syndromes can be investigated without a gold standard. The first goal is to refine the diagnostic criteria for the regional pain syndromes. In radiological imaging studies, the association between the radiological findings and the disease is demonstrated by comparing the occurrence of the abnormal radiological findings in patients with the disease and in controls. If abnormal findings are found in a substantial number of controls, it is concluded that these findings have no relation with the disease (Witt 1984). Analogous to Witt's study design, in the present study design, the presence of the diagnostic criteria for the four regional pain syndromes are compared in LBP patients and in controls. Diagnostic criteria, that are present almost exclusively in LBP patients and rarely in controls, are useful and should be included in the set of diagnostic criteria for each pain syndrome (Wulff 1980, Campbell 1986).
The second aspect is to determine the interobserver agreement on the diagnostic criteria. Especially in clinical syndromes without a known source of pathology, it is crucial that the interobserver agreement on the diagnostic criteria is quantified. Characteristics that have low interobserver agreement are not suitable to be incorporated in the set of diagnostic criteria.
The third aspect is to determine the prognosis of the regional pain syndromes. The clinical relevance of regional pain syndromes is established if LBP with a regional pain syndrome has a prognosis other than LBP without a regional pain syndrome. This would suggest that regional pain syndromes form homogeneous entities within the diffuse group of nonspecific LBP.
Publications on these syndromes deal with them separately. Whether or not regional pain syndromes occur simultaneously has not been investigated

The four regional pain syndromes described in Chapter 2 and investigated in this study are:

a. Quadratus Lumborum Pain Syndrome (QLPS)
b. Gluteus Medius Pain Syndrome (GMPS)
c. Iliac Crest Pain Syndrome (ICPS)
d. Long Ligament Pain Syndrome (LLPS)

The objectives of the present study are to:

1. Refine the diagnostic criteria for the four regional pain syndromes by investigating the association on the diagnostic criteria with LBP, and the interobserver agreement of the diagnostic criteria.
2. Describe the occurrence of the four regional pain syndromes in LBP patients in general practice.
3. Determine the prognosis of these regional pain syndromes.
4. Investigate the occurrence of combinations of regional pain syndromes.

The research questions for each regional pain syndrome include:
1. Which diagnostic criteria are present in LBP patients compared with control subjects?
2. What is the level of interobserver agreement on the diagnostic criteria and these regional pain syndromes?
3. What is the occurrence of each regional pain syndrome in general practice?
4. What is the prognosis of each regional pain syndrome after 4 weeks and after 5 years?
5. What is the occurrence of a combination of regional pain syndromes?

Results of the first three questions are presented in Chapter 4 for QLPS and GMPS, in Chapter 5 for ICPS and in Chapter 6 for LLPS. In Chapter 7 the results on prognosis are presented and in Chapter 8 the occurrence of a combination of regional pain syndromes.

Material and methods

Patient population

The patients and controls were recruited during two consecutive 3-month periods in winter of 1989 and in spring of 1990. In the first period the participants came from one health centre (4 GPs) and 2 private practices (2 GPs) in a semi-rural community; and in the second period from one health centre (5 GPs) in a suburb of the city of Rotterdam. During office hours the patients and controls were invited by their GPs to participate. It was explained that the study was performed solely for the purpose of research and that the results would not affect their present evaluation or therapy.

Selection criteria

The participants were selected as follows:

1. Inclusion criteria:

   age between 20 and 60 years;

   able to complete a written questionnaire.

2. Exclusion criteria:

- concurrent signs of malaise or fever or involuntary weight loss;
- concurrent malignant disease;
- current treatment by a neurologist;
- a previous operation in the low back region;
- pathological reflexes and positive Straight Leg Raising test on physical examination by the GP;
- pregnancy at the time of study;
- psychiatric history (past and present).

LBP patients were subjects who had consulted their GP for LBP. The present LBP episode had started less than 2 months prior to this consultation and the preceding LBP episode had been at least 3 months ago, i.e. disease free interval. LBP was defined as pain between Th12 and the gluteal folds.

Control subjects were the tenth patient on the daily appointment list who met the selection criteria and who consulted their GP for reasons other than LBP. All control subjects were asked to confirm that they did not have LBP at the time of the consultation. If the patient did not fulfil the selection criteria, the next eligible patient was asked.

Procedure

The study was carried out at the location of the participating health centres. All participating patients received an information leaflet about the study objectives and protocol. The GP's assistant scheduled the appointments. Most participants were seen immediately after their visit to the GP.

All participants answered a written semi-structured questionnaire and underwent a standard physical examination. The questionnaire investigated participants' socio-

demographic and medical variables. Each participant was examined by two observers. The first observer, who was aware of the participant's status; i.e. LBP patient or control, performed a standard physical examination. The examination consisted of a general (orthopaedic/neurological) examination for LBP and an examination of the characteristics of the regional pain syndromes under study. The findings were recorded on a form. The first observer then positioned the participant on the examination couch so that the second examiner could start his examination. The second observer, who was unaware of the participant's status, examined and registered only the characteristics of the regional pain syndromes under study (Appendix I, II, III).

Observers

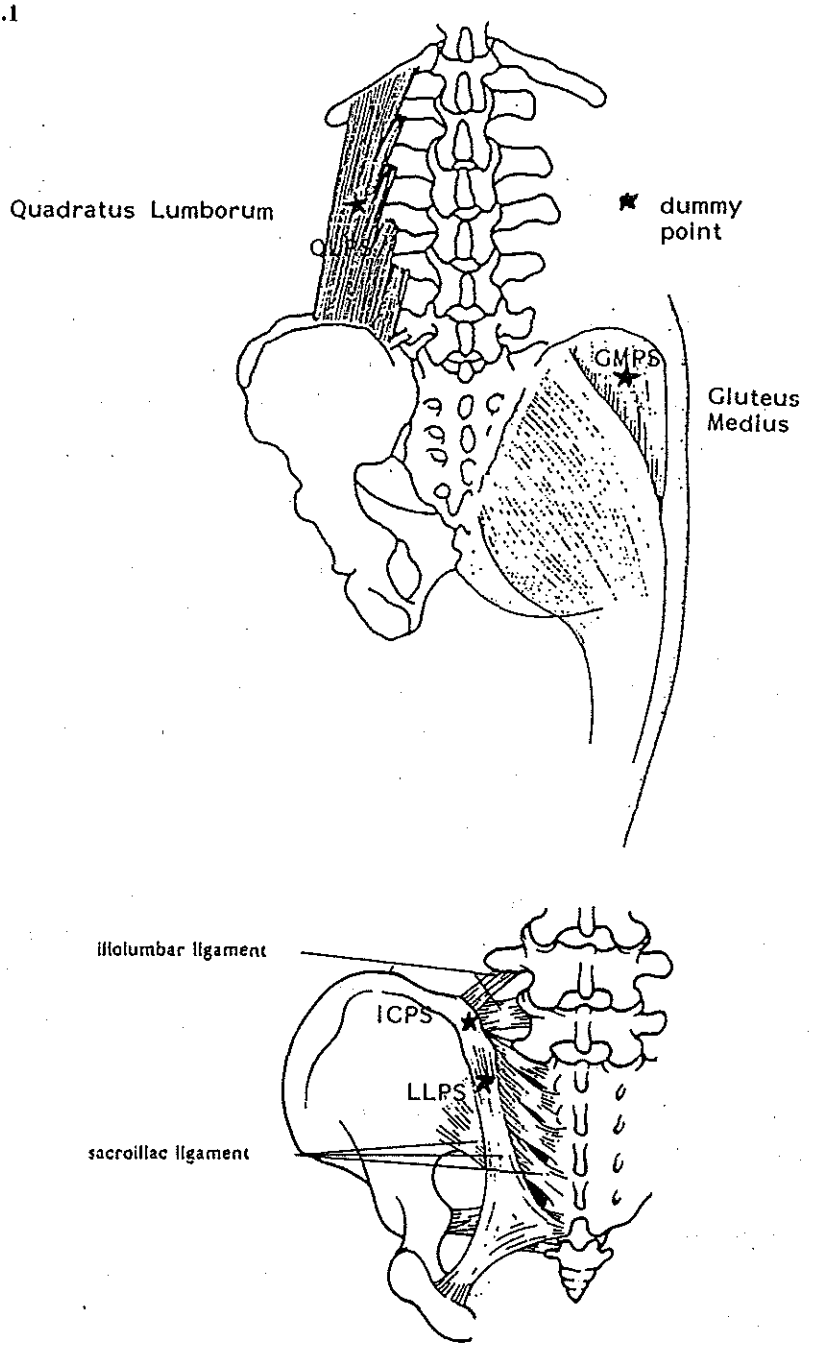
The observers were an experienced GP (KN) with additional training in the department of rheumatology at a university hospital (Leiden), and medical students who had completed their internship and were soon to attain their medical degree. Each 3-month period, two medical students were trained by the GP. Together with the researcher there was always a team of three observers. Any combination of two could act as a first and a second observer.

Pre-study training

Before data collection, a training session was held to ascertain that the examiners had similar interpretations of all items of the physical examination. The training consisted of the performance, interpretation and registration of the physical examination. A force of approximately 2 kg applied with the index finger on a balance was used as training for application of the standard pressure. This is well below the pressure threshold of pain in healthy persons. A threshold in a muscle at a pressure of 3 kg or less can be considered as abnormal (Fisher 1987).

The examination of the pre-study patients (n=15) was registered independently, and compared and evaluated with the patient present. If there was disagreement, the patient was examined again. The patients were also asked to give feedback as to which of the examiners pressed harder.

Figure 3.1


Quadratus Lumborum
dummy point
GMPS
Gluteus Medius
iliolumbar ligament
ICPS
LLPS
sacroiliac ligament

When the actual study was performed, the LBP patients were asked to return 2 and 4 weeks after the first examination to ascertain that the examiners still performed consistently. The follow-up sessions at 2 and 4 weeks were carried out in a similar way to the pre-study practice examinations.

Physical examination

The following anatomical structures were palpated bilaterally for tenderness and other characteristics (Figure 3.1):

1. Trigger point in the medial part of the M. Quadratus lumborum: the patient was positioned on his side on the examining table. The uppermost arm was abducted above his head and his knees bent, with the uppermost knee behind the other knee. The lateral border of the Quadratus lumborum muscle was palpated with the tip of the index finger. The trigger point was located in the middle between the thoracal rib and the iliac crest (Travell 1992).
2. Trigger point in lateral upper quadrant of the M. Gluteus medius: the patient was positioned prone. The muscle was palpated in the upper lateral quadrant of the buttock with the tip of the index and middle finger (Travell 1992).
3. Point of tenderness near the insertion of the iliolumbar ligament at the Iliac Crest: the patient was positioned prone. The iliac crest was palpated from lateral to medial. The tender point was located at the medial part of the iliac crest near the attachment of the iliolumbar ligament.
4. Long Ligament: the patient was positioned prone. The area dorsal to the bony land mark of the Posterior Superior Iliac Spine was palpated.

The following diagnostic criteria were assessed by palpation of the four locations described above:

1. localized tenderness = a spot of maximal tenderness.
2. recognition = patient's recognition of the pain as their characteristic pain.

The criteria were scored as "present" or "absent". For QLPS and GMPS the following criteria were added to the examination:

3. referred pain = a pattern of referred pain. Referred pain was considered present when any referred pain was experienced by the patient on palpation of the painful spot. The pattern was recorded on a mannequin drawing.

4. palpable band = a taut band or knot in the affected muscle.
5. twitch response = an involuntary contraction of the muscle.
6. limited stretch range = stretch range was considered limited, when there was a left to right difference. For QLPS lateral flexion of the trunk and for GMPS adduction of the upper leg were tested. According to the study protocol the second observer did not perform the tests for limited stretch range, because this required the patient standing up from the examination couch and moving around, thereby unblinding the situation.
7. jump sign = patient vocalisation or withdrawal.

Dummy point

A dummy point, 2 cm lateral to the border of the spinal muscles, was also examined bilateral for tenderness. In this area no trigger or tender points have been described (Wolfe 1990, Travell 1992). In the present study this point served as a test of low pressure threshold in the subjects examined (Figure 3.1).

Follow-up

Prognosis was investigated by follow-up of the LBP patients at 2 and 4 weeks, and at 5 years. At 2 and 4 weeks, the LBP patients were examined again at the health centres.

Statistical analysis

Chi-square test for proportions or, when appropriate, Fisher's exact test were used to detect statistically significant differences of the nominal variables. Student's t-test or one-way analysis of variance (ANOVA) were used to detect statistically significant differences of means. A p-value of <0.05 was considered as statistically significant.

Several measures of interobserver agreement were calculated: percentage of agreement, kappa, the prevalence index (PI), the bias index (BI) and prevalence adjusted kappa (PAK).

Percentage of agreement indicates the observed proportion of concordance and does not correct for chance agreement. Kappa is adjusted for the proportion of agreement that is expected by chance alone (Cohen 1960).

Interobserver agreement studies on physical signs in LBP provide an estimate as to what is a reasonable cut-off level for kappa. On intuition, a cut-off level of 0.8 is considered as reasonable, but the general level of interobserver agreement on relevant signs for the physical examination in the low back ranges between a kappa of 0.4 for pain in the leg at the Straight Leg Raising test (McCombe 1989) and 0.6 for root compression signs (Waddell 1982). This is at about the ususal level for most components of the clinical examination (Sackett 1985). Therefor, for the present study a kappa of 0.5 was selected as a cut-off point of good interobserver agreement.

Another problem of kappa is that it is very sensitive to small prevalences, resulting in the paradox of high percentage of agreement and low kappa (Feinstein 1990). Kappa can be adjusted for the prevalence and/or the bias effect by calculating kappa with the mean of the concordant or the discordant cells. The prevalence index (PI) is an index of differences between the overall proportion of "Yes" and "No" assessments. The bias index (BI) is an index of the bias between examiners. Prevalence adjusted kappa (PAK) allows better comparison despite the prevalence in a certain population (Byrt 1993). PAK is presented here to serve for future comparison.

The interobserver agreement values were calculated on the basis of the observations on the left and right side of the body. Thus the number of observations was 122 (2 x 61) for LBP patients and 126 (2 x 63) for controls. Table 3.1 and Note 3.1 shows how interobserver agreement values and the indexes were calculated. Analyses were performed with the Statistical Package for the Social Sciences (SPSS) version 5.01.

Table 3.1 Indexes on interobserver agreement

		First observer		
		positive	negative	
Second observer	positive	a	b	Sp=a+b
	negative	c	d	Sn=c+d
		Fp=a+c	Fn=b+d	N

Note 3.1

% agreement = (a + d)/N

$$\text{Kappa} = \frac{\text{observed agreement - expected agreement}}{100 - \text{expected agreement}}$$

$$= \frac{(a + d)/N - [\{(Fp \times Sp)/N\} + \{(Fn \times Sn)/N\}]/N}{100 - [\{(Fp \times Sp)/N\} + \{(Fn \times Sn)/N\}]/N}$$

Ppos = a : {(Fp+Sp)/2}
Pneg = d : {(Fn+Sn)/2}

BI = (b - c) / N
PI = (a - d) / N

PAK = kappa calculated with the mean number of positive and negative agreement: a and d are replaced by g = (a + d)/2

References

Byrt T, Bishop J, Carlin JB. Bias, prevalence and Kappa. J Clin Epidemiol 1993;46:423-9.

Campbell SM. Is the tender point concept valid? Am J Med 1986;81(suppl3A):33-7.

Cobb S. On the development of diagnostic criteria. Arthritis Rheum 1960;3:91-5.

Cohen J. A coefficient of agreement for nominal scales. Educ Psychol Meas 1960; 20:37-46.

Cohen ML, Quinter JL. Fibromyalgia syndrome, a problem of tautology. Lancet 1993;342:906-9.

Feinstein AR, Cicchetti DV. High agreement but low kappa: I. The problems of two paradoxes. J Clin Epidemiol 1990;43:543-549.

Fisher AA. Pressure algometry over normal muscles. Standard values, validity and reproducibility of pressure threshold. Pain 1987;30:115-26.

McCombe PF, Fairbank JCT, Cockersole BC, Pynsent PB. Reproducibility of physical signs in low back pain. Spine 1989:14:908-18.

Nachemson AL, Andersson GBJ. Classification of low-back pain. Scan J Work Environ Health 1982;8:134-6.

Sackett DL, Haynes RB, Tugwell P. Clinical epidemiology. A basic science for clinical medicine. Little, Brown and Comp, Boston, 1985.

Travell JG, Simons DG: Myofascial pain and dysfunction. The trigger point manual. Baltimore: Williams and Wilkins, 1992.

Waddell G, Main CJ, Morris EW, Venner RM, Rae PS, Sharmy SH, et al. Normality and reliability in the clinical assessment of backache. BMJ 1982;284:1519-23.

Witt I, Vestergaard A, Rosenklint A. A comparative analysis of X-ray findings of the lumbar spine in patients with and without lumbar pain. Spine 1984;9:298-300.

Wolfe F, Smythe HA, Yunus MB, Bennett RM, Bombardier C, Goldenberg DL, et al. The American College of Rheumatology 1990 criteria for the classification of fibromyalgia. Report of the multicenter criteria committee. Arthritis Rheum 1990;33:160-72.

Wolfe F, Simons DG, Fricton J, Bennett RM, Goldenberg DL, Gerwin R, et al. The fibromyalgia and myofascial pain syndromes. J Rheumatol 1992;19:944-51.

Wulff H. Principes van klinisch denken en handelen. Bohn, Scheltema en Holkema, Utrecht, 1980. [Rational diagnosis and treatment. Blackwell Scientific Publications, Oxford, 1976.]

Chapter 4

Results: Quadratus Lumborum Pain Syndrome (QLPS) and Gluteus Medius Pain Syndrome (GMPS)

This chapter presents the results on: 1) the association between the diagnostic criteria for QLPS and GMPS, and LBP; 2) the interobserver agreement; and 3) the occurrence of QLPS and GMPS in general practice.

Introduction

QLPS and GMPS are part of Myofascial Pain Syndrome (MFPS). MFPS is defined as: "Regional pain referred from trigger points with associated dysfunction." (Travell 1983). A trigger point is a hyperirritable spot, usually within a taut band of skeletal muscle or in the muscle fascia, that is painful under compression and can give rise to characteristic referred pain, localized tenderness and autonomic phenomena. Localized tenderness is considered a prerequisite for a trigger point; without localized tenderness there is no trigger point (Travell 1983). The identification of a trigger point is dependent on the clinical examination of the physician.

