Winanda Wolfs[1], Zbigniew Zylicz[2]
[1]Department of General Practise, University of Maastricht, The Netherlands
[2]Dove House Hospice, Hull, United Kingdom

Myofascial and nerve compression pain as an important factor of uncontrolled pain in advanced disease

Abstract

Background. To examine how often patients, with advanced disease and in need of improvement of pain control, present with trigger points due to myofascial pain and nerve compression and if these trigger points can be related to the pain spontaneously reported by patients.
Material and methods. Thirty three patients with advanced disease referred to different hospice services for pain control participated in this study, which involved a pain history interview, palpation of areas prone to development of trigger points and measurement of pressure pain thresholds (PPT) of these areas.
Results. On palpation, trigger points were found in 94% of the patients. In 67% of them trigger points could be matched with the pains mentioned during the pain history interview. A significant correlation was found between the low pressure pain thresholds of the control areas and those of the investigated areas ($r = 0.83$, $p < 0.01$). An inverse relation was found between the number of trigger points and the mean pressure pain threshold of the investigated areas ($r = –0.63$, $p = 0.01$).
Conclusion. Many patients with unrelieved pain in advanced disease may present with trigger points. The origin can be peripheral. However, when multiple, a central cause such as spinal cord sensitisation should be considered. Attention to trigger points and to their treatment has the potential for further improvement of pain control.
Key words: myofascial pain, nerve compression, trigger point, advanced disease

Adv. Pall. Med. 2009; 8, 1: 13–22

Introduction

Despite considerable developments in pain control in advanced disease, actual prevalence of pain is not changing. This is true for cancer, and probably to an even greater extend for other chronic diseases [1]. Several types of pain, usually less responsive to opioids, are still difficult to treat. Patients tend to complain of many pains at the same time [2]. In a group of 111 cancer patients 370 pains were found with an average of 3 pains per patient [3]. After treating the patients for 4 weeks this number dropped to 1.5 pains per patient. Pains unrelated to cancer still contributed considerably to the overall pain. Several pains unrelated to cancer were identified and described in more detail [4]. Myofascial but also nerve compression pain can be related to cachexia, progressive debility and lack of movement. Strain and muscle overuse can also be causes. These types of pain are certainly not

Address for correspondence: Zbigniew Zylicz
Dove House Hospice, Chamberlain Road, HU8 8DH, United Kingdom
e-mail: b.zylicz@dovehouse.org.uk
Advances in Palliative Medicine 2009, 8, 13–22

unique to cancer and may be encountered in nearly all chronic and non-cancer diseases [5–8].

There is also a suggestion that malnutrition and other co-morbidities may increase the vulnerability of the nerve to damage [9–11]. Muscle weakness and atrophy may result in joint hypermobility and increased traction on the nerves. This type of pain is suggested in case of suprascapular nerve entrapment [12].

Until now little attention has been given to myofascial and nerve compression pain. As they tend to be less responsive to opioids, the doses of these drugs can be high and not infrequently toxic [4]. Recently we described several clinical situations in which nerve compression pain played an important role (in press). Once interested in the subject we suddenly started to see more and more patients with these afflictions and wondered how important myofascial and nerve compression pain is when the pain seems to respond insufficiently to the most basic pharmacological approaches. To answer this question we investigated a population of patients, most of them with cancer, referred to us for improvement of pain control. The aim of this study was to find out how often these kinds of patients present with trigger points. If so, can these trigger points be related to the pain spontaneously reported by the patients. We also wanted to objectively measure the pressure pain thresholds at the trigger points.