Over time, Simons and Travell have published several sets of criteria to establish the presence of MFPS and trigger points (Simons 1983, Travell 1983, Simons 1990). However, their description of the diagnostic criteria is inconsistent and confusing. The phrasing of the symptom of referred pain varies: "Characteristic patterns that are specific to individual muscles are referred from myofascial trigger points. The trigger point is rarely located where the patient reports pain." (Travell 1983), or "The tender spot must cause referral of pain, or change of sensation, at a distance of at least 2 cm beyond the spot of local tenderness" (Simons 1990). In addition, referred pain is also a diagnostic criterium of MFPS and a criterium for the presence of a trigger point (Chapter 2; Table 2.1). For these reasons, MFPS continues to be regarded by some researchers and clinicians as subjective, or simply nonexistent (Fricton 1990).

The rationale behind the changes in the reported criteria sets remains unclear. Recognition and jump sign have disappeared from the original list (Travell 1983) without any explanation. There is no indication that these changes were based on epidemiological data.
Furthermore, no international criteria, based on consensus of clinical experts in the field of MFPS, have been defined. In 1990, Simons proposed the following criteria: 1) localized tenderness and 2) referred pain; and when applicable: 3) a taut, palpable band in the muscle concerned; 4) limited stretch range of the muscle concerned, and/or 5) a 'twitch response' on needling (Simons 1990). The first two characteristics are considered to be the major characteristics of MFPS. The other characteristics palpable band, limited stretch range, and twitch response, are present in the two other criteria lists (Simons 1983, Travell 1983). Reproduction of the patient's pain, and jump sign are present in only one of the different criteria sets (Simons 1983, Travell 1983). In the present study in addition to Simons' 1990 criteria, reproduction of the patient's pain, and jump sign will also be included in the investigation.

MFPS and trigger points can be found in various parts of the body, not only in the low back. Simons and Travell suggest that MFPS in the M. Quadratus lumborum and M. Gluteus medius is frequently found in LBP patients (Simons 1983, Travell 1992). These two muscles are highly suitable for research, because there is a clear anatomical distinction between them, and the trigger point localizations are accessible without the necessity of palpation through layers of other muscles or a thick fascia. For these reasons, this study will examine only QLPS and GMPS.

In addition to the confusion concerning definition of MFPS, the major criticism on the work of Simons and Travell is that they did not test the trigger point symptoms in a controlled study (Bennett 1990), and that MFPS symptoms are too subjective to have strong interobserver agreement (Nice 1992, Wolfe 1992). Although epidemiological studies have been performed in outpatient clinics (Fricton 1985b, Fishbain 1986, Bernard 1987, Sootsky 1989, Cassisi 1993), the occurrence of QLPS or GMPS is not known in general practice.

Therefore, the research questions addressed in this chapter are:
1) Which diagnostic criteria of QLPS and GMPS are present in LBP patients compared with control subjects?
2) What is the level of interobserver agreement on the diagnostic criteria of both pain syndromes, and QLPS and GMPS separately?
3) What is the occurrence of QLPS and GMPS in LBP patients in general practice?

Material and methods

See Chapter 3.

Results

A total of 163 participants (81 LBP patients) were invited to participate in the study. After exclusion of 5 participants (4 LBP patients) who did not meet the study criteria, 158 participants (77 LBP patients) were eligible.
Thirty-three participants (16 LBP patients) did not make an appointment or did not keep the appointment, and one subject (control) withdrew cooperation. Thus, the total nonresponse rate for the initial examination was 21%. On telephone inquiry, the reasons for nonresponse were: already returned to work, not willing to take time off, too busy, second thoughts about participating because the pain had subsided, and/or the study offered little personal benefit. There was no statistically significant difference between respondents and nonrespondents with regard to gender and age.

Finally, a total of 124 participants entered the study: 61 nonspecific LBP patients and 63 controls. There was no significant difference between patients and controls with regard to age, gender or employment status (Table 4.1).

Table 4.1 Baseline characteristics of the LBP patients and control subjects.

Characteristic	LBP patients n=61	Controls n=63
Mean age (yr)	36.2 (SD 9.8)	38.1 (9.9)
Sex (% female)	44.2	50.7
Employed (total)	46	38
Full-time	38	29
Part-time	8	9
Unemployed (total)	15	23
Compensation	6	14
Housewife	9	9
Unknown (total)		2
Mean duration of present episode (days)	11 (SD 11.8)	
Abrupt onset (no.)	30	
Pain in leg (no.)	15	
Night pain (no.)	26	
Morning stiffness > 15 minutes (no.)	12	

no. Number of patients
SD Standard deviation
Differences between LBP patients and control subjects for all variables were not significant ($p > 0.05$). Means were tested with Student's t-test; the other variables were tested with Chi-square test.

Tables 4.2 and 4.3 present the occurrence of the diagnostic criteria of QLPS and GMPS, respectively, in LBP patients compared with controls. Localized tenderness is a prerequisite for a trigger point and also for MFPS. Thus, the occurrence of the other diagnostic criteria in Tables 4.2 and 4.3, are on condition that localized tenderness was present. In both pain syndromes the occurrence of localized tenderness, jump sign, recognition and palpable band was much higher in LBP patients than in controls (significant, Chi-square test, $p < 0.05$). Limited stretch range (lateral flexion of the trunk in QLPS; hip adduction in GMPS) was found significantly more often in LBP patients for QLPS only (significant, Chi-square test, $p < 0.05$). The difference found for referred pain and twitch response was statistically not significant (Fisher's exact test $p \geq 0.05$).

Table 4.2 Occurrence of the diagnostic criteria for QLPS in LBP patients and controls.

First observer	LBP n=61	Controls n=63	level of significance
QLPS criteria			
localized tenderness	32	11	S[x]
referred pain	6	3	ns[x]
palpable band	18	6	S[x]
limited stretch range	12	1	S[x]
twitch response	2	0	ns*
jump sign	19	4	S[x]
recognition	13	0	S[x]

* Fisher's exact test $p < 0.05$ S = significant
[x] Chi-square test $p < 0.05$ ns= not significant

Table 4.3 Occurrence of the diagnostic criteria for GMPS in LBP patients and controls.

First observer	LBP n=61	Controls n=63	level of significance
GMPS criteria			
localized tenderness	29	9	S[x]
referred pain	8	3	ns[x]
palpable band	23	5	S[x]
limited stretch range	1	0	ns*
twitch response	2	0	ns*
jump sign	17	4	S[x]
recognition	13	2	S[x]

* Fisher's exact test $p < 0.05$ S = significant
[x] Chi-square test $p < 0.05$ ns= not significant

Tables 4.4 and 4.5 give the observations on the diagnostic criteria by the first and the second (blinded) observer for QLPS and GMPS, respectively.
Tables 4.6 and 4.7 present the interobserver agreement on the diagnostic criteria of QLPS and GMPS, respectively. Interobserver agreement values were calculated on the basis of the observations on the left and right side of the body. Thus, the number of observations for LBP patients was 122 (2 x 61) and for controls 126 (2 x 63). Interobserver agreement was calculated for the diagnostic criteria of both pain syndromes, and for QLPS and GMPS separately.

From Tables 4.4 and 4.5 it can be seen that the observations of the second blinded observer were not significantly different from those of the first observer, either for

LBP patients or for controls. Note that in QLPS, palpable band was also found in 8 (first observer) and 10 (second observer) controls without the presence of localized tenderness.

Table 4.4 Observations by the first (I) and second (II) observer on the diagnostic criteria for QLPS.

	LBP n=122		Controls n=126	
	I	II	I	II
QLPS criteria				
localized tenderness	52	50	18	15
referred pain	6	9	3	1
palpable band	25 (3)	24 (2)	8 (8)	5 (10)
limited stretch range	33	--	5	--
twitch response	4	4 (1)	0	1
jump sign	29	28 (1)	5	7
recognition	18	23 (2)	0	0

In parentheses: number of observations without the presence of localized tenderness.

Table 4.5 Observations by the first (I) and second (II) observer on the diagnostic criteria for GMPS.

	LBP n=122		Controls n=126	
	I	II	I	II
GMPS criteria				
localized tenderness	43	40	14	13
referred pain	11 (2)	16	3	3
palpable band	29 (4)	32 (3)	8	9 (4)
limited stretch range	3	--	0	--
twitch response	2	2	0	0
jump sign	22 (3)	25 (2)	6	5
recognition	17 (2)	20 (1)	2	0

In parentheses: number of observations without the presence of localized tenderness.

Results presented in Tables 4.6 and 4.7 show that the diagnostic criteria for both pain syndromes have a percentage agreement ranging between 81% and 94%. For the purpose of this study, a kappa of 0.5 was selected as a cut-off point of good interobserver agreement. In both pain syndromes kappa was above 0.5 for localized tenderness, jump sign and recognition. Palpable band had a kappa above 0.5 in GMPS only. Referred pain and twitch response had a kappa below 0.5 in both pain

syndromes.

According to the study protocol the second observer did not perform the tests for limited stretch range, because this required the patient standing up from the examination couch and moving around; thereby unblinding the situation. Therefore, the interobserver agreement of this criterium could not be determined.

Kappa can be adjusted for the prevalence and/or the bias effect by calculating kappa with the mean of the concordant or the discordant cells. The bias index (BI) is an index of the bias between examiners. For all diagnostic criteria the BI in both pain syndromes was very low, indicating that bias is not a problem.

The prevalence index (PI) is an index of differences between the overall proportion of "Yes" and "No" assessments. Prevalence index is high when prevalence is low, which causes unstable kappa values. Only localized tenderness in QLPS had a low prevalence index.

Prevalence adjusted kappa (PAK) allows better comparison of levels of interobserver agreement between different studies, regardless of the prevalence in a study population (Byrt 1993). According to the PAK calculated in the present study, if the problem of low prevalence is solved in future studies, kappa values are expected to improve.

Table 4.6 Interobserver agreement on the diagnostic criteria for QLPS in LBP patients.

QLPS criteria	%	Kappa (95% CI)	BI	PI	PAK (95% CI)
localized tenderness	87	0.73 (0.61 - 0.85) 0.36	0.02	0.16	0.73 (0.61 - 0.85) 0.85
referred pain	93	(0.04 - 0.68)	0.02	0.88	(0.76 - 0.94)
palpable band	82	0.47 (0.29 - 0.65)	0.02	0.56	0.63 (0.50 - 0.77)
limited stretch range	--	--	--	--	--
twitch response	94	0.19 (-0.18 - 0.56)	0.01	0.93	0.88 (0.80 - 0.96)
jump sign	89	0.68 (0.53 - 0.83)	0	0.52	0.77 (0.65 - 0.88)
recognition	88	0.57 (0.38 - 0.76)	0.06	0.65	0.75 (0.63 - 0.87)

Table 4.7 Interobserver agreement on the diagnostic criteria for GMPS in LBP patients.

GMPS criteria	%	Kappa (95% CI)	BI	PI	PAK (95% CI)
localized tenderness	81	0.58 (0.43 - 0.73)	0.02	0.32	0.62 (0.48 - 0.76)
referred pain	89	0.46 (0.22 - 0.70)	0.04	0.78	0.78 (0.67 - 0.89)
palpable band	80	0.51 (0.34 - 0.68)	0.02	0.44	0.60 (0.46 - 0.74)
limited stretch range	--	--	--	--	--
twitch response	97	-.02 (-.03 - 0.00)	0	0.96	0.93 (0.87 - 0.99)
jump sign	90	0.71 (0.55 - 0.85)	0.02	0.57	0.80 (0.69 - 0.90)
recognition	89	0.58 (0.39 - 0.77)	0.02	0.67	0.77 (0.65 - 0.88)

Table 4.8 shows the interobserver agreement on QLPS and GMPS. When applying Simons' 1990 major criteria of localized tenderness and referred pain, kappa remained below 0.5. Kappa was 0.36 for QLPS and 0.46 for GMPS, due to the considerable prevalence index of 0.87 and 0.77, respectively.
The set of criteria found eligible in this study, namely: localized tenderness, and jump sign or recognition, resulted in an interobserver agreement level above 0.5. Kappa for the presence of QLPS and GMPS was 0.66 and 0.62, respectively. The prevalence index for both pain syndromes was 0.43 and 0.51, respectively.

Table 4.8 Interobserver agreement on QLPS and GMPS.

	%	Kappa (95% CI)	BI	PI	PAK (95% CI)
Simons 1990 definition					
QLPS	92	0.36 (0.18 - 0.74)	0.02	0.87	0.85 (0.76 - 0.94)
GMPS	89	0.46 (-0.04 - 0.76)	0.04	0.77	0.78 (0.67 - 0.89)
This study definition					
QLPS	86	0.66 (0.51 - 0.81)	0.01	0.43	0.72 (0.59 - 0.84)
GMPS	86	0.62 (0.45 - 0.79)	0.02	0.51	0.72 (0.59 - 0.84)

% percentage agreement
CI confidence interval
BI Bias index
PI Prevalence index
PAK Prevalence adjusted kappa

Table 4.9 presents the occurrence of QLPS and GMPS in LBP patients in general practice. When applying Simons' 1990 major diagnostic criteria, the occurrence of QLPS in LBP patients was 10% (6 patients: mean age=39 SD 12 years; 1 female) and for GMPS 13% (8 patients: mean age=37 SD 12 years; 3 female). QLPS

occurred in 3 controls (5%) and GMPS occurred in another 3 controls (5%). The difference in occurrence between LBP patients and control subjects was not significant (Chi-square test, $p < 0.05$).

When applying the criteria found eligible in the present study, i.e. localized tenderness, and jump sign or recognition, the occurrence of QLPS and GMPS was 36% (22 patients: mean age=36 SD 10 years; 11 female) and 34% (21 patients: mean age= 35 SD 11 years; 11 female), respectively. The use of the definition of the present study leads to considerably more QLPS and GMPS in nonspecific LBP patients than in controls; 36% compared to 6% (significant; Chi-square test, $p < 0.05$).

No difference in gender, age, disturbed sleep and interference with routine daily activities was found between patients with and without QLPS or GMPS (significant; Chi-square test, $p < 0.05$).

Table 4.9 Occurrence of QLPS and GMPS in general practice (first observer).

MFPS	LBP n=61	Controls n=63	level of significance
Simons' 1990 definition			
QLPS	6 (10%)	3 (5%)	ns*
GMPS	8 (13%)	3 (5%)	ns*
This study definition			
QLPS	22 (36%)	4 (6%)	S*
GMPS	21 (34%)	4 (6%)	S*

* Fisher's exact test $p < 0.05$
* Chi-square test $p < 0.05$
S = significant
ns= not significant

Discussion

One of the aims of this study was to refine the diagnostic criteria for QLPS and GMPS by investigating the association between the diagnostic criteria for QLPS and GMPS, and LBP, and interobserver agreement on the diagnostic criteria. Symptoms are considered to be signs of a disease or of pathology if these symptoms are present in patients with the disease and absent in persons without the disease (Wulff 1980). The interobserver agreement on the physical signs is of great

importance when a syndrome has no gold standard. Diagnostic criteria for a clinical syndrome are preferably composed of symptoms with a good level of interobserver agreement (Sackett 1985).

In both pain syndromes, a statistically significant higher occurrence of localized tenderness, jump sign, recognition, and palpable band was found in LBP patients compared with controls (Tables 4.2, 4.3). The results of the present study on palpable band are in contrast to those of a preliminary study by Wolfe et al., who reported palpable taut bands to be equally present in MFPS patients (n=8) and in healthy controls (n=8) (Wolfe 1992). This discrepancy may be due to the fact that the muscles examined in Wolfe's study were localized mainly in the upper part of the body and were different from those studied in the present work. Palpable band also occurred without the presence of localized tenderness (Tables 4.4, 4.5), which raises some doubts about the specificity of this criterium.
The occurrence of referred pain was low both in QLPS and GMPS. This low occurrence is consistent with the observations of Wolfe and Sootsky (Sootsky 1989, Wolfe 1992). Sootsky also found that referred pain was more common in the upper than lower body (Sootsky 1989). The difference between occurrence of referred pain in LBP patients and controls was not significant.
In the present study, twitch response was seldom observed. This infrequent occurrence is not unexpected because different techniques to elicit this response have been described including snapping palpation (Travell 1983, Fricton 1985a), needling (Simons 1990), and pinching or pressing (McCain 1988). In this study simple palpation was applied.

In the present study a kappa of 0.5 was selected as a cut-off point of good interobserver agreement. As a result of this only localized tenderness, jump sign and recognition had good interobserver agreement for both pain syndromes. Kappa for palpable band was 0.47 for QLPS and 0.51 for GMPS. One problem with kappa is that it is sensitive to small occurrences (Feinstein 1990). Infrequent occurrence of twitch response and referred pain therefore resulted in kappas below 0.5.

An objection may be raised that for assessment of the interobserver agreement, the

observers are not equally informed about the patient's status. The first observer may have been biased by knowledge of the patient's LBP history or the patient's status, and may be inclined to find more characteristics present in the LBP patients. This is contradicted by the fact that no marked differences in the number of observations made by the two observers were found (Tables 4.4 and 4.5). Also, successive training sessions at the patient's follow-up provided a means to keep skills and the degree of agreement between observers at the same level.
Secondly, medical students may be considered to have moderate clinical experience and be novices in the diagnosis of regional pain syndromes. However, intertester agreement on physical examination does not appear to relate to years of experience (Potter 1985). Thus, the level of agreement in this study is probably the best that can be achieved.
Nice et al. conducted an interrater agreement study on regional pain syndromes in LBP patients (n=50). For the presence of the regional pain syndromes in the Iliocostalis Lumborum and the Longissimus Thoracis muscle, they used Simons' 1990 major criteria, i.e. localized tenderness and referred pain. Their level of interobserver agreement did not exceed a kappa of 0.4 (Nice 1992). This matches the kappa value found in the present study for Simons' 1990 definition (Table 4.8). The finding of Nice et al. may be due to the low kappa for referred pain. Unfortunately, they did not or could not differentiate between the two diagnostic criteria of localized tenderness and referred pain.

Use of Simons' criteria versus this study criteria
Based on the association between the diagnostic criteria for QLPS and GMPS, and LBP, and the interobserver agreement on the diagnostic criteria, the results of the present study suggest that localized tenderness, jump sign, and recognition are clinically useful for the presence of QLPS and GMPS. The latter two criteria may seem subjective, because both require the patient's reaction to the physician's examination. As a compromise between subjectivity and reliability of these criteria, only the presence of either jump sign or recognition will be incorporated in the definition of QLPS and GMPS. Referred pain, twitch response, and palpable band were not found to be eligible criteria.
Diagnostic criteria for QLPS and GMPS are described on the basis of observations

in clinical practice; the pathophysiology is unknown. Therefore, it is unknown which criteria are pathognomonic and which are accompanying symptoms. Until the aetiology for QLPS and GMPS is disclosed, in research and clinical settings, it would be appropriate to use the diagnostic criteria found eligible in the present study, i.e. localized tenderness, and jump sign or recognition.

When applying the diagnostic criteria found eligible in the present study, the occurrence of QLPS and GMPS in LBP patients was 36% and 34%, respectively. Bernard found a 2% prevalence of muscle syndromes in LBP patients attending an outpatient pain clinic (Bernard 1987). The discrepancy between his results and ours may be due to registration bias (Bernard's study was retrospective), the use of different diagnostic criteria and/or study population.
Another reason for this discrepancy in occurences may be the low admission rate of LBP patients in the present study. Based on the incidence of LBP without radiating pain of 26 per 1000 enlisted patients per year in general practice (Van der Velden 1991), 65 LBP patients could be expected per year in an average general practice of 2500 enlisted patients. In the present study, a total of 11 GPs participated. Thus, the expected number of LBP patients would have been 715 per year, or 179 in 3 months. The GPs recruited 81 LBP patients for the present study, which is 45% of the expected number.
The selection criteria of the present study can only be partially responsible for this low admission rate. Selection bias by the GPs may be involved. It is not expected, however, that the selection bias is directed towards the presence of QLPS or GMPS, since Dutch GPs are not accustomed to diagnose myofascial pain syndromes in LBP patients. It still remains possible that selection bias is directed towards a confounding and unidentified variable, causing an overestimation of the occurrence of QLPS and GMPS.
In addition to the low admission rate by the GPs, suitable LBP patients were not very motivated to participate, 16 of 81 LBP patients (20%) had second thoughts and eventually did not participate. Surprisingly, the same percentage, i.e. 17 of 82 (21%) of control subjects, had second thoughts on participation. It was expected that the number of non-participants would be higher in controls than in LBP patients.

Conclusion

Based on the association between the diagnostic criteria for QLPS and GMPS, and LBP, and the interobserver agreement on the diagnostic criteria, the results of the present study suggest that localized tenderness, and the presence of either jump sign or patient's recognition of his pain complaint, is a clinically useful definition for the presence of QLPS and GMPS. Of the major criteria proposed by Simons in 1990 only localized tenderness proved to be a clinically useful diagnostic criterium. Thus, until the aetiology for QLPS and GMPS is disclosed, it would be appropriate to use the diagnostic criteria found eligible in the present study.

The use of the criteria defined in the present study leads to markedly more QLPS and GMPS in nonspecific LBP patients than in controls. The occurrence in nonspecific LBP patients is 36% for QLPS and 34% for GMPS; the occurrence of both regional pain syndromes is 6% in the control subjects (significant, Chi-square test $p < 0.05$). Finally, the set of criteria found eligible in the present study resulted in good interobserver agreement, 0.66 for the QLPS and 0.62 for GMPS.

References

Bennett RM. Myofascial pain syndromes and the fibromyalgia syndrome: A comparative analysis. In: Fricton JR, Awad E ed. Advances in pain research and therapy volume 17. Raven Press, New York, 1990: 43-65.

Bernard TN, Kirkaldy-Willis WH. Recognizing specific characteristics of non specific low back pain. Clin Orthop 1987;217:266-80.

Byrt T, Bishop J, Carlin JB. Bias, prevalence and Kappa. J Clin Epidemiol 1993;46:423-9.

Cassisi JE, Sypert GW, Lagana L, Friedman EM, Robinson ME. Pain, disability, and psychological functioning in chronic low back pain subgroups: myofascial versus herniated disc syndrome. Neurosurgery 1993;33:379-85.

Feinstein AR, Cicchetti DV. High agreement but low kappa: I The problems of two paradoxes. J Clin Epidemiol 1990;43:543-49.

Fishbain DA, Goldberg M, Meafher BR, Steele R, Rosomoff H. Male and female chronic pain patients categorized by DSM-III psychiatric diagnostic criteria. Pain 1986;26:181-97.

Fricton JR, Auvinen MD, Dijkstra D, Schiffman E. Myofascial pain syndrome: Electromyographic changes associated with local twitch response. Arch Phys Med Rehabil 1985a;66:314-17.

Fricton JR, Kroening R, Haley D, Siegert R. Myofascial pain syndrome of the head and neck: a review of clinical characteristics of 164 patients. Oral Surg 1985b;60:615-23.

Fricton JR. Myofascial Pain Syndrome. Characteristics and epidemiology. In: Fricton JR, Awad E ed. Advances in pain research and therapy, vol. 17. New York: Raven Press,1990:107-25.

McCain GA, Scudds RA. The concept of primary fibromyalgia (fibrositis) clinical value, relation and significance to other chronic musculoskeletal pain syndromes. Pain 1988;33:273-87.

Nice DA, Riddle DL, Lamb RL, Mayhew TP, Rucker K. Intertester reliability of judgments of the presence of trigger points in patients with low back pain. Arch Phys Med Rehabil 1992;73:893-98.

Potter NA, Rothstein JM. Intertester reliability for selected clinical tests of the sacro iliac joint. Phys Ther 1985;65:1671-5.

Sackett DL, Haynes RB, Tugwell P. Clinical epidemiology. A basic science for clinical medicine. Little, Brown and Company, Boston/Toronto, 1985.

Simons DG, Travell JG. Myofascial origins of low back pain. 1. Principles of diagnosis and treatment. Postgrad Med 1983;73:66-77.

Simons DG. Muscular pain syndromes. In: Fricton JR, Awad E ed. Advances in pain research and therapy volume 17. Raven Press, New York,1990:1-41.

Sootsky SA, Jaeger B, Oye RK. Prevalence of myofascial pain in general internal medicine practice. West J Med 1989;151:157-60.

Travell JG, Simons DG. Myofascial pain and dysfunction. The trigger point manual. Vol. 1. The upper extremities. Baltimore, Williams and Wilkins, 1983.

Van der Velden J, De Bakker DH, Claessens AAMC, Schellevis FG. Een nationale studie naar ziekten en verrichtingen in de huisartspraktijk. Basisrapport: Morbiditeit in de huisartspraktijk. Utrecht, NIVEL 1991. [Morbidity in general practice. Dutch national survey of general practice. Utrecht: Netherlands Institute of Primary Health Care (NIVEL), 1992.]

Wolfe F, Simons DG, Fricton J, Bennett RM, Goldenberg DL, Gerwin R, et al. The fibromyalgia and myofascial pain syndromes. J Rheumatol 1992;19:944-51.

Wulff H. Principes van klinisch denken en handelen. Bohn, Scheltema en Holkema, Utrecht, 1980. [Rational diagnosis and treatment. Blackwell Scientific Publications, Oxford, 1976.]

Chapter 5

Results: Iliac Crest Pain Syndrome (ICPS)

This chapter presents the results on: 1) the association between the diagnostic criteria for ICPS and LBP; 2) the interobserver agreement; and 3) the occurrence of ICPS.

Introduction

ICPS is defined as a point of localized maximal tenderness found by digital palpation of the medial part of the iliac crest, near the attachment of the iliolumbar ligament. The pain is recognized by the patient as their own "typical" pain (Collee 1991c).

ICPS has been described in earlier reports under the names of the Iliolumbar Syndrome, the Iliolumbar Ligament Syndrome, or Maigne's Syndrome (Hirschberg 1979, Broudeur 1981, Naeim 1982, Bernard 1987). In the Netherlands, Collee et al. investigated this syndrome in an epidemiological study (Collee 1991a), and in a clinical trial using injection therapy (Collee 1991b). The prevalence of ICPS in LBP was reported to be 53% in general practice (n=40), 33% in an occupational health service (n=124), and 58% in a university rheumatology outpatient clinic (n=40) (Collee 1991a). In another study (n=170), however, general practitioners found a prevalence of 27% (Collee 1991c). The discrepancy in the prevalence in general practice could be attributed to selection bias or poor interobserver agreement on the presence of ICPS.

A few reports describe good results from local injections with steroids (Sonne 1985), NSAID (Broudeur 1981), or with a local anaesthetic (Fairbank 1983). In a double-blind, randomized trial a beneficial effect was demonstrated on the pain score and pain severity of a local lignocaine injection compared to isotonic saline. The effect was evident in LBP patients in the rheumatology clinic (total n=24), but not in general practice (total n=17) (Collee 1991b).

The questions addressed in this chapter are:
1) Which diagnostic criteria of ICPS are present in LBP patients compared with control subjects?
2) What is the level of interobserver agreement on the diagnostic criteria of the syndrome? What is the level of interobserver agreement on ICPS?
3) What is the occurrence of ICPS in LBP in general practice?

Material and methods

See Chapter 3.

Results

A total of 163 participants (81 LBP patients) were invited to participate in the study. After exclusion of 5 participants (4 LBP patients) who did not meet the study criteria, 158 participants were eligible. Thirty-three participants (16 LBP patients) did not make an appointment or did not keep the appointment, and one subject (control) withdrew cooperation. Finally, a total of 124 participants entered the study: 61 nonspecific LBP patients and 63 controls. There was no significant difference between patients and controls with regard to age, gender or employment status (Chapter 4; Table 4.1).

Table 5.1 shows the occurrence of the diagnostic criteria for ICPS in LBP patients compared to the occurrence in control subjects. Localized tenderness and recognition occurred more often in LBP patients than in controls (significant; Chi-square test, $p < 0.05$).

Table 5.1 Diagnostic criteria for ICPS in LBP patients and controls (first observer).

ICPS diagnostic criteria	LBP n=61	Controls n=63	level of significance
localized tenderness	25	3	S[x]
recognition, i.e.typical pain	13	0	S[x]

S significant
[x] Chi-square test $p < 0.05$

Table 5.2 shows the observations made by the first observer compared to the observations by the second (blinded) observer. The differences between the observers were not significant (Chi-square test, $p < 0.05$). This is also illustrated by a bias index of almost 0 (Table 5.3).

Table 5.2 Number of observations made by the first (I) and second (II) observer on the diagnostic criteria for ICPS.

ICPS diagnostic criteria	LBP n=122		Controls n=126	
	I	II	I	II
localized tenderness	36	36	3	4
recognition	17 (5)	18 (6)	0	1

In parentheses: number of observations without localized tenderness.

Interobserver agreement values were calculated on the basis of the observations on the left and right side of the body. Thus, the number of observations was 122 (2 x 61) for LBP patients and 126 (2 x 63) for controls. A kappa value of 0.5 was selected as a cut-off point of good interobserver agreement. Interobserver agreement was calculated for the diagnostic criteria of ICPS as well as for ICPS itself. Table 5.3 presents the level of interobserver agreement on the diagnostic criteria of ICPS. For the ICPS criteria localized tenderness and recognition, kappa was 0.57 (95% CI: 0.40 - 0.73) and 0.66 (95% CI: 0.48 - 0.83), respectively (Table 5.3).

Table 5.3 Interobserver agreement on the diagnostic criteria for ICPS in LBP patients.

ICPS diagnostic criteria	%	Kappa (95% CI)	BI	PI	PAK (95% CI)
localized tenderness	82	0.57 (0.40 - 0.73)	0	0.41	0.64 (0.50 - 0.78)
recognition	89	0.66 (0.48 - 0.83)	0.02	0.61	0.79 (0.68 - 0.89)

% percentage agreement
CI confidence interval
BI Bias index
PI Prevalence index
PAK Prevalence adjusted kappa

Table 5.4 summarizes the interobserver agreement on ICPS in LBP patients. The percentage of agreement for ICPS was 89%. Kappa for ICPS was 0.57 (95% CI: 0.34 - 0.79). Since prevalence bias (0.71) was considerable PAK is 0.79 (95% CI: 0.68 - 0.89).

Table 5.4 Interobserver agreement on ICPS in LBP patients (n=122).

	%	Kappa (95% CI)	BI	PI	PAK (95% CI)
ICPS	89	0.57 (0.34 - 0.79)	0.01	0.71	0.79 (0.68 - 0.89)

% percentage agreement
CI confidence interval
BI Bias index
PI Prevalence index
PAK Prevalence adjusted kappa

Table 5.5 shows the occurrence of ICPS in LBP patients in general practice. ICPS was found in 13 (21%) LBP patients and in none of the controls (significant; Chi-square test; $p < 0.05$). In 9 patients ICPS was on one side only. It was found to be equally prevalent in male (n=7) and female (n=6) patients. The mean age of this group of ICPS patients was 34 (SD 10) years.
Differences between LBP patients with and without ICPS for age, gender, disturbed sleep and interference with routine daily activities, were not significant (Chi-square test; $p < 0.05$).

Table 5.5 Occurrence of ICPS in LBP patients in general practice.

	LBP n=61	Controls n=63	level of significance
ICPS	13 (21%)	0	S[x]

S significant
[x] Chi-square test, $p < 0.05$

Discussion

A significant higher occurrence of both diagnostic criteria for ICPS, i.e. localized tenderness and recognition, was found in LBP patients than in controls (Table 5.1). Recognition proved to be highly specific, as none of the controls presented this symptom.

Furthermore, the level of interobserver agreement for localized tenderness had a kappa of 0.57 (Table 5.3). In Collee's definition, the site is clearly defined as the medial part of the dorsal iliac crest near the iliolumbar ligament. In McCombe's study on the reliability of physical signs in LBP, kappa for tenderness anywhere at the dorsal iliac crest is 0.50 (McCombe 1989). Perhaps, to improve the level of kappa the description of localized tenderness should be more specific, for example: tenderness in an area of 2 cm at the projection of the iliolumbar ligament on the iliac crest.
Based on the association between the diagnostic criteria of ICPS and LBP, and the interobserver agreement of the diagnostic criteria, it is concluded that Collee's diagnostic criteria, i.e. localized tenderness and recognition, are clinically useful.

The present study found an occurrence of 21% ICPS in LBP patients in general practice. This is in agreement with another report (n=170) from Dutch general practitioners who have reported a prevalence of 27% (Collee 1991c). The low admission rate of 45% (Chapter 4, discussion) of LBP patients in the present study is not expected to be selective towards the presence of ICPS in LBP patients.
A university rheumatology clinic (n=40) reported a prevalence of 58%. In the rheumatology clinic study, the mean duration of the LBP episode was 52 weeks (Collee 1991a) compared to only 11 days for the patients in the present study (Chapter 3, Table 3.1). This suggests that ICPS may be more prevalent in patients with a history of LBP of longer duration.

Conclusion

Based on the association between the diagnostic criteria of ICPS and LBP, and interobserver agreement on the diagnostic criteria, the results of the present study indicate that localized tenderness and recognition are clinically useful diagnostic criteria for the presence of ICPS. The presence of ICPS could be judged with good interobserver agreement; kappa was 0.57. The occurrence of ICPS in LBP is 21% in general practice. ICPS was equally prevalent in male and female patients and the mean age of these ICPS patients was 34 years.

References

Bernard TN, Kirkaldy-Willis WH. Recognizing specific characteristics of non specific low back pain. Clin Orthop 1987;217:266-80.

Broudeur P, Larroque CH, Passeron R, Pellegrino J. Les aspects nouveaux du syndrome ilio-lombaire (SIL). Lyon Mediterraneé Medicine du Sud-Est 1981;17:4708-33.

Collee G, Dijkmans BAC, Vandenbroucke JP, Cats A. Iliac Crest Pain Syndrome in low back pain: Frequency and features. J Rheumatol 1991a;18:1064-7.

Collee G, Dijkmans BAC, Vandenbroucke JP, Cats A. Iliac Crest Pain Syndrome in low back pain: A double blind, randomized study of local injection therapy. J Rheumatol 1991b;18:1060-4.

Collee G. Clinical syndromes in low back pain. [Thesis] University of Leiden, Leiden 1991c.

Fairbank JCT, O'Brien JP. The iliac crest syndrome. A treatable cause of low back pain. Spine 1983;8:220-4.

Hirschberg CG, Froetscher L, Naeim F. Iliolumbar syndrome as a common cause of LBP: diagnosis and prognosis. Arch Phys Med Rehabil 1979;60:415-9.

McCombe PF, Fairbank JC, Cochersole BC, et al. 1989 Volvo Award in clinical sciences. Reproducibility of physical signs in low back pain. Spine 1989;14:908-18.

Naeim F, Froetscher L, Hirschberg GG. Treatment of the chronic iliolumbar syndrome by infiltration of the iliolumbar ligament. West J Med 1982;136:372-4.

Sonne M, Christensen K, Hansen SE, Jensen EM. Injection of steroids and local anaesthetics as therapy for low back pain. Scan J Rheumatol 1985;14:343-5.

Chapter 6

Results: Long Ligament Pain Syndrome (LLPS)

This chapter presents the results on: 1) the association between the diagnostic criteria for LLPS and LBP; 2) the interobserver agreement; and 3) the occurrence of LLPS in LBP in general practice.

Introduction

LLPS is diagnosed when the patient complains of pain in the low back region and maximal tenderness is found on palpation of the long dorsal sacroiliac ligament (long ligament). The long ligament can be palpated directly underneath the skin caudal to the posterior superior iliac spine. It is covered by the fascia of the gluteus maximus muscle and feels solid on palpation, giving the impression of a bony structure. Fibres of the long dorsal sacroiliac ligament connect the posterior superior iliac spine and a small part of the iliac crest with the lateral crest of the third and fourth segments of the sacrum (Vleeming 1996).

There are few reports on LLPS. From a survey on 394 women with peripartum pelvic pain, 42% of the women reported pain in the area of the long ligament (Mens 1992). LLPS has been described under different names with slightly different diagnostic criteria, i.e. Sacroiliac Pain Syndrome, Sacroiliac Joint Syndrome, Sacroiliac Dysfunction Syndrome, or Sacroiliac Sprain Syndrome (Bernard 1987, Broadhurst 1989, Leblanc 1992). In a retrospective study on 1293 LBP patients referred to a pain centre, Sacroiliac Joint Syndrome was found in 23% (Bernard 1987). Although the Sacroiliac Joint Syndrome included more diagnostic signs than LLPS, tenderness over the posterior superior iliac spine was one of them (Bernard 1987). Personal reports from Dutch GPs indicate that LLPS is "common knowledge".

The questions addressed in this chapter are:

1) Which diagnostic criteria for LLPS are present in LBP patients compared with control subjects?

2) What is the level of interobserver agreement on the diagnostic criteria for LLPS, and on LLPS itself?

3) What is the occurrence of LLPS in LBP in general practice?

Material and methods

See Chapter 3.

Results

A total of 124 participants entered the study: 61 nonspecific LBP patients and 63 controls. There was no significant difference between patients and controls with regard to age, gender or employment status (Chapter 4; Table 4.1).

Table 6.1 presents the occurrence of the diagnostic criteria for LLPS in LBP patients compared with the occurrence in controls. Localized tenderness was found in 28 LBP patients (46%) and in 12 controls (19%). Recognition was found in 14 LBP patients (23%) and in only 1 control (2%). For both diagnostic criteria the difference between LBP patients and controls was statistically significant (Chi-square test $p < 0.05$).

Table 6.1 Occurrence of the diagnostic criteria for LLPS (first observer).