Material and methods

Patients

The study protocol was approved by the ethical committee of the Hospice Board and was performed in a period of two months. The patients were referred to the different hospice services for improvement of their pain control. During this period 80 patients were admitted to the bedded unit, 32 of them for pain control. Of the 62 patients attending the day therapy at that time, at least 27 had unrelieved pain symptoms. Nineteen patients were referred to the outpatient clinic, all for pain control. Thus the potential population of patients consisted of 78 patients. Thirty three patients satisfied all inclusion criteria and were asked to participate in the study. The inclusion criteria were: age > 18 years, referral for pain control, prognosis longer than 4 weeks, Karnofsky performance score > 50%, able to communicate in English and capable to give informed consent. Patients treated with steroid injections for myofascial and/or nerve compression pain in the past 2 months were excluded. Verbal and written information about the procedure was given and all 33 patients gave their written consent. None of the participating patients finished the study prematurely, although they were aware they could withdraw at any moment.

Table 1. Patient demographics and characteristics

	Subjects (n = 33)
Age (years; mean ± SD)	73 ± 11
Gender	
Female	18 (55%)
Male	15 (45%)
Diagnosis	
Multiple sclerosis	2 (6%)
Parkinson's disease	1 (3%)
COPD	2 (6%)
Diabetes mellitus	1 (3%)
Malignancy	27 (82%)
Head-neck region	1
Heamatolo-oncology	3
Breast	3
Lung	6
Urologic	6
Gastrointestional	7
Unknown primary	1

SD — standard deviation; COPD — chronic obstructive pulmonary disease

Patient's demographics and diagnoses are presented in Table 1. Twenty seven patients (82%) suffered from different types of malignancy. The cancer was disseminated in 17 of them (63%). All but 8 patients suffered from different co-morbidities. Seven patients suffered from one co-morbidity, 8 patients from 2, 7 patients from three and 3 patients had 4 or more co-morbidities. These co-morbidities consisted of inactive malignancies (5), diabetes mellitus (3), respiratory (6), cerebrovascular (5) and gastrointestinal disease (7). However, most frequent were cardiovascular (16) and musculoskeletal disease like arthritis and spondylosis (14).

Algometry

To measure the pressure pain threshold (PPT) a mechanical force gauge or algometer, the Force dialTM FDK/FDN 20 (Wagner Instruments, Greenwich, Connecticut, USA), was used. The gauge has a scale marked from 1 to 10 kg/cm^2, in increments of 0.1 kg/cm [13]. Readings were obtained by applying a gradually increasing force with the algometer. The instrument was new and calibrated by the manufacturer.

Study protocol

The pain history interview was based on a protocol corroborated by Oldenmenger et al [14]. The subjects were asked to mark the areas of ongoing pain on a body chart and estimated their overall resting pain intensity using a numeric rating scale

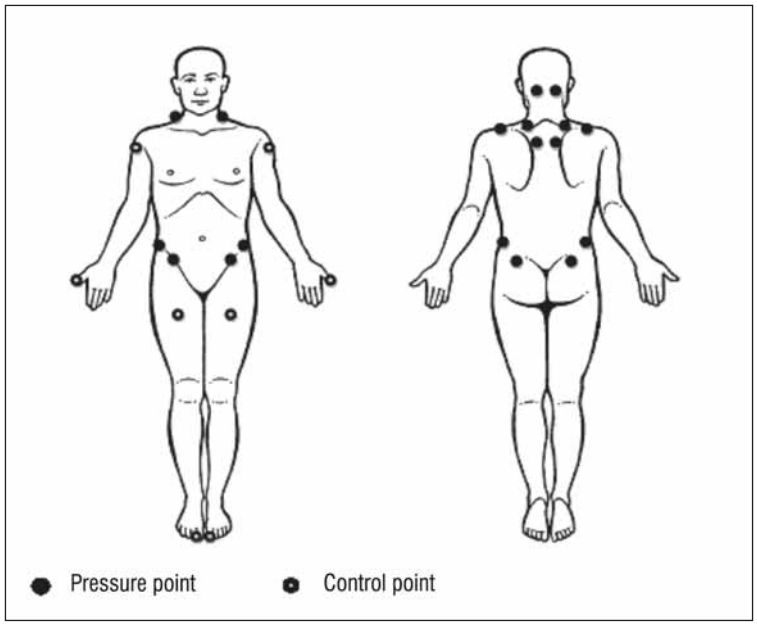

Figure 1. The areas investigated for the presence of trigger points

(NRS) where 0 means no pain at all and 10 the worst pain possible. Then the patients were asked about pain characteristics and the interference with daily life activities. The effect of analgesic medication and their adverse effects were also part of this interview.