LLPS diagnostic criteria	LBP n=61	Controls n=63	level of significance
localized tenderness	28 (46%)	12 (19%)	S[x]
recognition	14 (23%)	1 (2%)	S[x]

S significant

[x] Chi-square test $p < 0.05$

Table 6.2 shows the frequency of observations by the first and second observer. There was no significant difference between observations of the two observers; consequently, the bias index was 0.03 and 0.01 for the two criteria, respectively (Table 6.3).

Table 6.2 Observations by the first (I) and second (II) observer on the diagnostic criteria for LLPS.

LLPS diagnostic criteria	LBP n=122		Controls n=126	
	I	II	I	II
localized tenderness	44	48	17	13
recognition	19 (2)	18 (4)	1	0 (2)

In parentheses: number of observations without localized tenderness.

Interobserver agreement values were calculated on the basis of the observations on the left and right side of the body. Thus, the number of observations was 122 (2 x 61) for LBP patients and 126 (2 x 63) for controls. A kappa value of 0.5 was selected as a cut-off point of good interobserver agreement. Interobserver agreement was calculated for the diagnostic criteria of LLPS as well as for LLPS itself. Table 6.3 summarizes the interobserver agreement on the diagnostic criteria for LLPS.

Both symptoms had a kappa value above 0.5; for localized tenderness kappa was 0.76 (95% CI: 0.64 - 0.88) and for recognition 0.69 (95% CI: 0.51 - 0.87). Kappa is very sensitive to small prevalence (Feinstein 1990). This has repercussions on the level of the prevalence index, which was much higher for recognition (0.65) than for localized tenderness (0.25). Therefore, prevalence adjusted kappa (PAK) for localized tenderness was almost the same as simple kappa, i.e. 0.77 (95% CI: 0.66 - 0.88), but PAK for recognition was 0.82 (95% CI: 0.72 - 0.92).

Table 6.3 Interobserver agreement on the diagnostic criteria for LLPS.

LLPS diagnostic criteria	%	Kappa (95% CI)	BI	PI	PAK (95% CI)
localized tenderness	89	0.76 (0.64 - 0.88)	0.03	0.25	0.77 (0.66 - 0.88)
recognition	91	0.69 (0.51 - 0.87)	0.01	0.65	0.82 (0.72 - 0.92)

Table 6.4 summarizes the interobserver agreement on LLPS. The presence of the LLPS could be found with good interobserver agreement. Kappa was 0.65 (95% CI: 0.45 - 0.85). Prevalence index was high resulting in PAK of 0.90 (95% CI: 0.85 - 0.96).

Table 6.4 Interobserver agreement on LLPS.

	%	Kappa (95% CI)	BI	PI	PAK (95% CI)
LLPS	91	0.65 (0.45 - 0.85)	0.01	0.69	0.90 (0.85 - 0.96)

Table 6.5 presents the occurrence of LLPS in LBP patients. LLPS was found in 13 (21%) LBP patients and in 1 control (2%) (significant; Chi-square test, $p < 0.05$). LLPS was almost equally distributed between men (n=6) and women (n=7). The mean age of LLPS patients was 37 (SD 10) years. Differences between LBP patients with and without LLPS for age, gender, disturbed sleep and interference with routine daily activities, were not significant (Chi-square test, $p < 0.05$).

Table 6.5 Occurrence of LLPS in LBP patients in general practice.

	LBP n=61	Controls n=63	level of significance
LLPS	13 (21%)	1 (2%)	S[x]

S significant
[x] Chi-square test $p < 0.05$

Discussion

Both diagnostic criteria, localized tenderness and recognition, occurred more often in LBP patients than in controls. The difference was statistically significant. The interpretation of this finding is that there is an association between LLPS symptoms and LBP. However, 12 controls (19%) were observed to experience localized tenderness when palpated at the long ligament. This indicates that the ligament could be under strain in persons without LBP complaints. The stability of the sacroiliac joint is basically dependent on the ligaments that surround the joint. The sacrotuberous ligament (Vleeming 1989) and the long dorsal sacroiliac ligament (Vleeming 1996) play an important role in this respect. In a postmortem study (n=6), it was found that displacement of the sacroiliac joint altered the tension in the long ligament (Vleeming 1996). Consequently, this diagnostic criterium alone cannot be considered specific for LLPS; only in combination with recognition does it gain specificity. Recognition was found in 1 control only. Because no control subject could have LBP at the moment of investigation implies that pain was provoked in this subject, that was recognized as pain experienced previously. The combination of both diagnostic criteria for LLPS, localized tenderness and recognition, is clinically useful.

Interobserver agreement studies on physical signs in LBP provide an estimate as to what is a reasonable cut-off level. The general level of interobserver agreement on signs in LBP is between 0.4 (McCombe 1989) and 0.6 (Waddell 1982). For this reason a cut-off level at a kappa of 0.5 can be considered as realistic for clinical practice. In the present study, localized tenderness had a kappa of 0.76 (95% CI: 0.64 - 0.88) and recognition 0.69 (95% CI: 0.51 - 0.87). Therefore, the presence of the diagnostic criteria can be observed at a good level of interobserver agreement, i.e. kappa is above 0.5.

The occurrence of LLPS in LBP patients in general practice was 21% and only 1 control had LLPS. This occurrence of LLPS is in agreement with the prevalence (26%) found in Bernard's study on the Sacroiliac Joint Syndrome, which also includes tenderness over the posterior superior iliac spine (Bernard 1987).
In a Dutch survey, 42% of 394 women with peripartum pelvic pain reported pain

in the area of the long ligament (Mens 1992). Because the survey was held in a highly selected group of women, all members of the Dutch society for patients with pelvic complaints related to symphiolysis, it remains inconclusive whether women after childbirth are at risk for developing LLPS.

Conclusion

LLPS was related to the presence of LBP and was rarely found in control subjects. The presence of LLPS could be established at a good level of interobserver agreement; kappa was 0.65. LLPS occurred in 21% of the LBP patients in general practice; it was equally distributed between male and female LBP patients and the mean age of the LLPS patients was 37 years.

References

Bernard TN, Kirkaldy-Willis WH. Recognizing specific characteristics of nonspecific low back pain. Clin Orthop 1987;217:266-80.

Broadhurst NA. Sacroiliac dysfunction as cause of low back pain. Aust Fam Physician 1989;18:623-8.

Feinstein AR, Cicchetti DV. High agreement but low kappa. I. The problems of two paradoxes. J Clin Epidemiol 1990;43:543-9.

Leblanc KE. Sacroiliac sprain: an overlooked cause of back pain. Am Fam Phys 1992;46:1459-63.

McCombe PF, Fairbank JCT, Cockersole BC, Pynsent PB. Reproducibility of physical signs in low back pain. Spine 1989:14:908-18.

Mens JAM, Stam HJ, Stoeckart R, Vleeming A, Snijders CJ. Peripartum pelvic pain; a report of analysis of an inquiry among patients of a Dutch patient society. Proceedings of the First International Congress on Low Back Pain and the SI joint. San Diego, 1992.

Vleeming A. The sacrotuberous ligament: a conceptual approach to its dynamic role in stabilizing the sacroiliac joint. Clin Biomech 1989;4:201-3.

Vleeming A, Pool-Goudzwaard AL, Hammudoghlu D, Stoeckart R, Snijders CJ, Mens JMA. The function of the long dorsal sacroiliac ligament; its implication for understanding low back pain. Spine 1996 (in press).

Waddell G, Main CJ, Morris EW, Venner RM, Rae PS, Sharmy SH, et al. Normality and reliability in the clinical assessment of backache. BMJ 1982;284:1519-23.

Chapter 7

Results: Prognosis

Introduction

In general practice, the clinical course of LBP is usually self-limiting and most episodes (80%) resolve in less than 4 weeks (Lamberts 1991). Recurrent episodes, however, occur frequently (Biering-Sörensen 1983). Of all LBP patients in general practice, 6% (Lamberts 1991) to 9% (Chavannes 1992) become chronic LBP patients, i.e. LBP persisting longer than 3 months (Frymore 1988).
Furthermore, serious diseases, which could account for the previous nonspecific LBP episodes, are estimated to occur in less than 1% of the cases in general practice (Barker 1977). In a 4 year follow-up study in one general practice, 9 (2%) of 380 LBP patients developed a disc prolapse (Hoekstra 1982).
Unfortunately, the outcome for an individual nonspecific LBP patient cannot be predicted. Chronic LBP can be diagnosed in patients both with and without a specific cause of LBP. Ideally, patients at risk for chronic outcome should be identified early in the course of an episode of LBP as a means of preventing disability and avoiding the high costs associated with chronic LBP (Chavannes 1992, Faas 1996).

The prognosis of regional pain syndromes has not been investigated. No follow-up studies have investigated the clinical course and outcome of the regional pain syndromes.
A subdivision of nonspecific LBP into regional pain syndromes is only relevant and useful if regional pain syndromes prove to have a prognosis different from that of nonspecific LBP. If this is the case, then it would imply that regional pain syndromes form separate categories within the heterogeneous group of nonspecific LBP. Knowledge on the prognosis of regional pain syndromes is important, because it helps to decide which course research and clinical practice should take.

The research questions addressed in this chapter are:
1. What is the level of persistence of pain and reproducibility of the regional pain

syndromes in LBP patients with and without regional pain syndromes after 2 and 4 weeks?
2. How many LBP recurrences and diagnoses, which could be related to the LBP episodes, occur in LBP patients with and without regional pain syndromes 5 years after the initial examination?

Material and method

Prognosis was evaluated by the results of follow-up at 4 weeks and at 5 years. Outcome measures for the follow-up at 4 weeks included: LBP reported by the patient and associated reproducibility of the regional pain syndrome.
The outcome measures for the follow-up at 5 years included: the number of recurrent LBP episodes, the duration of recurrent LBP episodes and diagnoses which could account for the nonspecific LBP complaints. All outcome measures were calculated for patients with a recurrent LBP episode only. The mean number of patient-practice contacts was evaluated to assess which of the patients had a high medical consumer behaviour.
Where appropriate, Chi-square test or Student's t-test were applied. A p-value less than 0.05 was considered statistically significant.
In this chapter, QLPS and GMPS are defined by the diagnostic criteria found to be eligible in the present study, i.e. localized tenderness, and jump sign or recognition (Chapter 4).

Follow-up at 2 and 4 weeks

The moment of the initial examination is designated t=0. All LBP patients (n=61) were asked to return after 2 (t=2) and 4 weeks (t=4) to answer a questionnaire and undergo a physical examination. After 2 weeks, 14 (23%) and after 4 weeks an additional 13 (21%) LBP patients were lost to follow-up, leaving only 34 LBP patients (56%) with a complete follow-up at 4 weeks. Patients were called by telephone when they did not show up at the follow-up appointment. Some had already returned to work and were not willing to take time off. Others refused to cooperate further, because they were too busy or the pain had subsided.
In this chapter, only the results of the 4 week follow-up are presented; the follow-up at 2 weeks reveals no valuable information.

Follow-up at 5 years

Five years after the initial examination the medical records of the LBP patients (n=61) and control subjects (n=63), participating in this study were traced. The following topics were registered from the patient's record: number and duration of recurrent LBP episodes, management of the GP concerning each LBP episode, diagnoses related to LBP, and the number of all patient-practice contacts over the previous 5 years. The number of patient-practice contacts was evaluated to assess which of the patients had a high medical consumer behaviour.

For interpretation of the contents of the medical records, the observer did not know the person's status, i.e. LBP patient or control subject, nor whether a regional pain syndrome had been present at the examination 5 years previously.

Results

Medical records of 96 subjects (77%) could be found immediately. The remaining 28 patients had moved to a different GP or to another town. Additionally, 14 of the 28 medical records could be studied by contacting the new GP or the patient at their new address. More subjects (n=5) were traced by this procedure, but 3 refused to cooperate and in 2 cases most of the content of the medical record was lost due to subsequent moves. Finally, 110 (89%) complete medical records could be retrieved; 53 (87%) for LBP patients and 57 (90%) for control subjects (Table 7.1).

Table 7.1 Number of patients with complete follow-up.

Follow-up period	LBP patients	Controls
	n=61	n=63
2 weeks	47 (77%)	-
4 weeks	34 (56%)	-
5 years	53 (87%)	57 (90%)

Tables 7.2 and 7.3 show the results on reported pain and reproducibility of QLPS and GMPS, respectively. Patients with QLPS and GMPS at the initial examination (t=0) were designated QLPS+ and GMPS+, respectively; patients without QLPS and GMPS at the initial examination were designated QLPS- and GMPS-, respectively. After 4 weeks (t=4), 8 (37%) QLPS+ and 19 (49%) QLPS- patients, and 11 (52%) GMPS+ and 16 (40%) GMPS- patients were lost to follow-up.
At 4 weeks, 8 (57%) of 14 QLPS+ patients, and 7 (35%) of 20 QLPS- patients reported pain; 7 (70%) of 10 GMPS+ patients and 8 (33%) of 24 GMPS- patients reported pain.
At 4 weeks, QLPS remained present in 5 (36%) of 14 QLPS+ patients and did not occur in QLPS- patients. GMPS persisted in 4 (40%) QLPS+ patients. In 4 patients who initially were negative for GMPS, the syndrome was present at 4 weeks.

Table 7.2 Reported pain and reproducibility of QLPS at 4 week follow-up (n=61).

Number of patients (t=0)	QLPS+ 22		QLPS- 39	
Number of patients (t=4)	14 (63%)		20 (51%)	
Reported pain	pain	no pain	pain	no pain
	8	6	7	13
- patients with QLPS	3	2	0	0

Reported pain at t=4 for QLPS+ versus QLPS- was not significant (Chi-square test, $p < 0.05$)

QLPS	Quadratus Lumborum Pain Syndrome
QLPS+	QLPS present at initial examination (t=0)
QLPS-	QLPS not present at initial examination (t=0)
t=0	at initial examination
t=4	at 4 week follow-up

Table 7.3 Reported pain and reproducibility of GMPS at 4 week follow-up (n=61).

Number of patients (t=0)	GMPS+ 21		GMPS- 40	
Number of patients (t=4)	10 (48%)		24 (60%)	
Reported pain	pain 7	no pain 3	pain 8	no pain 16
- patients with GMPS	3	1	2	2

Reported pain at t=4 for GMPS+ versus GMPS- was not significant (Chi-square test, $p < 0.05$)

GMPS	Gluteus Medius Pain Syndrome
GMPS+	GMPS present at initial examination (t=0)
GMPS-	GMPS not present at initial examination (t=0)
t=0	at initial examination
t=4	at 4 week follow-up

Table 7.4 shows the results on reported pain and reproducibility of ICPS. Patients with ICPS at the initial examination (t=0) were designated ICPS+; patients without ICPS at the initial examination were designated ICPS-. At 4 weeks (t=4), 5 (38%) ICPS+ and 22 (46%) ICPS- patients were lost to follow-up.
The pain was still present in 5 (63%) of 8 ICPS+ patients compared to 10 (38%) of 26 ICPS- patients. After 4 weeks, ICPS was reproducible in 4 (50%) of 8 ICPS+ patients and started to appear in 6 (21%) of 26 patients, who were free of ICPS at t=0.

Table 7.4 Reported pain and reproducibility of ICPS at 4 week follow-up (n=61).

Number of patients (t=0)	ICPS+ 13		ICPS- 48	
Number of patients (t=4)	8 (62%)		26 (54%)	
Reported pain	pain 5	no pain 3	pain 10	no pain 16
- patients with ICPS	3	1	5	1

Reported pain at t=4 for ICPS+ versus ICPS- was not significant (Chi-square test, $p < 0.05$)

ICPS	Iliac Crest Pain Syndrome
ICPS+	ICPS present at initial examination (t=0)
ICPS-	ICPS not present at initial examination (t=0)
t=0	at initial examination
t=4	at 4 week follow-up

Table 7.5 presents the results on reported pain and reproducibility of LLPS. Patients with LLPS at the initial examination (t=0) were designated LLPS+; patients without LLPS at the initial examination were designated LLPS-. After 4 weeks (t=4), 7 (54%) LLPS+ and 20 (42%) LLPS- patients were lost to follow-up.

Three of 6 LLPS+ patients still reported pain, compared to 12 (43%) of 28 patients without LLPS at t=0.

LLPS remained present only in 2 patients, and started to occur in 3 patients who were free of LLPS at the initial examination.

Table 7.5 Reported pain and reproducibility of LLPS at 4 week follow-up (n=61).

Number of patients (t=0)	LLPS+ 13		LLPS- 48	
Number of patients (t=4)	6 (46%)		28 (58%)	
Reported pain	pain	no pain	pain	no pain
	3	3	12	16
- patients with LLPS	1	1	3	0

Reported pain at t=4 for LLPS+ versus LLPS- was not significant (Chi-square test, $p < 0.05$)

LLPS	Long Ligament Pain Syndrome
LLPS+	LLPS present at initial examination (t=0)
LLPS-	LLPS not present at initial examination (t=0)
t=0	at initial examination
t=4	at 4 week follow-up

Follow-up at 5 years

The results presented in Table 7.6 concern only those patients who visited their GP with one or more recurrent LBP episodes in the 5 year follow-up period; 49 (79%) LBP patients and 22 (39%) controls had consulted the GP for a recurrent LBP episode (significant, Chi-square test, $p < 0.05$).

A total of 48 LBP patients were reported to have 1 to 5 recurrent LBP episodes in 5 years; of these patients 31 had 1, 10 patients had 2, and 7 had 3 recurrent LBP episodes. One LBP patient with more than 5 recurrent LBP episodes actually had 6 recurrent LBP episodes in 5 years. A total of 22 control subjects were reported to have 1 to 5 recurrent LBP episodes in 5 years.

Four LBP patients (8%) had a recurrent LBP episode of subacute duration (4

weeks to 3 months), of whom 2 patients had 1 subacute episode and 2 patients had 2 subacute episodes in 5 years. Another 4 LBP patients (8%) had 1 recurrent LBP episode, which had lasted longer than 3 months, i.e. chronic LBP.
In the LBP group, the diagnoses which could be attributed to LBP included: lumbar disc disease (n=1), arthrosis (n=2) and scoliosis (n=1); in the control group: pseudoradicular syndrome (n=1), disc degeneration (n=1), scoliosis (n=2), and leg length difference > 2 cm (n=2).
The mean number of patient-practice contacts per year for LBP patients was 4.8.

Table 7.6 Recurrent LBP episodes and LBP related diagnoses in LBP patients based on data from the medical records at 5 year follow-up.

At 5 yr follow-up		LBP n=53
Patients with recurrent LBP episode after t=0		49 (79%)
1 - 5 recurrent LBP episodes/5yr		48
> 5 recurrent LBP episodes/5yr		1
acute episode	(< 4 weeks)	41
subacute episode	(4 weeks - 3 months)	4
chronic episode	(> 3 months)	4
LBP related diagnoses		4 (10%)
GP management of LBP /5 yr		
Number of patients with:		
Medication		31 (78%)
Physiotherapy		15 (38%)
X-ray		9 (23%)
Referral to medical specialist		1 (3%)
LBP contacts (mean/5 yr)		2.5
LBP episodes (mean/5 yr)		1.6
LBP contacts/episode		1.5
Patient-practice contacts (mean/yr)		4.8

Table 7.7 shows the results of the follow-up at 5 years of QLPS+ and QLPS- patients. There were no statistically significant differences between the two groups for the number and duration of the recurrent LBP episodes.
The mean number of LBP episodes after t=0 was 2.0 for QLPS+ patients, and

1.4 for QLPS- patients (significant, Student's t-test; $p < 0.05$). The mean number of patient-practice contacts per year for QLPS+ patients was 6.5, and 2.9 for QLPS- patients (significant, Student's t-test; $p < 0.05$).

Table 7.7 LBP recurrences and LBP related diagnoses in QLPS+ and QLPS- patients at 5 year follow-up.

	t=0	QLPS+ n=22	QLPS- n=39	
Patients with a complete 5 yr follow-up		15	38	
Patients with recurrent LBP episode after t=0		15 (68%)	34 (87%)	ns
1-5 recurrent LBP episodes		14	34	ns
> 5 recurrent LBP episodes		1	0	
acute episode	(< 4 weeks)	12	29	ns
subacute episode	(4 weeks - 3 months)	3	1	
chronic episode	(> 3 months)	0	4	
LBP related diagnoses		0	4	
LBP contacts (mean/5yr)		3.0	2.1	ns
LBP episodes (mean/5yr)		2.0	1.4	S*
LBP contacts/episode		1.4	1.5	
Patient-practice contacts (mean/yr)		6.5	2.9	S*

QLPS+ Quadratus Lumborum Pain Syndrome present at initial examination (t=0)
QLPS- Quadratus Lumborum Pain Syndrome not present at initial examination (t=0)
ns not significant
S* significant (Student's t-test; $p < 0.05$)

Table 7.8 presents the results of the follow-up at 5 years of GMPS+ and GMPS- patients. There were no statistically significant differences between the two groups for the number and duration of the recurrent LBP episodes.
The relevant diagnosis in the LBP patient who initially was positive for GMPS, was arthrosis. The diagnoses in GMPS- patients were: lumbar disc disease, arthrosis and scoliosis.
The mean number of patient-practice contacts was higher for the GMPS+ patients than GMPS- patients (5.6 versus 3.5; not significant, Student's t-test).

Table 7.8 LBP recurrences and LBP related diagnoses in GMPS+ and GMPS- patients at 5 year follow-up.

	t=0	GMPS+ n=21	GMPS- n=40	
Patients with a complete 5 yr follow-up		14	39	
Patients with recurrent LBP episode after t=0		12 (57%)	37 (93%)	ns
1-5 recurrent LBP episodes		12	36	ns
> 5 recurrent LBP episodes		0	1	
acute episode	(< 4 weeks)	10	31	ns
subacute episode	(4 weeks - 3 months)	1	3	
chronic episode	(> 3 months)	1	3	
LBP related diagnoses		1	3	ns
LBP contacts (mean/5yr)		2.5	2.4	ns
LBP episodes (mean/5yr)		1.6	1.6	
LBP contacts/episode		1.6	1.4	ns
Patient-practice contacts (mean/yr)		5.6	3.5	ns

GMPS+ Gluteus Medius Pain Syndrome present at initial examination (t=0)
GMPS- Gluteus Medius Pain Syndrome not present at initial examination (t=0)
ns not significant

Table 7.9 shows the results of the 5 year follow-up for the ICPS+ and ICPS- patients. There were no statistically significant differences between the two groups for the number and duration of the recurrent LBP episodes.
The LBP related diagnoses in the ICPS- patients were: lumbar disc disease (n=1), arthrosis (n=2), and scoliosis (n=1).

Table 7.9 LBP recurrences and LBP related diagnoses in ICPS+ and ICPS- patients at 5 year follow-up.

	t=0	ICPS+ n=13	ICPS- n=48	
Patients with a complete 5 yr follow-up		8	45	
Patients with recurrent LBP episode after t=0		7 (54%)	42 (88%)	ns
1-5 recurrent LBP episodes		7	41	ns
> 5 recurrent LBP episodes		0	1	
acute episode	(< 4 weeks)	6	39	ns
subacute episode	(4 weeks - 3 months)	1	3	
chronic episode	(> 3 months)	1	3	
LBP related diagnoses		0	4	
LBP contacts (mean/5yr)		2.9	2.3	ns
LBP episodes (mean/5yr)		1.7	1.6	ns
LBP contacts/episode		1.7	1.4	ns
Patient-practice contacts (mean/yr)		4.4	3.9	ns

ICPS+ Iliac Crest Pain Syndrome present at initial examination (t=0)
ICPS- Iliac Crest Pain Syndrome not present at initial examination (t=0)
ns not significant

Table 7.10 shows the results of the 5 year follow-up for LLPS+ and LLPS- patients. There were no statistically significant differences between the two groups for the number and duration of the recurrent LBP episodes. The LBP related diagnoses in the LLPS- patients were the same as reported in ICPS- patients. The mean number of patient-practice contacts was higher (6.3 versus 3.6) for the LLPS+ patients than for the LLPS- patients (not significant, Student's t-test, $p \geq 0.05$).

Table 7.10 LBP recurrences and LBP related diagnoses in LLPS+ and LLPS- patients at 5 year follow-up.

	t=0	LLPS+ n=13	LLPS- n=48	
Patients with a complete 5 yr follow-up		10	43	
Patients with recurrent LBP episode after t=0		8 (62%)	41 (85%)	ns
1-5 recurrent LBP episodes		8	40	ns
> 5 recurrent LBP episodes		0	1	
acute episode	(< 4 weeks)	6	35	ns
subacute episode	(4 weeks - 3 months)	1	3	
chronic episode	(> 3 months)	1	3	
LBP related diagnoses		0	4	
LBP contacts (mean/5yr)		2.6	2.3	ns
LBP episodes (mean/5yr)		1.6	1.6	
LBP contacts/episode		1.6	1.5	ns
Patient-practice contacts (mean/yr)		6.3	3.6	ns

LLPS+ Long Ligament Pain Syndrome present at initial examination (t=0)
LLPS- Long Ligament Pain Syndrome not present at initial examination (t=0)
ns not significant

Discussion

At 4 week follow-up, there was a difference in the persistence of pain between QLPS+ (57%) and QLPS- (35%), GMPS+ (70%) and GMPS- (33%), and ICPS+ (63%) and ICPS- (38%) patients, but these differences were not significant. There was no difference concerning the persistence of pain between LLPS+ and LLPS- patients. No association was found between the presence of persistent pain and findings of regional pain syndromes. GMPS, ICPS and LLPS even started to occur in patients who at the initial examination (t=0) had no signs of any regional pain syndrome.

Due to the high overall loss to follow-up, the small numbers of patients with regional pain syndromes, and to the lack of association between persistent pain and finding of regional pain syndromes, any conclusion concerning persistence of pain after 4 weeks in regional pain syndromes must be viewed with caution. In the present study, therefore, no clinical inference can be based on these findings.

In the present study, the overall loss to follow-up at 4 weeks (44%) was considerable. Koes et al. performed a randomized controlled trial on LBP patients in general practice (n=256). They encountered a drop-out of 9% at the follow-up after a period of 12 weeks; an explanation for this could have been the procedure of recruitment, the selection of the LBP patients or the fact that treatment was offered. In Koes' study, most patients (68%) were recruited via announcements in local newspapers and comprised patients with LBP persisting longer than 6 weeks. In fact, in Koes' study population the median duration of the LBP episode was 52 weeks. Furthermore, the selected LBP patients were assigned to manual therapy, physiotherapy, placebo therapy or therapy by the GP (Koes 1992). These factors could have accounted for a higher motivation and compliance to the follow-up regimen in Koes' study.

In the present study, it is unknown whether the patients who were lost to follow-up still experienced LBP at 4 weeks. Some reported that LBP had subsided, but not all of those lost to follow-up reported this or could be reached by telephone. Other reasons may account for non-compliance at the follow-up sessions; it is not always simply because the pain has subsided. For example, the patient may have felt reassured because no serious pathology was found on examination by two other physicians, or the patient wished to avoid another extensive physical examination. If the patients who were lost to follow-up after 4 weeks were considered to be free of pain, the results would be less striking. In roughly 30% of the patients with QLPS or GMPS (this study criteria), or ICPS, in contrast to 20% of the patients without these regional pain syndromes, LBP would still be present. In fact, these percentages on persistent pain come within the range of the estimate from a Dutch morbidity study of 38 GPs in which 20% of the LBP episodes lasted longer than 4 weeks (Lamberts 1991).

Another source of bias could have been the influence of treatment by the GP. The GP was free to decide on the treatment and management of the participating LBP patients. However, Dutch GPs are not accustomed to diagnose QLPS, GMPS, ICPS or LLPS in nonspecific LBP patients. Therefore, it is unlikely that the GPs would have prescribed or applied specific therapy, e.g. injections, for regional pain syndromes.

Only 4 LBP patients (8%) were found to have had a chronic LBP recurrence, i.e. longer than 3 months. This is within the range of chronic LBP estimated in the studies of Lamberts (6%) and Chavannes (9%) (Lamberts 1991, Chavannes 1992). These chronic recurrences did not occur more often in the LBP patients who initially showed signs of one of the four regional pain syndromes.
The follow-up at 5 years showed no difference between LBP patients with the regional syndromes at initial examination and those without the regional pain syndromes for the number of recurrences or duration of the recurrent LBP episodes. However, the number of LBP patients with regional pain syndromes in the present study was too low to substantiate this conclusion with sufficient power. In addition, the results of the 5 year follow-up were based on retrospective analysis of the medical records, much depended on the quality of the GPs' registration. The GPs were not instructed to pay special attention to the participating patients or to register the LBP episodes more carefully. However, the GPs did not know whether or not a regional pain syndrome was found in these patients, so they are not expected to be selective in their recordings.
Most diagnoses which could account for the LBP complaints were found in patients negative for regional pain syndromes at the initial examination. The diagnoses were registered from the medical records and could not be validated. From studies on medical records in general practice, it is known that an average of 63% of major activities are reported in the patient's records (Meyboom 1990). But it is unlikely that serious systemic or malignant diseases would not be recorded by the GP. The type of diagnoses found after 5 years suggests that most were mainly based on X-ray findings. The diagnoses in LBP patients included: lumbar disc disease (n=1), arthrosis (n=2) and scoliosis (n=1). In the present study, 1 case of lumbar disc prolapse occurred (2%) in 61 nonspecific LBP patients. This follows the findings in Hoekstra's study (n=380); at 4 year follow-up a 2% incidence of disc prolapse was found in LBP patients in his own general practice population (Hoekstra 1982).

The mean number of LBP contacts per episode in the present study was approximately 1.5. The Transition Study, a morbidity study in general practice, also reported 1.5 contacts per episode for LBP (Lamberts 1991). One unexpected finding in the present study was that the mean number of patient-practice contacts

per year in patients positive for QLPS, GMPS, and LLPS (6.5, 5.6 and 6.3, respectively) was higher than the 4.7 found in the Dutch National Survey (Groenewegen 1992) on morbidity in general practice. The higher mean value of patient-practice contacts must be due to morbidity other than LBP. Since the present study focused only on LBP related diagnoses, co-morbidity was not registered.

Conclusion: prognostic relevance

Although, at 4 week follow-up, approximately 60% of the patients with QLPS, GMPS or ICPS at initial examination experienced persistent LBP, compared to approximately 35% in patients without QLPS, GMPS or ICPS at initial examination, no clinical inference can be drawn from this finding. Furthermore, the presence of the four regional pain syndromes did not correlate with the persistence of pain.

The follow-up at 5 years revealed no differences for the number of recurrences and duration of the recurrent LBP episodes between LBP patients with the regional syndromes and LBP patients without the regional pain syndromes at the time of the initial examination. No serious diagnoses have been found in patients with the four regional pain syndromes investigated in the present study. Based on the low numbers of the LBP patients with regional pain syndromes these conclusions at 5 year follow-up cannot be substantiated by sufficient power.

References

Barker ME. Pain in the back and leg: a general practice survey. Rheumatol Rehabil 1977;16:37-45.

Biering-Sörensen F. A prospective study of low back pain in a general population. I. Occurrence, recurrence and aetiology. Scand J Rehabil Med 1983;15:71-9.

Chavannes AW. Oefenen bij lage rugpijn. [Thesis] Rijksuniversiteit Utrecht, Utrecht 1992.

Faas A, Chavannes AW, Koes BW, Van den Hoogen JMM, Mens JMA, Smeele LJM, et al. NHG-standaard Lage-Rugpijn. Huisarts Wet 1996;39:18-31.

Frymore JW. Back pain and sciatica. N Engl J Med 1988;318:291-300.

Groenewegen PP, De Bakker DH, Van der Velden J. Een nationale studie naar ziekten en verrichtingen in de huisartspraktijk. Basisrapport: Verrichtingen in de huisartspraktijk. Nederlands Instituut voor onderzoek van de eerstelijnsgezondheidszorg (NIVEL), Utrecht 1992.

Hoekstra GR. Patiënten met lage rugklachten in een huisartspraktijk. [Thesis] Rijksuniversiteit Groningen,

Groningen 1982.

Koes BW, Bouter LM, van Mameren H, Essers AH, Verstegen GM, Hofhuizen DM, Houben JP, Knipschild PG. The effectiveness of manual therapy, physiotherapy, and treatment by the general practitioner for nonspecific back and neck complaints. A randomized clinical trial. Spine 1992;17:28-35.

Lamberts H. In het huis van de huisarts. Verslag van het Transitieproject. Meditekst, Lelystad 1991.

Meyboom WA. Verslagleggen van huisartsgeneeskundig handelen. [Thesis]. Katholieke Universiteit Nijmegen, Nijmegen 1990.

Chapter 8

Combinations of regional pain syndromes

Introduction

The present study observed that regional pain syndromes occur in combinations. Most publications on regional pain syndromes deal with them as separate entities. Some reports, however, indicate that regional pain syndromes are closely related and have been observed to occur in combinations.

A combination of the Sacroiliac Joint Syndrome, Maigne's Syndrome, and Myofascial Pain Syndrome (MFPS) in the gluteus maximus, medius and quadratus lumborum muscles was reported by Bernard (Bernard 1987). Although the names of the syndromes were different, the definition of Sacroiliac Joint Syndrome shows a resemblance to the definition of LLPS, and Maigne's Syndrome to the definition of ICPS. Combinations of these syndromes occurred in 31 (2%) of the 1293 LBP study group (Bernard 1987).

In the Netherlands, Collee performed a prevalence study on ICPS and Greater Trochanter Pain Syndrome in LBP patients in general practice (n=170). Greater Trochanter Pain Syndrome is defined by typical localized tenderness over the trochanteric region. A combination of both syndromes was found in 5 (3%) of the patients (Collee 1991).

Regional pain syndromes are relatively unknown in general practice. Reports on combinations of regional pain syndromes in general practice are not available, with the exception of Collee's study.

Simultaneous occurrence of regional pain syndromes is interesting, because this may imply that regional pain syndromes should not be regarded as separate entities, but as indicators of the severity of pain, or as precursors or signs of another clinical entity, i.e. a syndrome or a disease. The definition of Fibromyalgia Syndrome (FS), for example, includes the criterium of the finding of tender points in at least 11 of the 18 predefined sites (Wolfe 1990). In this definition the tender points are used as an indicator of widespread pain.

In addition, the diagnostic criteria of regional pain syndromes show great similar-

ity, except for the site where the symptoms can be found. All regional pain syndromes incorporate the criterium of a spot of maximal tenderness and when the site of tenderness is palpated the patient recognises the pain as his own "typical" pain. Simultaneous occurrence, therefore, may indicate a similar pathophysiological process. Diagnostic criteria for rheumatoid arthritis, for example, include arthritis in several joints. Inflammation in the joints is considered a presentation of the same disease pathology (Arnett 1988).

Therefore, in this chapter, the following three diagnostic categories will be investigated for LBP: 1) those without regional pain syndrome; 2) those with one regional pain syndrome; and 3) those with a combination of regional pain syndromes. The clinical relevance of such a subdivision is determined, based on whether any differences in prognosis are found between the three categories.

The questions addressed in this chapter are:
1. What is the occurrence of a combination of regional pain syndromes in LBP?
2. Can the occurrence of a combination be attributed to change, interobserver variation or low pain threshold?
3. What is the prognosis of the three diagnostic categories at 4 weeks and at 5 year follow-up?

Material and methods

All LBP patients (n=61) answered a written semi-structured questionnaire and underwent a standard physical examination. The questionnaire investigated patients' sociodemographic and medical variables. Each participant was examined by two observers at the initial examination (t=0). The physical examination consisted of a general (orthopaedic/neurological) examination for LBP and an examination for the characteristics of the regional pain syndromes under study. The presence of QLPS and GMPS was established by the application of the diagnostic criteria found eligible in the present study (Chapter 4). In addition to the sites of the four regional pain syndromes, a dummy site 2 cm lateral to the border of the spinal muscles, was also examined bilateral for tenderness. In this area no trigger or tender points

have been described (Wolfe 1990, Travell 1992). In the present study, the dummy point served as a test of low pressure threshold in the subjects examined (Fig. 3.1).

Follow-up at 4 weeks

All LBP patients (n=61) were asked to return after 4 weeks to answer a questionnaire and undergo another physical examination (t=4). After 4 weeks 27 LBP patients were lost to follow-up, leaving only 34 patients (56%) with a complete 4 week follow-up.

Follow-up at 5 years

Five years after the initial examination the medical records of the LBP patients (n=61) and control subjects (n=63) who had participated in this study were traced. When interpreting the contents of the medical records, the observer did not know whether the participant was a LBP patient or a control subject, nor whether a regional pain syndrome had been present at the examination 5 years previously. Finally, 110 (89%) complete medical records could be retrieved; 53 (87%) for LBP patients and 57 (90%) for control subjects (Table 7.1).

Prognosis was investigated by analyzing the findings of the physical examination at the 4 week follow-up and by review of the medical records at the 5 year follow-up. The results on the prognosis were based on patients in whom both observers were in agreement on the presence of QLPS, GMPS, ICPS, or LLPS.

Outcome measures for the follow-up at 4 weeks included: LBP reported by the patient and associated reproducibility of the regional pain syndrome.

The outcome measures for the follow-up at 5 years included: the number of recurrent LBP episodes, the duration of recurrent LBP episodes, and diagnoses which could account for the nonspecific LBP complaints.

Statistical analysis

One-way analysis of variance (ANOVA) was used to detect differences in the means for the baseline characteristics and outcome measures of the three diagnostic categories investigated in this chapter. Where appropriate, Chi-square test or Fisher's exact test was applied. A p-value below 0.05 was considered statistically significant.