The patients were then subjected to a physical examination. Thirteen bilateral areas of the body were investigated for the presence of trigger points. Nine areas were known to be frequently affected with myofascial (M), nerve compression (N) or combination (C) trigger points. Four areas were selected as control areas as they were known not to be related to trigger points. This method was derived from the literature [13, 15–17].

Areas investigated (see Figure 1):

— occiput: bilateral, at the suboccipital muscle insertions (M);
— upper trapezius: bilateral, angle of the neck (M);
— lower cervical: bilateral, at the anterior aspects of the intertransverse spaces at C5–C7 [M];
— supraspinatus: bilateral, suprascapular notch (C);
— trapezius: bilateral, mid back along scapula (M);
— subcostal nerve Th12: bilateral, ± 9 cm from anterior superior iliac spine (N);
— lateral cutaneous branch Iliohypogastric nerve: bilateral, ± 13 cm from anterior superior iliac spine (N);
— gluteal: bilateral, in upper outer quadrants of buttocks in anterior fold of muscle (M);
— lateral femoral cutaneous nerve: bilateral, ±1 cm inferior of anterior superior iliac spine (N).

Control areas:

— deltoid: bilateral, middle portion;
— thumbnail: bilateral;
— quadriceps femoris: bilateral, mid anterior;
— big toenail: bilateral.

To avoid patient and investigator related bias the sequence in which the areas were investigated, was randomised by a computer program that was especially designed for this study.

Due to poor mobility of most patients, the physical examination was done in a sitting position. Minimal criteria, local tenderness and/or nodules on palpation, were used to diagnose a trigger point [18–20]. Following palpation, the pressure pain threshold (PPT) was measured using an algometer. The shaft of the instrument was placed vertical to the examined surface. The patient was instructed to say "yes" when the sensation of pressure changed to pain. Pressure was increased at approximately 1 kg/cm^2/s. The registered pressure representing PPT was then recorded.

The interview and the physical examination were taken in one session and by the same investigator (WW). To gain experience with the method and with the protocol a pilot study on 6 consecutive patients was performed. The results of the pilot are not included in this report.

Statistical analysis

The statistical analysis was done using Excel for Windows and the Statistical Package for the Social Sciences (SPSS). Descriptive statistics were sum-

Table 2. Pain localisation

	Head/neck region (n = 1)	Heamotolo-oncology (n = 3)	Breast (n = 3)	Lung (n = 6)	Urologic (n = 6)	Gastroin-testinal (n = 7)	Unknown primary (n = 1)	Multiple sclerosis (n = 2)	Parkinson's disease (n = 1)	COPD (n = 2)	Diabetes mellitus (n = 3)	Total (n = 3)
Head, face, mouth	1	0	1	2	2	3	0	1	0	0	0	10 (13.6)
Cervical region	0	1	0	1	1	3	0	0	0	1	1	8 (10.8)
Shoulder region	1	0	2	2	0	2	1	0	1	1	1	11 (14.9)
Upper limbs	0	1	0	1	0	2	0	1	0	0	0	5 (6.7)
Thoracic region	0	1	0	4	1	2	0	0	0	0	0	8 (10.8)
Abdominal region	0	1	0	0	1	2	0	1	0	0	0	5 (6.7)
Lower back, lumbar spine, coccyx	0	1	2	1	2	4	0	1	0	0	0	11 (14.9)
Lower limbs	0	0	0	2	3	2	0	2	0	1	1	11 (14.9)
Pelvic region	0	0	1	1	0	3	0	0	0	0	0	5 (6.7)
Total	2	5	6	14	10	23	1	6	1	3	3	74 (100)

COPD — chronic obstructive pulmonary disease

marised using mean, median values and standard deviation. Correlation analysis was undertaken with the Pearson correlation test. The p values ≤ 0.05 were accepted as significant.