Results

Table 8.1 shows the occurrence of combinations of regional pain syndromes in 61 nonspecific LBP patients, according to the observations of the first and the second blinded observer. The third column of Table 8.1 shows the occurrence of combinations of regional pain syndromes for which the two observers agreed on the presence of the regional pain syndromes.

According to the first observer, 14 (23%) LBP patients had one regional pain syndrome, and 20 (33%) had a combination of regional pain syndromes. As has been concluded previously (Chapters 4 to 6), the observations of the first and second observer did not differ substantially. The combinations of regional pain syndromes occurred in 7 (first observer) or 8 (second observer) different patterns. No single predominant pattern could be distinguished.

When agreement was present between the two observers, one regional pain syndrome was found in 16 (26%) patients, and a combination of regional pain syndromes in 15 (25%) patients. Combinations occurred in 8 different patterns. However, no single predominant pattern could be distinguished, but the combination of all four regional pain syndromes disappeared.

According to the first observer the dummy point was present in 5 patients, of whom 2 had no regional pain syndrome. The second observer found the dummy point to be present in 10 patients, of whom 3 patients were without a regional pain syndrome. When both observers were in agreement, the dummy point was observed in 3 patients, of whom 1 had no regional pain syndrome.

The chance for combinations of regional pain syndromes in LBP patients can be calculated. The chance of one regional pain syndrome to occur is 16/61 = 26%. The chance to find an additional regional pain syndrome is: 26% of the remaining 74% = 19%. Therefore, the expected chance for combinations is 19% x 26% = 5%.

Table 8.1 Combinations of QLPS, GMPS, ICPS, LLPS, and dummy points at t=0 in LBP patients (n=61).

n = 61	First observer	Second observer	Both observers agree
No regional pain syndrome	27 (1D+1d)	24 (2D+1d)	30 (1D)
One regional pain syndrome (uni- and bilaterally)	14 5 QLPS 3 GMPS 3 ICPS 3 LLPS	14 7 QLPS (2d) 2 GMPS 2 ICPS 3 LLPS	16 5 QLPS 5 GMPS 2 ICPS 4 LLPS
Combinations of regional pain syndromes	20 7 QLPS+GMPS (1D) 1 GMPS+ICPS 3 QLPS+GMPS+LLPS 2 QLPS+ICPS+LLPS 2 GMPS+ICPS+LLPS 2 QLPS+GMPS+ICPS 3 QL/GM/IC/LLPS (1D+1d)	23 6 QLPS+GMPS (1d) 2 QLPS+ICPS 3 GMPS+LLPS 3 QLPS+GMPS+LLPS (1d) 2 QLPS+ICPS+LLPS 2 GMPS+ICPS+LLPS 3 QLPS+GMPS+ICPS (1d) 2 QL/GM/IC/LLPS (1d+1D)	15 3 QLPS+GMPS 1 GMPS+ICPS 2 QLPS+ICPS 1 GMPS+LLPS 3 QLPS+GMPS+LLPS (1d) 2 QLPS+ICPS+LLPS 1 GMPS+ICPS+LLPS 2 QLPS+GMPS+ICPS (1d)

(D) = bilateral dummy point
(d) = unilateral dummy point

Table 8.2 shows the demographic and clinical variables at t=0 for the 3 diagnostic categories. These 3 categories of patients were similar for age, reports of LBP duration from onset of the current LBP episode, disturbed routine activities, age at LBP onset and the number of LBP episodes per year. There was a slight female predominance in the category with a combination of regional pain syndromes.
A lower percentage of patients with a combination compared to patients without a regional pain syndrome, reported that the LBP pain had diminished already in the current episode, 27% versus 37%, respectively. Also, of the patients with one or more regional pain syndromes 80% reported sleep disturbances which they attributed to the pain in the low back, compared to 53% of the patients without regional pain syndromes. The pain-free interval between the current and preceding LBP episode for the group with one regional pain syndrome was 40 days; for the group with a combination of regional pain syndromes it was 57 days. The group without regional pain syndromes reported the longest pain-free interval (92 days). The mean duration of the preceding LBP episodes was longer for the groups with one or more regional pain syndromes (9 days) than for the group without regional

pain syndromes (5 days). None of the differences between the 3 diagnostic categories of patients were significant.

Table 8.2 Demographic and LBP variables of the 3 diagnostic categories at the initial examination (t=0).

	No regional pain syndrome	One regional pain syndrome	Combination of regional pain syndromes
	n=30	n=16	n=15
Mean age (yr, (SD))	37 (SD 10)	33 (SD 8)	38 (SD 12)
Sex (% females)	11 (37%)	8 (50%)	8 (53%)
Current LBP episode			
Mean duration from onset (days)	11	13	13
Diminished pain since onset (%)	37	27	27
Disturbed sleep (%)	53	80	80
Impaired daily activities (%)	93	87	87
Mean duration of pain-free interval (days)	92	40	57
Medical history on LBP			
Mean age first LBP episode (yr)	21	22	19
Mean number of LBP episodes/yr	2	3	3
Mean duration of episodes (days)	5	9	9

Means were tested for differences between the 3 groups by ANOVA. Percentages were tested by Chi-square test or Fisher's exact test.

Table 8.3 shows the results from patients with a complete follow-up at 4 weeks. When an LBP patient had a combination of regional pain syndromes at 4 week follow-up, these syndromes persisted in 5 of 9 cases (56%). Patients without regional pain syndromes at initial examination, remained free of regional pain syndromes in 16 of 19 cases (90%) at 4 week follow-up. LBP patients with one regional pain syndrome at initial examination, presented with a combination in 4 of the 8 cases (50%) at 4 week follow-up.

The persistence of reported pain was more frequent in the groups with one or more regional pain syndromes. After 4 weeks, 11 of 17 patients (65%) with one or more regional pain syndromes reported pain, in contrast to 6 of 18 patients (33%) without a regional pain syndrome who reported pain.

Table 8.3 Reproducibility of classification and pain at 4 week follow-up (t=4) of the 3 diagnostic categories.

n=35	Classification at initial examination (t=0)					
	No regional pain syndrome n=18		One regional pain syndrome n=8		Combination of regional pain syndr. n=9	
Classification at t=4		with pain		with pain		with pain
No regional pain syndrome (n=22)	16	5	4	2	2	1
One regional pain syndrome (n=3)	1	-	-		2	2
Combination of regional pain syndromes (n=10)	1	1	4	2	5	4

Table 8.4 shows the results at 5 year follow-up of the 3 diagnostic categories. There were no statistically significant differences between the 3 groups for the number and duration of the recurrent LBP episodes. The patient with more than 5 recurrent LBP episodes had 6 recurrent LBP episodes in 5 years.

The diagnoses in the group without regional pain syndromes were: lumbar disc disease, arthrosis, and scoliosis. The diagnosis in the group with one regional pain syndrome was arthrosis. The number of X-rays, medication prescribed and referrals to the medical specialist were almost the same for each group during the 5 year period.

No differences between the 3 groups were detected (ANOVA, $p < 0.05$) for the mean number of LBP contacts and episodes. The mean number of annual patient-practice contacts for reasons other than LBP was different for the 3 groups. The group without regional pain syndromes had 2.8, the group with one regional pain syndrome 4.4, and the group with combinations 6.1 patient-practice contacts per year (ANOVA, $p < 0.05$). The differences between the 3 groups were not significant.

If the groups with one regional pain syndrome and a combination were combined, the differences for the mean number of LBP contacts and episodes between the combined group and the group without regional pain syndromes was significant (Student's t-test, $p < 0.05$).

Table 8.4 LBP recurrences and diagnoses related to LBP at 5 year follow-up in the 3 diagnostic categories.

	No regional pain syndrome	One regional pain syndrome	Combination of regional pain syndromes
Patients with complete 5 yr follow-up	25	15	13
Patients with recurrent LBP episode after t=0	24	13	12
1-5 recurrent LBP episodes	24	12	12
> 5 recurrent LBP episodes	-	1	-
acute episode (< 4 weeks)	21	11	9
subacute episode (4 weeks - 3 months)	1	1	2
chronic episode (> 3 months)	2	1	1
LBP related diagnoses (No.)	3	1	0
LBP contacts (mean/5yr)	1.8	3.2	2.7
LBP episodes (mean/5yr)	1.3	1.9	1.8
LBP contacts/episode	1.4	1.7	1.5
Patient-practice contacts (mean/yr)	2.8	4.4	6.1 *

t=0 at initial examination
* difference detected (ANOVA, p <0.05)

Discussion

In the present study, a combination of regional pain syndromes occurred in 15 (25%) LBP patients. Bernard's study retrospectively reviewed medical records of 1293 LBP patients who were referred to an outpatient pain clinic; combinations of almost comparable regional pain syndromes were present in 31 (2%) of the LBP patients (Bernard 1987). The discrepancy between the results of Bernard's study and those of the the present study for occurrence, may be due to different methods used for data collection and differences in the patient population. However, in a prospective study (n=170), Collee found a combination of ICPS and the Greater Trochanteric Pain Syndrome in 5 (3%) of the LBP patients in general practice (Collee 1991). The Greater Trochanteric Pain Syndrome is located outside the defined low back region (Collee 1991). The present study has examined regional pain syndromes confined to the low back region. This could account for the higher occurrence of combinations of the regional pain syndromes. Nevertheless, it is investigated whether the high occurrence of combinations found in the present

study are produced by chance, interobserver variance, or by a low threshold for pain.

The expected chance of more than one regional pain syndrome is 5%. In the present study 15/61 (25%) of the LBP patients showed a combination of regional pain syndromes. This is 5 times more than could be expected by chance alone. The occurrence of combinations of regional pain syndromes could be attributed to the level of interobserver agreement on the four regional pain syndromes. In Chapters 4 to 6 the kappa for the four regional pain syndromes was good, but not complete. When both observers were in agreement on the presence of the regional pain syndromes, combinations of regional pain syndromes still occurred at approximately the same rate.

Also, a dummy point was included in the physical examination to test for a low threshold of pain. This was seldom the case, because the finding of a dummy point proved to be very rare.

Thus, it was concluded that combinations of regional pain syndromes were not the result of chance, nor of interobserver variation, nor of low pain threshold. The implication of this finding will be addressed in the general discussion. Consequently, the higher occurrence of combinations in the present study must be real.

The relevance for prognosis of the 3 categories could not be established by the results of the follow-up at 4 weeks and at 5 years. At 5 year follow-up, no differences between the 3 diagnostic categories for the number or duration of the LBP recurrences, and no serious diagnoses were found. Therefore, the classification of nonspecific LBP patients into these 3 categories is not considered useful. Even if the group with one regional pain syndrome was joined with the group with a combination, the difference between the joined group, i.e. one or more pain syndromes, and the group without regional pain syndromes is minor at both times of follow-up. At the 4 week follow-up differences were found in the persistence of pain. The differences at the 5 year follow-up involved the mean number of contacts and episodes for LBP, and the mean number of patient-practice contacts per year. The number of patient-practice contacts was 2.8 for the group without a regional pain syndrome. In the Dutch National Survey of general practice, which registered

all patient-practice contacts in 161 general practices, the number of patient-practice contacts was 4.7 per year (Groenewegen 1992). Thus, LBP patients without regional pain syndromes may be considered low consumers of general practice services.

Conclusion on the 3 diagnostic categories

In the present study, a combination of regional pain syndromes occurred in 15 (25%) LBP patients. Because combinations were more prevalent in the present study than in other studies, several sources of bias were investigated. The high occurrence of combinations in regional pain syndromes was not the result of chance, nor of interobserver variation, nor of low pain threshold. Consequently, the higher occurrence of combinations in the present study can be considered as real.

The relevance for prognosis of the 3 categories, i.e. LBP patients 1) without regional pain syndrome; 2) with one regional pain syndrome; and 3) with a combination of regional pain syndromes, could not be established by the results of the follow-up at 4 weeks and at 5 years. Therefore, the classification into these three groups is not considered to have prognostic value in nonspecific LBP patients.

References

Arnett FC, Edworth SM, Bloch DA et al. The American Rheumatism association 1987 revised criteria for the classification of rheumatoid arthritis. Arthritis Rheum 1988;31:315-24.

Bernard TN, Kirkaldy-Willis WH. Recognizing specific characteristics of nonspecific low back pain. Clin Orthop 1987;217:266-80.

Collee G. Clinical syndromes in low back pain. [Thesis] University of Leiden, Leiden 1991

Groenewegen PP, De Bakker DH, Van der Velden J. Een nationale studie naar ziekten en verrichtingen in de huisartspraktijk. Basisrapport: Verrichtingen in de huisartspraktijk. Nederlands Instituut voor onderzoek van de eerstelijns gezondheidszorg (NIVEL), Utrecht 1992. [Dutch national survey of general practice. Utrecht, Netherlands Institute of Primary Health Care (NIVEL)]

Wolfe F, Smythe HA, Yunus MB, Bennett RM, Bombardier C, Goldenberg DL, et al. The American College of Rheumatology 1990 criteria for the classification of fibromyalgia. Report of the multicenter criteria committee. Arthritis Rheum 1990;33:160-72.

Chapter 9

General discussion

The main objective of the present study was to investigate the clinical relevance of a subdivision of nonspecific low back pain (LBP) in four regional pain syndromes. The four regional pain syndromes are Quadratus Lumborum Pain Syndrome (QLPS), Gluteus Medius Pain Syndrome (GMPS), Iliac Crest Pain Syndrome (ICPS), and Long Ligament Pain Syndrome (LLPS). These regional pain syndromes are considered clinically relevant, when there is an association between the presence of the diagnostic criteria of the syndromes and LBP; a good level of interobserver agreement for the criteria and for each syndrome; a sufficient occurrence of the four regional pain syndromes in LBP patients; and a prognosis other than for nonspecific LBP patients in general practice. This chapter addresses the main results of the present study and discusses the validity and the possible implications of the conclusions.

Concerning prognosis of the presence of QLPS, GMPS, ICPS and LLPS in nonspecific LBP patients in general practice, a difference in the number or duration of LBP recurrences, or the development of serious diagnoses could not be demonstrated between LBP patients with and LBP patients without the regional pain syndromes at the time of the initial examination (Chapter 7). These conclusions are based mainly on data retrieved from the general practitioner's (GP) reports in the medical records at 5 year follow-up. This implies that much depended on the quality of the content of these medical records, which are known to serve merely as mental notes of the GP than as an accurate registration of all medical problems presented at the encounters with the patients. As to the registration of serious diagnoses in the medical history of a patient, such as disc prolaps or metastases, medical records are reliable. The choice for reviewing the medical records is based on the high drop-out rate at 2 and 4 week follow-up in the present study, combined with the expected recall bias by the LBP patients as to the number and duration of their recurrent LBP episodes over long periods of time, let alone the past 5 years. Secondly, the data were retrieved retrospectively, which may have caused individ-

ual interpretation concerning the number and duration of the recurrent LBP episodes. Finally, the number of LBP patients in the present study was too low to substantiate this conclusion with sufficient power.
Thus, to come to a conclusion with regard to prognosis of the four regional pain syndromes, a prospective standard registration on the outcome of LBP patients with these syndromes is necessary. To detect a 10% increase of the present percentage of 6% of chronic outcome, at a level of .05 for a type I error and with a 90% probability of detecting a true difference, the estimated number of patients needed in each group is 148. In the present era of computerization in general practice, it becomes feasible to attach a specific message to the patient's record, reminding the GP to pay special attention to register LBP episodes when they occur. Also, it is possible to register the LBP episodes in a standard manner. On paper, reminders tend to get overlooked or lost; and registration, especially for a long period of follow-up, may be forgotten.

The results of the follow-up at 4 weeks also remain inconclusive. It was found that approximately 60% of the patients with QLPS, GMPS or ICPS at initial examination, experienced persistent LBP, compared to approximately 35% in patients without QLPS, GMPS or ICPS at initial examination. At 4 weeks, there was no difference in reported pain between patients with LLPS and without LLPS at initial examination (Chapter 7). These results on QLPS, GMPS, and ICPS may seem striking, considering that in general practice 20% of the LBP episodes last longer than 4 weeks (Lamberts 1991). However, this finding must be interpreted with great caution, because it may have been influenced by the considerable loss to follow-up (44%) and the consequently low number of patients with each regional pain syndrome at 4 weeks.
A reason for the high drop-out rate could be that no "reward" of any kind, such as education or some other form of information on LBP, was offered to the LBP patients, perhaps making them less inclined to participate or to comply with the follow-up appointments. Other reasons that may account for the non-compliance at the follow-up sessions are: reassurance that no serious pathology was found on examination by two other physicians; dissatisfaction with the study procedure; avoidance of another extensive physical examination; or reduction of the pain.

If the latter is the case, this may have introduced an overestimation of the percentage of LBP patients experiencing persistent pain at 4 weeks.
To prevent the high drop-out rate at follow-up, a home visit may have been considered at 2 and 4 weeks. However, home visits impedes the physical examination, because conditions at home are not optimal to perform a standardized physical examination.

In clinical research the identification of subgroups can be useful, because it may reduce the clinical heterogeneity of nonspecific LBP. Homogeneous groups with regard to symptoms and signs, may facilitate research on aetiology, clinical course and results of interventions. The present study provides for more specific knowledge on the four regional pain syndromes in nonspecific LBP. The clarity of the diagnostic criteria and definition especially for QLPS and GMPS, have been improved. Moreover, the association between the regional pain syndromes and LBP; the level of interobserver agreement; and the occurrence in the general practice population have been established.

The presence of the diagnostic criteria for QLPS, GMPS, ICPS, and LLPS was examined by a controlled study and an interobserver agreement study. The two diagnostic criteria, localized tenderness and the patient's recognition of the typical pain, prove to be associated with the presence of LBP. Jump sign is associated with the presence of LBP only for QLPS and GMPS.
The diagnostic criteria for the regional pain syndromes are generally considered as subjective, but the level of interobserver agreement on the diagnostic criteria mentioned above was good. Kappa was in the range of more accepted symptoms for the physical examination of the low back, e.g. between a kappa of 0.4 for pain in leg at the Straight Leg Raising test (McCombe 1989) and 0.6 for root compression signs (Waddell 1982). This means that, after some training sessions, the presence of these four regional pain syndromes can be established in the clinical setting and in research at a good level of reproducibility between observers.
Based on the association between the regional pain syndromes and LBP, and the good level of interobserver agreement, some of the inconsistencies and confusion on the diagnostic criteria for and definition of QLPS and GMPS were clarified. A

recommendation is made to use the definition found eligible in the present study, i.e. localized tenderness, and the presence of either jump sign or patient's recognition of the pain complaint (Chapter 4). Because this definition is investigated for QLPS and GMPS only, similar studies on other regional pain syndromes that are associated with LBP, should be performed.
The regional pain syndromes may be considered as more homogeneous groups with regard to the diagnostic criteria. Theoretically, this improves the feasibility of clinical trials and the comparability of the treatment groups. A clinical trial on injection therapy with a local anaesthetic can thus be performed in more homogeneous groups.
The claimed success of injection therapy may be based on the immediate effect of a local anaesthetic, and/or the psychological impact following an injection. The placebo effect of such measures is not to be underestimated. Studies on the beneficial effects of dry needling (Lewitt 1979, Gunn 1980) or saline injections (Frost 1980) can be found in medical literature.

In the present study, the four pain syndromes are present in a relatively large percentage of LBP patients in general practice. The occurrence of QLPS and GMPS (according to this study criteria), ICPS and LLPS in LBP patients is 36%, 34%, 21% and 21%, respectively (Chapters 4, 5, 6).
In the present study, a relatively low admission rate of 45% of the expected number of LBP patients and the surprisingly large number (21%) of selected LBP patients, who did not show up at the appointment for the initial examination (Chapter 4, discussion) may have influenced these occurrences.
The low admission rate can only be partially explained by the application of selection criteria; selection bias by the GPs may also be involved. It is not expected, however, that selection bias is directed towards the presence of regional pain syndromes, since Dutch GPs are not accustomed to diagnose these pain syndromes in LBP patients. In addition, LBP patients did not appear to be more motivated than the control subjects to participate in the present study, but there is little chance that non-compliance for the initial examination is related to the presence or absence of regional pain syndromes.
Accordance with the prevalence for ICPS (27%) found in another clinical study in

Dutch general practice (Collee 1991) give some evidence that selective drop-out or non-compliance is not the case, at least for ICPS. Furthermore, at 5 year follow-up, the selected LBP patients are observed to show approximately the same estimates for the mean number of contacts per LBP episode (1.5), the number of recurrent LBP episodes of a duration longer than 3 months (8%), and the percentage of referrals for X-ray in 1 year (5%) as reported in another study in general practice (Lamberts 1991) (Chapter 7). This substantiates the impression that the selected LBP patients were representative for the group of LBP patients usually seen in general practice.

Comparison with the prevalence for QLPS, GMPS, ICPS and LLPS found in other clinical studies (Bernard 1987, Collee 1991, Mens 1992) leads to interesting hypotheses on the aetio-genesis of the regional pain syndromes.
QLPS and GMPS were present in 2% and 1%, respectively, of LBP patients attending a pain clinic in the USA (Bernard 1987). Although Bernard collected his data retrospectively, registration bias cannot be the only reason for the low prevalence of QLPS and GMPS in his study. Likely, these syndromes are more prevalent in LBP episodes of recent onset, supporting the assumption of Simons and Travell that Myofascial Pain Syndrome (MFPS) develops under an acute stress on the muscles (Simons 1983).
ICPS has been reported to be more prevalent in a rheumatology clinic (58%) (Collee 1991) than in general practice (27%). This could suggest that ICPS is associated with a history of LBP of a longer duration or with rheumatological syndromes. In the rheumatology clinic, ICPS has clinical relevance because an injection of local anaesthetic demonstrated a beneficial effect on the pain score and pain severity compared to an injection of isotonic saline (Collee 1991). Collee also performed a randomized trial in general practice (total n=17); here both treatments resulted in the same outcome. This confirms the rule that results from the clinical setting should not be applied to general practice without critical evaluation (Wulff 1980). Whether there was no difference or whether Collee's result in general practice was due to the small numbers of the randomized trial, remains to be investigated.
The case histories on LLPS described by GPs (Broadhurst 1989, Leblanc 1992)

could be confirmed by the present study. The occurrence of LLPS in LBP patients in general practice is 21%. In a Dutch survey, 42% of 394 women with chronic pain reported tenderness in the area of the long ligament (Mens 1992). This is a higher occurrence than found in the present study, but Mens' study provides only circumstantial evidence. His survey was held in a highly selected group of women, all members of the Dutch society for patients with pelvic complaints related to symphiolysis. Therefore, it remains inconclusive whether women after childbirth are at risk for developing LLPS.

The present study showed that combinations of the regional pain syndromes can occur in the same patient. A combination was found present in 25% of the nonspecific LBP patients (Chapter 8). Because combinations were more prevalent in the present study than in two other studies, i.e. 2% (Bernard 1987) and 3% (Collee 1991), several sources of bias were investigated. The high occurrence of combinations of regional pain syndromes in nonspecific LBP patients was not the result of chance, nor of interobserver variation, nor of low pain threshold (Chapter 8).

This could imply that the regional pain syndromes investigated in the present study should not be regarded as separate entities, but rather as an indicator for the extent of the area of pain, or as precursors or signs of another clinical entity, i.e. a syndrome or a disease. However, by the choice of the syndromes, sites and the diagnostic criteria, the present study may have introduced this close relation between the four regional pain syndromes. Two of the four regional pain syndromes investigated in the present study belong to the category of MFPS and were located in muscles. MFPS is suggested to have a similar underlying pathophysiological process. Except for the site of localized tenderness, the diagnostic criteria of LLPS were formulated analogous to that of ICPS, because both are 'ligamentous' regional pain syndromes.

It remains possible that LBP patients with regional pain syndromes have a soft tissue pain syndrome, which comes to expression at different locations. This is analogous to the concept of Fibromyalgia Syndrome (FS), which includes widespread pain with findings of tender points in at least 11 of the 18 predefined sites (Wolfe 1990).

However, the main difference between FS and MFPS remains the finding of widespread pain. Patients in the present study complained about pain in the low back. No accompanying symptoms such as severe sleep disturbances, were found. Also, outcome for LBP patients with a combination of regional pain syndromes at 5 year follow-up was not different than that for LBP patients with one regional pain syndrome or with no regional pain syndrome.
In the medical literature on FS and MFPS, it is postulated that MFPS and the other regional pain syndromes could be considered as a precursor of FS (Bennett 1990). However, this hypothesis cannot be confirmed in the present study, since in 87% of the patient records examined over a period of 5 years no indication of FS was found.
In conclusion, the clinical relevance of the four regional pain syndromes could only be substantiated for an association between these syndromes and LBP, a good level of interobserver agreement and a sufficient occurrence in LBP. Concerning prognosis no inference can be concluded, because the lack of power of the results in the present study.

The problem of all research on nonspecific LBP remains that knowledge on its pathophysiology and aetiology is not as far advanced as, for instance, knowledge on diabetes or cardiac failure. Pain remains largely a mystery to physicians and researchers alike. Critical evaluation of clinical observations and subsequent research studies may slowly disclose the underlying mechanisms. In my opinion, further research is needed to explore the factors which influence the prognosis of LBP and to determine subgroups in nonspecific LBP on the basis of clinical observations.

References

Bennett RM. Myofascial pain syndromes and the fibromyalgia syndrome: A comparative analysis. In: Fricton JR, Awad E ed. Advances in pain research and therapy volume 17. Raven Press, New York, 1990: 43-65.

Bernard TN, Kirkaldy-Willis WH. Recognizing specific characteristics of non specific low back pain. Clin Orthop 1987;217:266-80.

Broadhurst NA. Sacroiliac dysfunction as cause of low back pain. Aust Fam Physician 1989;18:623-8.

Collee G. Clinical syndromes in low back pain. [Thesis] University of Leiden, Leiden, the Netherlands 1991.

Faas A, Chavannes AW, Koes BW, Van den Hoogen JMM, Mens JMA, Smeele LJM, et al. NHG-standaard Lage-Rugpijn. Huisarts Wet 1996;39:18-31.

Frost FA, Jessen B, Siggaard-Andersen J. A control, double blind comparison of mepivacaine injection versus saline injection for myofascial pain. Lancet 1980;1(8167):499-501.

Gunn CC, Milbrandt WE, Little AS, Mason KE. Dry needling of muscle motor points for chronic low back pain. A randomized clinical trial with long term follow-up. Spine 1980;5:279-91.

Lamberts H. In het huis van de huisarts. Verslag van het Transitieproject. Lelystad: Meditekst 1991.

Leblanc KE. Sacroiliac sprain: an overlooked cause of back pain. Am Fam Phys 1992;46:1459-63.

Lewitt K. The needle effect in the relief of myofascial pain. Pain 1979;6:83-90.

McCombe PF, Fairbank JCT, Cockersole BC, Pynsent PB. Reproducibility of physical signs in low back pain. Spine 1989:14:908-18.

Mens JAM, Stam HJ, Stoeckart R, Vleeming A, Snijders CJ. Peripartum pelvic pain; a report of analysis of an inquiry among patients of a Dutch patient society. Proc of the First Int Congr on Low Back Pain and the SI joint. San Diego, 1992.

Simons DG, Travell JG. Myofascial origins of low back pain. 1. Principles of diagnosis and treatment. Postgrad Med 1983; 73:66-77.

Van der Velden J, De Bakker DH, Claessens AAMC, Schellevis FG. Een nationale studie naar ziekten en verrichtingen in de huisartspraktijk. Basisrapport: Morbiditeit in de huisartspraktijk. Utrecht, NIVEL 1991. [Morbidity in general practice. Dutch national survey of general practice. Utrecht: Netherlands Institute of Primary Health Care (NIVEL), 1992.]

Waddell G, Main CJ, Morris EW, Venner RM, Rae PS, Sharmy SH, et al. Normality and reliability in the clinical assessment of backache. BMJ 1982;284:1519-23.

Wolfe F, Smythe HA, Yunus MB, Bennett RM, Bombardier C, Goldenberg DL, et al. The American College of Rheumatology 1990 criteria for the classification of fibromyalgia. Report of the multicenter criteria committee. Arthritis Rheum 1990;33:160-72.

Wulff H. Principes van klinisch denken en handelen. Bohn, Scheltema en Holkema, Utrecht, 1980. [Rational diagnosis and treatment. Blackwell Scientific Publications, Oxford, 1976.]

Chapter 10

Summary

The main objective of the present study was to investigate the clinical relevance of regional pain syndromes in nonspecific low back pain (LBP).

Chapter 1 introduces the problems of LBP. In general practice, LBP is the most prevalent of all musculoskeletal disorders. Its clinical course is usually self-limiting and most episodes (80%) resolve within 4 weeks. In most cases no underlying pathology, or specific cause can be found. It is estimated that 90% of LBP patients is classified as nonspecific. Patients with nonspecific LBP are a heterogeneous group regarding aetiology and prognosis. Nonspecific LBP probably comprises of several partially unidentified subgroups.
In clinical research, however, the identification of subgroups may be useful, because this may reduce the heterogeneity of nonspecific LBP. Homogeneous groups, preferably with regard to prognosis, facilitate research on aetiology, clinical course, and effects of interventions. This implies that research on nonspecific LBP should focus primarily on diagnostics and not on therapy.
In clinical practice, the purpose of recognizing diagnostic categories is not a goal in itself, but a tool for prognosis. Chronic outcome for nonspecific LBP cannot be predicted. The GP needs to discriminate beforehand those patients with a favourable outcome from those likely to have a chronic outcome, this because the latter group will make greater demands on health care resources and on the social insurance.
LBP is not a disease, but a symptom. Clinical knowledge on nonspecific LBP has barely evolved from the stadium of symptom diagnosis. Research should aim to distinguish subgroups on the syndrome or causal diagnosis level, in order to establish the clinical relevance of these diagnoses.
In this work, four regional pain syndromes have been studied: Quadratus Lumborum Pain Syndrome (QLPS), Gluteus Medius Pain Syndrome (GMPS), Iliac Crest Pain Syndrome (ICPS) and Long Ligament Pain Syndrome (LLPS).

Chapter 2 presents a review of the medical literature on these four regional pain syndromes. Each syndrome is known by different names. The diagnostic criteria to establish the presence of the syndromes are not unambiguous. There is great variation in both the quantity and quality of research on the four regional pain syndromes.
No international consensus on the criteria or definition of QLPS and GMPS has been formulated. The definition proposed by Simons in 1990 seems to be the most recent and operational definition, which includes: 1) localized tenderness and 2) referred pain; and when applicable: 3) a taut, palpable band in the muscle concerned; 4) limited stretch range of the muscle concerned, and/or 5) a twitch response on needling. Reproduction of the patient's pain, and jump sign have also been reported in other criteria sets.
ICPS has been described under different names. Recently, Collee proposed a clear definition of ICPS. The diagnostic criteria included localized tenderness, defined as a point of localized maximal tenderness on palpation of the medial part of the iliac crest near the attachment of the iliolumbar ligament; and typical pain, which is recognized by the patients as their own typical characteristic pain, i.e. recognition. In the present study the diagnostic criteria of LLPS are considered to be analogous to those of ICPS, which is also a regional pain syndrome in a ligament: 1) maximal tenderness is found on palpation of the long dorsal sacroiliac ligament, and 2) the tenderness is recognized by the patients as their own "typical" pain.
Although imprecise in definition and lacking evidence, regional pain syndromes have the advantage of being rooted in clinical observations and can be managed by the GP. Furthermore, they provide for a diagnosis on a syndrome level and, consequently, a subdivision of nonspecific LBP.

Chapter 3 introduces the present study and describes the material and methods used. The aims of this study are to: 1) refine the diagnostic criteria for the four regional pain syndromes by investigating the association between the diagnostic criteria of the syndromes and LBP, and the interobserver agreement on the diagnostic criteria; 2) estimate the occurrence of the four regional pain syndromes in LBP patients in general practice (Chapters 4 to 6); 3) study the prognosis of the regional pain syndromes (Chapter 7); and 4) investigate simultaneous occurrence of

the four regional pain syndromes (Chapter 8).

A total of 124 participants were recruited from 11 general practices; 61 nonspecific LBP patients and 63 control subjects. All participants answered a written semi-structured questionnaire and underwent a standard physical examination. Each participant was examined by 2 observers. All LBP patients (n=61) were asked to return after 2 and 4 weeks to answer a questionnaire and undergo another physical examination. Only results of patients with a complete 4 week follow-up will be presented, because the follow-up at 2 weeks reveals no valuable information. Only 34 LBP patients (56%) showed up at the 4 week follow-up.
Five years after the initial examination the medical records of 53 LBP patients (87%) and 57 controls (90%) were retrieved. The observer was kept uninformed as to who were LBP patients and control subjects, or who had shown signs of regional pain syndromes at the initial examination 5 years previously.

Chapter 4 presents the results on the association between the diagnostic criteria of QLPS and GMPS, and LBP. Interobserver agreement and the occurrence of QLPS and GMPS in LBP patients in general practice are also reported.
The results of the present study indicate that localized tenderness and the presence of either jump sign or patient's recognition of his pain complaint are clinically useful for the presence of QLPS and GMPS. When applying these criteria in nonspecific LBP patients the occurrence of QLPS is 36% and 34% for GMPS; in controls it is 6% for both syndromes (significant, Chi-square test, $p < 0.05$). The occurrence of both syndromes is equally distributed between male and female LBP patients; the mean age is 36 years. The level of interobserver agreement is good: kappa is 0.66 for QLPS and 0.62 for GMPS.
Of the major criteria reported by Simons in 1990 only localized tenderness proved clinically useful for QLPS and GMPS. The diagnostic criteria: referred pain (major criterium); palpable band; limited stretch range, and twitch response were clinically not useful, because there was no association between these diagnostic criteria and LBP, and a poor level of interobserver agreement.
Thus, until the underlying pathophysiological cause is disclosed, in research and

clinical practice it would be appropriate to use the definition found eligible in the present study for QLPS and GMPS.

Chapter 5 presents the results on the association between the diagnostic criteria of ICPS and LBP, together with the level of interobserver agreement and the occurrence of ICPS. Collee's diagnostic criteria of ICPS prove to be clinically useful. ICPS is present in LBP patients (21%) but is absent in controls. The prevalence is equally distributed between males and females and the mean age of ICPS patients is 34 years. The presence of ICPS can be judged with good interobserver agreement; kappa is 0.57.

Chapter 6 presents the results on the association between the diagnostic criteria of LLPS and LBP, the interobserver agreement, and the occurrence of LLPS. LLPS was found to be related to the presence of LBP and was rarely found in controls. The occurrence of LLPS in LBP patients in general practice is 21%. Prevalence is equally distributed between male and female LBP patients with a mean age of 37 years. The presence of LLPS can be established at a good level of interobserver agreement; kappa is 0.65.

Chapter 7 summarizes the results of the prognosis on the four regional pain syndromes. Prognosis was evaluated by the outcome of follow-up at 4 weeks and 5 years. Outcome measures for the follow-up at 4 weeks included: reported LBP by the patient and reproducibility of the regional pain syndrome. The outcome measures for the follow-up at 5 years included: the number and duration of the recurrent LBP episodes, and the diagnoses which could account for the recurrent LBP episodes.

Although, the follow-up at 4 weeks showed a difference for persistence of pain between LBP patients with a regional syndrome and without a regional pain syndrome at the time of initial examination, no clinical inference can be drawn from this finding, because the numbers were small and the overall loss to follow-up high

(44%). Furthermore, at 4 weeks, the presence of the four regional pain syndromes did not correlate with the persistence of pain.
The follow-up at 5 years showed no serious diagnoses in patients with the regional pain syndromes. Also no difference was found for the number or the duration of the recurrent LBP episodes between LBP patients with a regional syndrome and without a regional pain syndrome at the initial examination. The power of this conclusion is weak. Therefor, whether a subdivision of LBP in QLPS, GMPS, ICPS and LLPS has prognostic relevance, remains a subject for future research.

Chapter 8 explores the presence of combinations of the four regional pain syndromes. Publications on regional pain syndromes deal with them separately. In the present study, a combination of regional pain syndromes occurred in 15 (25%) LBP patients. Because combinations were more prevalent in the present study than in other studies, several sources of bias were investigated. The high occurrence of combinations in regional pain syndromes was not the result of chance, nor of interobserver variation, nor of low pain threshold. Consequently, the higher occurrence of combinations in the present study can be considered as real.
The relevance for prognosis of the 3 categories, i.e. LBP patients 1) without regional pain syndrome; 2) with one regional pain syndrome; and 3) with a combination of regional pain syndromes, could not be established by the results of the follow-up at 4 weeks and at 5 years. Therefor, the classification into these three groups is not useful for predicting outcome in nonspecific LBP patients.

Chapter 9 addresses the main conclusions of the present study and discusses the validity and the possible implications of these conclusions.

Due to the fact that Chapters 4 to 8 report on the results of the present study, the content of the introduction of these chapters, repetitions of sections of Chapters 1 to 3 are inevitable. An advantage of this approach is that the individual chapters can be read separately.

Samenvatting

(Summary in Dutch)

Het doel van deze studie is het vaststellen van de klinische betekenis en prognose van regionale pijn syndromen bij patiënten met aspecifieke lage rugpijn (LRP).