Results

Pain history

The body charts revealed 74 anatomically distinct locations. They were grouped into nine different categories (see Table 2). The most frequent pain locations were shoulders, lower back, sacrum and lower limbs. Fourteen patients (42%) presented with one, 11 (33%) with two, 4 (12%) with three and 4 (12%) with four or more painful areas. Two patients initially complained of "pain everywhere", but managed to localise the most painful areas of the body. Twelve patients (36%) suffered pain for more than a year. Three (10%) had pain for 7–12 months. Eight patients (24%) had been having pain for 1 to 6 months and 10 (30%) for less than a month.

In 16 patients (48%) the pain onset was gradual while more acute onset was recorded in 14 patients (42%). Three patients (10%) could not answer the question. Nineteen patients (58%) experienced continuous pain, with or without fluctuations. Ten patients (30%) experienced pain in attacks without pain in between. In 4 patients (12%) the pain course could not be determined in such a detail.

The average pain intensity score (NRS-score) was 4 (SD ± 3). The median was 5. Twenty patients (63%) described their pain as moderate to severe which corresponded with a NRS ≥ 4 (see Table 3). Five patients (15%) described their pain as unbearable. Nineteen patients (58%) indicated that the pain was bearable or even better than usual. Movement was aggravating pain in 21 of patients (63%).

Pain medication

Four patients were not taking pain medication on the day of assessment. Sixteen patients (55%) were taking combinations of various analgesics.

Paracetamol was used by 17 patients (52%) and 3 patients (9%) were using a combination of co-

Table 3. Overall pain intensity

NRS score	Number of patients
0 — no pain	8
1–3 — mild pain	4
4–6 — moderate pain	14
7–10 — severe pain	6
Missing value	1

NRS — numerical rating scale

Table 4. Patient demographics and characteristics

Drug name	Number of patients (%)
Controlled release	
Fentanyl	12 (60%)
Morphine sulphate	5 (25%)
Oxycodone	1 (5%)
Immediate release	
Morphine sulphate	12 (60%)
Oxycodone	2 (10%)
Methadone	1 (5%)

deine (30 mg)/paracetamol (500 mg). Two other patients (6%) were only using codeine. Two patients (6%) were taking tramadol. NSAIDs were prescribed in 4 patients (12%) and antidepressants (amitriptyline, citalopram) in 6 cases (18%). Four patients (12%) were taking gabapentin. Strong opioid analgesics were used by 20 patients (61%) (see Table 4).

Two patients were using ketamine next to opioid analgesics. One patient had just started on a syringe driver with low dose diamorphine for breathlessness.

Of the 29 patients (88%) using pain medication, 18 patients (62%) judged it to be sufficient. Eleven patients (38%) stated that the prescribed analgesics were unsatisfactory.

A dry mouth was the adverse effect most frequently reported (37%). Six patients (18%) complained of dizziness and 5 (15%) of unrelieved constipation. Other adverse effects were headache, confusion, drowsiness, itch, nausea and stomach-ache.

Physical examination

Missing values

In one patient it was not possible to assess all the pressure points because of reduced mobility. Another patient did not want to have the area of the iliac crest examined, because of "painful swelling" which she did not mention during the interview. In seven patients it was impossible to obtain control values of PPT from the toe nails because of ingrown toenails and loss of sensitivity due to diabetic neuropathy or peripheral vascular disease.

Palpation

Trigger points on palpation were found in 31 patients (94%). The histogram of trigger points is presented in Figure 2. The median number of trigger points per patient was 6.

The trigger point frequency per area is presented in Table 5. In all patients, palpation of the control areas did not bring any trigger points to light.

In 22 patients (67%), trigger points could be matched with the pain mentioned during the interview according to Table 6. This matching was carefully distilled out of different specific sources [18, 21–23]. In 9 subjects (27%), the presence of trigger points was unrelated to the pain identified by the patient prior to physical examination (see Figure 2).