Hoofdstuk 1. LRP is in de huisartspraktijk de meest prevalente musculoskeletale aandoening. Het natuurlijk beloop is zelflimiterend. Het merendeel van de LRP episodes (80%) is na 4 weken voorbij en er kan geen onderliggend lijden of specifiek oorzaak worden vastgesteld. De schatting is dat 90% van de of LRP patiënten wordt geclassificeerd als aspecifieke LRP. Patiënten met aspecifieke LRP vormen een heterogene groep voor wat betreft de etiologie en de prognose. Aspecifieke LRP bestaat waarschijnlijk uit verschillende subgroepen. Het onderscheiden van deze subgroepen is een het centraal thema van dit proefschrift.

Voor klinisch onderzoek is het onderscheid in homogene subgroepen zinvol. Homogene groepen leveren valide conclusies over de prognose, etiologie, natuurlijk beloop en het effect van therapeutische interventies.
Voor de klinische praktijk is het onderscheiden van subgroepen (diagnoses) niet een doel op zichzelf, maar een middel om tot een prognose te komen. Een huisarts zou een onderscheid moeten kunnen maken tussen patiënten met een gunstig natuurlijk beloop en de patiënten met een chronisch LRP. Momenteel is nog geen goed onderscheid te maken.
LRP is namelijk geen ziekte, maar een symptoom. Medische kennis over aspecifiek LRP heeft zich nauwelijks verder ontwikkeld dan het stadium van een symptoom diagnose. Onderzoek zou zich bezig moeten houden met het onderscheiden van homogene subgroepen op het niveau van een syndroom of causale diagnose.
In dit proefschrift wordt het onderscheid bestudeerd tussen vier regionale pijnsyndromen, te weten: het Quadratus Lumborum pijnsyndroom (QLPS), het Gluteus Medius pijnsyndroom (GMPS), het Iliac Crest pijnsyndroom (ICPS) en het Lange Ligament pijnsyndroom (LLPS), om de klinische betekenis en de prognose te kunnen vaststellen.

Hoofdstuk 2 geeft een overzicht van de medische literatuur over deze vier regionale pijnsyndromen. Elk syndroom is bekend onder verschillende namen en de diagnostische criteria voor de aanwezigheid van het syndroom zijn niet eenduidig. Zo is er geen internationale consensus over de criteria of definitie van QLPS en GMPS. De definitie van Simons (1990), is de meest operationele definitie: 1) gelokaliseerde pijn en 2) gerefereerde pijn; en indien van toepassing: 3) een palpabele streng; 4) een beperkte stretch range in de betreffende spier, en/of 5) een twitch response bij het aanprikken met een naald. In andere publikaties worden bovendien herkenning van de pijn en het 'jump sign' ook genoemd.
Ook ICPS is beschreven onder verschillende namen. Collee geeft een duidelijke definitie voor ICPS. De diagnostische criteria zijn: een gelokaliseerde plek van maximale drukpijn op het mediale deel van de crista iliaca in de buurt van de aanhechting van het iliolumbale ligament en pijn die door de patiënt wordt herkend als zijn typische pijn.
Diagnostische criteria van LLPS worden op dezelfde wijze als die van ICPS gekozen, omdat beide een regionaal pijnsyndroom in een ligament of aanhechting van een ligament zijn, te weten: 1) maximale drukpijn op de lange dorsale sacro-iliacale ligament, en 2) herkenning door de patiënt van zijn typisch pijn.
Hoewel de definities niet altijd even duidelijk zijn en de onderbouwing van de syndromen nog niet ver is gevorderd, zijn regionale pijnsyndromen gebaseerd op klinische observaties en kunnen door eenvoudig lichamelijk onderzoek in de huisartspraktijk worden vastgesteld. Deze pijnsyndromen geven bovendien de mogelijkheid om bij LRP diagnoses te stellen op het syndroom niveau en hiervan homogene subgroepen te onderscheiden.

Hoofdstuk 3 geeft een beschrijving van het patiënten-gebonden onderzoek. Het beschrijft de onderzoeksdoelen en methoden. De doelstellingen van het onderzoek zijn het vaststellen van: 1) de bruikbaarheid van de diagnostische criteria van de vier regionale pijnsyndromen, 2) het voorkomen van de vier regionale pijnsyndromen in LRP patiënten in de huisartspraktijk (Hoofdstuk 4 t/m 6), 3) de prognose van de regionale pijnsyndromen (Hoofdstuk 7); en 4) het voorkomen van combinaties van de vier regionale pijnsyndromen (Hoofdstuk 8).
De bruikbaarheid van de diagnostische criteria wordt onderzocht door de relatie tussen de diagnostisch criteria van de pijnsyndromen en LRP, en de onderlinge

overeenstemming tussen twee beoordelaars vast te stellen.
Door 11 huisartsen zijn 124 deelnemers aangemeld; 61 aspecifiek LRP patiënten en 63 controle personen. Alle deelnemers vulden een schriftelijke semigestructureerde vragenlijst in en kregen vervolgens een geprotocoleerd lichamelijk onderzoek. Elke deelnemer werd door 2 onderzoekers onderzocht. De LRP patiënten (n=61) werden verzocht om na 2 en 4 weken terug te komen voor een vervolgonderzoek. Er verschenen 34 LRP patiënten (56%) voor het vervolgonderzoek.
Vijf jaar na het eerste onderzoek zijn de medische dossiers van 53 LRP patiënten (87%) en 57 controle personen (90%) opgespoord. De onderzoeker die de medische dossiers bestudeerde, was niet op de hoogte welke dossiers van LRP patiënten en welke van controle personen waren, of bij welke patiënten 5 jaar geleden een regionale pijnsyndromen was vastgesteld.

Hoofdstuk 4 bevat de resultaten van het onderzoek naar de relatie tussen de diagnostische criteria van QLPS/GMPS en LRP, de onderlinge overeenstemming en het voorkomen van QLPS en GMPS bij LRP patiënten in de huisartspraktijk. Het blijkt dat de volgende diagnostische criteria bruikbaar zijn voor het vaststellen van QLPS en GMPS: gelokaliseerde pijn en de aanwezigheid van of 'jump sign' of de herkenning van de typische pijn door de patiënt. Op basis van deze criteria is het voorkomen bij aspecifiek LRP patiënten van QLPS 36% en GMPS 34%; bij controle personen komen beide syndromen voor bij 6% (significant, Chi^2 test, $p < 0,05$). Er is geen verschil tussen mannen en vrouwen; de gemiddelde leeftijd is 36 jaar. De onderlinge overeenstemming tussen observatoren is goed: kappa is 0,66 voor QLPS en 0,62 voor GMPS.
Op basis van deze bevinding wordt de definitie van Simons (1990) bijgesteld. Uit zijn definitie is alleen het criterium gelokaliseerde pijn bruikbaar gebleken voor QLPS en GMPS. De diagnostisch criteria: gerefereerde pijn (major criterium); palpabele streng; beperkte stretch range en twitch response bleken niet bruikbaar voor de kliniek, omdat er geen relatie is met de aanwezigheid van LRP en onvoldoende onderlinge overeenstemming tussen onderzoekers over de aan- of afwezigheid van elk criterium.
Totdat de oorzaak dat aan QLPS en GMPS ten grondslag ligt is gevonden, zou voor de diagnose van QLPS en GMPS de criteria die in dit onderzoek bruikbaar bleken te zijn gebruikt dienen te worden.

Hoofdstuk 5 beschrijft de resultaten van het onderzoek naar de relatie tussen de diagnostische criteria van ICPS en LRP, de onderlinge overeenstemming en het voorkomen van ICPS. Collee's diagnostische criteria van ICPS blijken bruikbaar. ICPS is aanwezig bij 21% van de LRP patiënten en afwezig bij controle personen. ICPS komt evenveel voor bij mannen als bij vrouwen en de gemiddelde leeftijd van ICPS patiënten is 34 jaar. De aanwezigheid van ICPS kan met goede onderlinge overeenstemming worden vastgesteld; kappa is 0,57.

Hoofdstuk 6 geeft de resultaten weer van het onderzoek naar de relatie tussen de diagnostische criteria van LLPS en LRP, de onderlinge overeenstemming en het voorkomen van LLPS. LLPS is aanwezig bij 21% van de LRP patiënten en zelden bij controle personen. LLPS komt evenveel voor bij mannen als bij vrouwen en de gemiddelde leeftijd van LLPS patiënten is 37 jaar. De aanwezigheid van ICPS kan met goede onderlinge overeenstemming worden vastgesteld; kappa is 0,65.

Hoofdstuk 7 gaat over de prognose van de vier regionale pijnsyndromen. De prognose werd geëvalueerd aan de hand van het vervolgonderzoek na 4 weken en na 5 jaar. Uitkomstmaten voor het vervolgonderzoek na 4 weken zijn: door de patiënt gerapporteerde LRP en reproduceerbaarheid van het regionaal pijn syndroom. De uitkomstmaten voor de follow-up na 5 jaar zijn: aantal en duur van de LRP recidieven en diagnoses die de recidiverende LRP episodes kunnen verklaren.

Hoewel het vervolgonderzoek na 4 weken een verschil liet zien tussen LRP patiënten met en zonder een regionaal pijnsyndroom over de door de patiënt gerapporteerde LRP, kan geen verstrekkende conclusie worden verbonden aan dit resultaat; de uitval (44%) na 4 weken is te hoog. De aanwezigheid van de vier regionale pijnsyndromen correleerde bovendien niet met het persisteren van de pijn.

Uit de follow-up na 5 jaar kwamen geen serieuze diagnoses tevoorschijn bij LRP patiënten met een regionaal pijnsyndroom. Ook werd er geen verschil gevonden tussen LRP patiënten met en zonder een regionale pijnsyndroom over het aantal LRP recidieven. De power van het onderzoek is echter te gering voor een valide conclusie. Daarom blijft de vraag of een onderverdeling van LRP in QLPS, GMPS, ICPS en LLPS prognostische betekenis heeft open voor toekomstig onderzoek.

Hoofdstuk 8 onderzoekt het gezamenlijk voorkomen van combinaties van de vier regionale pijnsyndromen. Publikaties over regionale pijnsyndromen behandelen deze meestal apart. De regionale pijnsyndromen komen voor in combinatie bij 15 (25%) LRP patiënten. Dit is veel hoger dan de prevalentie van combinaties zoals die in de literatuur wordt gemeld. Deze bevinding werd niet veroorzaakt door toeval, interobserver variatie, of een lage pijndrempel. Daarom lijkt de conclusie gerechtvaardigd dat regionale pijnsyndromen vaker in combinatie voorkomen dan wordt gedacht.
De betekenis van het voorkomen van combinaties voor de prognose werd onderzocht aan de hand van 3 categorieën, LRP patiënten 1) zonder regionale pijnsyndroom; 2) met een regionale pijn syndroom; en 3) met een combinatie. Uit het vervolgonderzoek na 4 weken en na 5 jaar blijk dat de aanwezigheid van combinaties van regionale pijnsyndromen geen klinische betekenis heeft.

Hoofdstuk 9 bevat een algemene discussie naar aanleiding van de voornaamste conclusies over dit onderzoek. Allereerst wordt bij de prognose stilgestaan. Daarna wordt aandacht besteed aan de klinische betekenis van regionale pijnsyndromen bij aspecifieke LRP in de huisartspraktijk.

In de NHG standaard Lage rugpijn wordt het niet zinvol geacht om diagnoses te onderscheiden binnen aspecifieke LRP, omdat niet is aangetoond dat deze subgroepen een andere prognose hebben of beter op specifieke therapie reageren.

Manuscripts based on the present study

Chapter 2	Njoo KH. Pijnpunten en lage rugpijn. Tijdschr Huisartsgeneesk 1992;9:937-9.
Chapter 3 and 4	Njoo KH, Van der Does E. The occurrence and inter-rater reliability of myofascial trigger points in the Quadratus Lumborum and Gluteus Medius. A prospective study in nonspecific low back pain patients and controls in general practice. Pain 1994;58(3):317-23.
	Njoo KH, Van der Does E. Trigger points in nonspecific low back pain patients: a critical perspective on the diagnostic criteria. Scand J Rheumatol 1992; suppl 94:60 (abstract).
Chapter 3 and 5	Njoo KH, Van der Does E, Stam HJ. Interobserver agreement on Iliac Crest Pain Syndrome in General Practice. J Rheumatol 1995;22:1532-35.

Dankwoord

"How can you possibly be lost, when every road has a final destination?"
(Uit: De dansende man, Onafhankelijk Toneel)

De produktie van een proefschrift is wel eens eerder vergeleken met een wandeling. De weg is geaccidenteerd en kronkelig, de route niet duidelijk aangegeven, en soms liggen er onoverkoombare obstakels op de weg. Gelukkig wandel je niet alleen, onderweg lopen mensen met je mee, voor kortere of langere stukken. Zonder anderen tekort te willen doen, wil ik op deze plaats enkele personen bedanken voor hun gezelschap op deze uiterst boeiende en leerzame wandeling.

Op de eerste plaats wil ik mijn beide promotoren bedanken. Emiel van der Does, je geloof in deze onderneming en je kijk als zeer ervaren huisdokter op dit onderzoek hebben me zeer geïnspireerd. Bewegen is een gezondheidsbevorderende bezigheid, heb je eens geschreven. Henk Stam, het komt me voor alsof je al vanaf het begin betrokken bent geweest bij dit onderzoek. Je plezier in publiceren en je wetenschappelijk inzicht heb ik als zeer stimulerend ervaren.

Aan de start van dit onderzoek hebben velen aan een goede voorbereiding bijgedragen. Met name wil ik prof. A. Cats en prof. B. Dijkmans bedanken voor hun grote bijdrage aan mijn kennis van de reumatologie. Frank van den Ouweland en Gerard Collee wil ik danken voor hun bijdrage aan de opzet en inhoud van dit onderzoek.

Zonder de deelnemende patiënten was dit onderzoek niet mogelijk geweest. De huisartsen C. Boer, I. Chang, A.C. de Jongh, J.W. de Waard, J.J.B. du Pon, J.D. Dijk, J. Dijkstra, W. Neijendorff, J.P. Rosendaal, R. Schoon, E.P.W. Zuidgeest en hun praktijkassistentes, hebben bij de recrutering van deze patiënten een belangrijke rol gespeeld. Verder hebben jullie het 'onderzoeksteam' een gastvrij onderdak geboden in het gezondheidscentrum De Burgh en het gezondheidscentrum De Beverwaard. Het 'onderzoeksteam' bestaande uit de keuze-onderzoekers: M. Heijkoop, P. Meulenbroek, mevr. A. Rensen en M. Schutte, hebben mij veel werk uit handen genomen. Mevr. S. Ramkisoen wil ik bedanken voor haar geduldig speurwerk bij de follow-up na 5 jaar. Het doet mij deugd, dat de meeste van jullie

voor het vak van huisarts hebben gekozen.

Op het Instituut Huisartsgeneeskunde Rotterdam wil ik Frits Bareman bedanken voor zijn heldere en geduldige uitleg over statistische analyses en de manier om mijn resultaten te interpreteren. Mijn 'stok achter de deur' Arthur Bohnen wil ik bedanken voor zijn bijdrage aan hoofdstuk 7 en 8. Vele, ook niet hier genoemde medewerkers van het Instituut, hebben een belangrijke bijdrage geleverd aan dit proefschrift door hun collegialiteit en persoonlijke belangstelling.

De afdeling Standaarden-Ontwikkeling van het NHG wil ik hierbij bedanken voor hun flexibiliteit t.a.v. het regelen van perioden met schrijfverlof binnen mijn toenmalige aanstelling.

I would like to thank Laraine Visser and Susy Heijblom for reviewing the English text.

Familie en vrienden hebben vooral aan mijn psychisch welbevinden bijgedragen. Hun niet aflatende vragen naar wanneer mijn 'scriptie' klaar zou zijn, plaatst het belang van dit proefschrift in een speciaal kader.

Peter, gebruikelijk is hier een zinsnede hoe moeilijk het was en hoe weinig aandacht er overbleef. Gelukkig zijn we leuke dingen blijven doen. Zelfs het 'schrijven' lijken we alternerend te doen. Onze discussies over De Wetenschap heeft me met beide benen op de grond gehouden.

Khing Njoo, 12 juni 1996.

Curriculum Vitae

Khing Hua Njoo werd op 27 maart 1958 te Bandung geboren. In 1965 verhuisde het gezin naar Nederland. Zij behaalde in 1976 het gymnasium-β aan het Christelijk Lyceum Delft. In datzelfde jaar begon zij haar studie geneeskunde aan de Erasmus Universiteit te Rotterdam. Na het behalen van haar artsexamen in december 1983 verrichtte zij literatuuronderzoek voor de vakgroep huisartsgeneeskunde Leiden. In 1985 volgde zij de eenjarige opleiding tot huisarts.

Van 1986 tot 1988 was zij als onderzoeksassistent werkzaam aan het instituut huisartsgeneeskunde van de Erasmus Universiteit te Rotterdam. In 1988 startte zij in het kader van het SGO-Reuma project het in dit proefschrift beschreven onderzoek. Daarnaast was zij als huisarts werkzaam in het gezondheidscentrum Krimpen a/d IJssel, later in Rotterdam-Zuid. Tussen 1990 en 1992 werkte zij mee aan het ontwikkelen van de ICPC-thesaurus zoekmodule. Van 1991 tot en met 1995 was zij datastaflid voor de afdeling Standaardenontwikkeling van het Nederlands Huisartsen Genootschap.

Momenteel werkt zij één dag in de week als huisarts in het Oude Noorden van Rotterdam. Verder is zij vanaf 1995 als coördinator van het huisartsen registratienet ROHAPRO werkzaam aan het instituut huisartsgeneeskunde en de GGD-Rotterdam. Daarnaast is zij projectsecretaris van een Transparant-project elektronische communicatie tussen huisarts en RIAGG. Incidenteel geeft zij voor de Landelijke Huisartsen Vereniging trainingen in het gebruik van de ICPC in de dagelijkse huisartspraktijk.

List of abbreviations in alphabetical order:

d	presence of an unilateral dummy point
D	presence of bilateral dummy point
FS	Fibromyalgia Syndrome
GMPS	Gluteus Medius Pain Syndrome
GMPS+	GMPS present at initial examination (t=0)
GMPS-	GMPS not present at initial examination (t=0)
GP	general practitioner
ICPS	Iliac Crest Pain Syndrome
ICPS+	ICPS present at initial examination (t=0)
ICPS-	ICPS not present at initial examination (t=0)
LBP	Low Back Pain
LLPS	Long Ligament Pain Syndrome
LLPS+	LLPS present at initial examination (t=0)
LLPS-	LLPS not present at initial examination (t=0)
MFPS	Myofascial Pain Syndrome
n or no.	number of patients
PSIS	posterior superior iliac spine
QLPS	Quadratus Lumborum Pain Syndrome
QLPS+	QLPS present at initial examination (t=0)
QLPS-	QLPS not present at initial examination (t=0)
I	first observer
II	second observer
t=0	at initial examination
t=4	at 4 week follow-up