Although 3 patients (9%) claimed not to have pain on assessment, only one of them did not have trigger points.

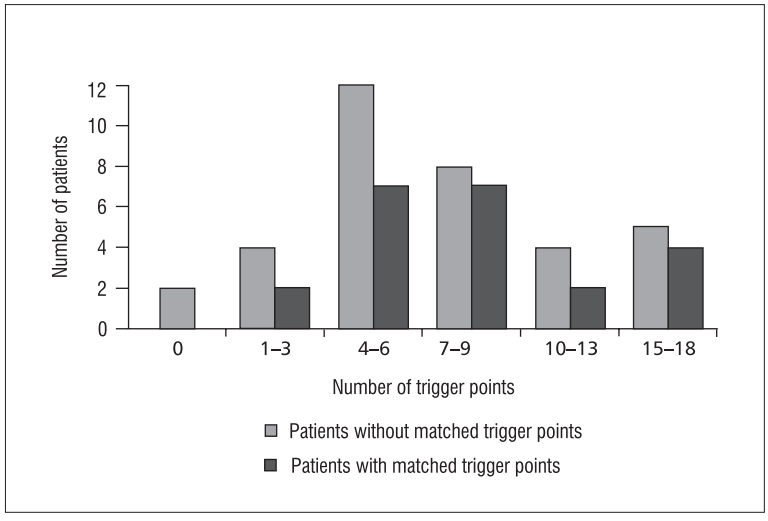

Figure 2. The amount of trigger points

Table 5. Number of trigger points per area

Pressure point	No trigger point present (n%)	Trigger point present (n%)
Occiput L	31 (94)	2 (6)
Occiput R	30 (91)	3 (9)
Upper trapezius L	19 (58)	14 (42)
Upper trapezius R*	15 (45)	18 (55)
Low cervical L*	10 (30)	23 (70)
Low cervical R*	12 (36)	21 (64)
Supraspinatus L	20 (61)	13 (39)
Supraspinatus R	19 (58)	14 (42)
Trapezius L*	16 (48)	17 (52)
Trapezuis R	21 (64)	12 (36)
Subcostal nerve th 12 L (n = 32)	23 (72)	9 (28)
Subcostal nerve th12 R	23 (70)	10 (30)
Iliohypogastric nerve L (n = 31)	19 (61)	12 (39)
Iliohypogastric nerve R (n = 32)	22 (69)	10 (31)
Gluteal L (n = 32)	18 (56)	14 (44)
Gluteal R (n = 32)	19 (59)	13 (41)
Lateral femoral cutaneous nerve L	24 (73)	9 (27)
Lateral femoral cutaneous nerve R	24 (73)	9 (27)

* — four most frequent present trigger points

Table 6. Pain pattern per trigger point

Trigger point	Pain pattern
Occiput	Torticollis, headache, shoulder, neck
Upper trapezius	Shoulder, headache (temporal region), neck
Low cervical	Headache, shoulder, neck
Supraspinatus	Shoulder, neck, upper arm
Trapezius	Mid back, neck, shoulder
Subcostal nerve th12	Abdomen, hip, leg
Iliohypogastric nerve	Abdomen, hip, leg
Gluteal	Low back
Lateral femoral cutaneous nerve	Groin, hip

Pressure pain threshold

In 8 patients (24%), PPT measurement of specific trigger points was less than 1 kg/cm^2. Because of inaccuracy of the algometer at this level, these readings were recorded as 0 kg/cm^2.

For each patient the mean PPT of the investigated areas was calculated. It varied from 1.0 to 7.4 kg/cm^2 (median 3.2 kg/cm^2). For the control points, the values varied from 1.7 to 8.9 kg/cm^2 (median 4.3 kg/cm^2). Low PPT values in the control areas were correlating with low values for the investigated areas. A strong and significant correlation was found ($r = 0.83$, $p < 0.01$) (see Figure 3). The inter-individual variability of the PPT was high. There was no correlation between the NRS-scores and the mean PPT of the investigated areas. A significant inverse relation was found between the number of the trigger points and the mean PPT (see Figure 4; $r = –0.63$, $p = 0.01$).

Discussion

The study population was very much comparable to populations studied previously [2, 3].

However, our study was performed on all patients referred for pain control, including those without cancer. Interestingly, in the first study done by Twycross and Fairfield 39% of patients had one or more pains unrelated to cancer or treatment; the most common of these was myofascial pain (12%) [2]. In the second study done by the same group and with the same method this latter category was practically absent (2%) [3]. The authors explained that the consultant involved in the first study took a

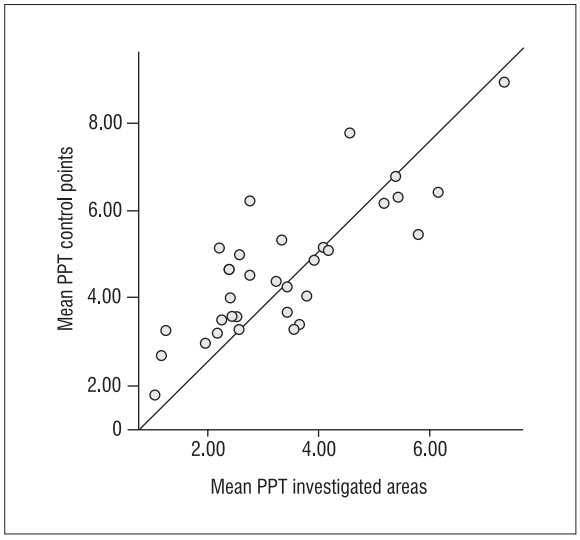

Figure 3. Relationship between the mean pressure pain threshold of the control points and the investigated areas. PPT — pressure pain treshold [kg/cm^3]

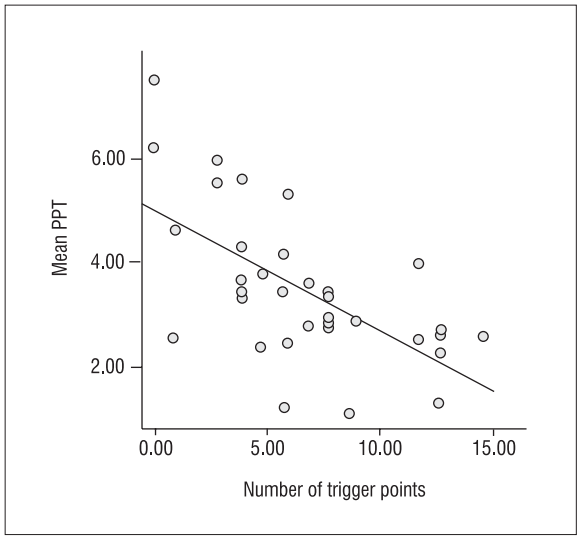

Figure 4. Relation between the number of trigger points and the mean pressure pain threshold of the investigated areas. PPT — pressure pain treshold [kg/cm^3]

special interest in myofascial pain. This suggests that the method used in these two studies was not specific for myofascial pain and could lead to misinterpretation. This was the reason why in our study the pain interview was supplemented by specific physical examination.

The main finding of this study is that in the majority of patients referred for improvement of pain control, both myofascial as nerve compression trigger points could be found. In two thirds of the patients the trigger points could be matched with the painful areas indicated by the patients. We do not know how specific this matching is. For some pain syndromes, such a matching can be straight forward. For example pain in the fronto-lateral part of the thigh can be matched with the trigger point suggesting compression of the lateral femoral cutaneous nerve (meralgia paraesthetica) [24]. Pain in the shoulder can frequently be matched with a positive trigger point above the suprascapular notch [25]. This suggests that in some cases treatment of the trigger points can potentially improve overall pain control. However, a multitude of trigger points in patients with advanced disease and frequent pains from bone and visceral tumour invasion, would support the idea that visceral pains may also be accompanied by trigger points [26, 27]. The convergence-projection mechanism appears to be consistently present in visceral pain pathways, explaining how visceral or other deep tissue pain can activate a trigger point [28]. It is unknown whether treatment of trigger points resulting from visceral or deep tissue pain will result in overall pain decrease. This relationship seems to be present in case of localised and peripheral origins of pain [29].

Another finding of this study is that low PPT values for the control points are usually accompanied by lower threshold values for the investigated areas ($r = 0.83$, $p < 0.01$), which suggest individual changes or predisposition to pain sensitivity. Patients with more intensive pain may suffer from generalised sensitisation and lowered pain thresholds which in some places may turn into in physical pain accompanied by trigger points.

There is an inverse relationship between the number of trigger points found and the mean PPT of the investigated areas ($r = -0.63$, $p = 0.01$). This suggests that in patients with few trigger points a regional or peripheral cause of pain can be present, as for example entrapment of one or two single nerves. In these patients local treatment of the trigger points with pharmacological or non-pharmacological methods could result in full pain control. In patients with a multitude of trigger points and low PPT, it is possible that there is a central cause such as spinal cord sensitisation [30]. It is reported that patients with chronic pain may have generalised hyperalgesia [31]. Another cause can be opioid-induced hyperalgesia [32]. Patients who are using opioids for pain relief, somewhat paradoxically, may become more sensitive to pain as a direct result of the opioid therapy [33].

This study has several limitations. The number of patients is small and several interesting questions such as the differences in trigger points be-

tween the left and the right part of the body, as well as between males and females could not be answered.

Algometry is described elaborately for myofascial trigger points, but not for nerve compression trigger points. In fact, the algometer was difficult to use at the bone edges of the iliac crest. Problems using the algometer for PPT measurements of nerve compression trigger points were mentioned before by Sterling et al [34]. Repeatability and reliability of the method were not assessed separately so only general conclusions could be drawn.

One important trigger point described by Maigne et al., localised on the posterior iliac crest, 7 cm from the median line, the place where the superior cluneal nerve crosses the iliac crest, was unfortunately not included in our study [35]. Only later we realised how important this trigger point is and how frequently it is encountered in practice. Adding this point to the list tested by us would greatly increase the strength of our conclusion.

Conclusion

Many patients with unrelieved pain in advanced disease may present with trigger points. Some of the trigger points probably originate because of regional or peripheral problems like muscle overload or nerve entrapment. However others, especially when multiple, may be due to more general causes such as spinal cord sensitisation and hence not amenable to regional measures. Pain by itself, but also opioids, may increase this sensitisation and result in increased sensitivity to pain and decreased pressure pain thresholds.

Attention to the trigger points and to their treatment has the potential of further improvement of pain control, with the pay-off of lower doses of opioids and fewer adverse effects.

References

1. van den Beuken-van Everdingen M.H., de Rijke J.M., Kessels A.G., Schouten H.C., van Kleef M., Patijn J. Prevalence of pain in patients with cancer: a systematic review of the past 40 years. Ann. Oncol. 2007; 18: 1437–1449.
2. Twycross R.G., Fairfield S. Pain in far-advanced cancer. Pain 1982; 14: 303–310.
3. Twycross R., Harcourt J., Bergl S. A survey of pain in patients with advanced cancer. J. Pain Symptom Manage 1996; 12: 273–282.
4. Marcus N.J. Pain in cancer patients unrelated to the cancer or treatment. Cancer Invest. 2005; 23: 84–93.
5. Kerns R.D., Kassirer M., Otis J. Pain in multiple sclerosis: a biopsychosocial perspective. J. Rehabil. Res. Dev. 2002; 39: 225–232.
6. Quinn N.P., Koller W.C., Lang A.E., Marsden C.D. Painful Parkinson's disease. Lancet 1986; 1: 1366–1369.
7. Nordgren L., Sorensen S. Symptoms experienced in the last six months of life in patients with end-stage heart failure. Eur. J. Cardiovasc. Nurs. 2003; 2: 213–217.
8. Skootsky S.A., Jaeger B., Oye R.K. Prevalence of myofascial pain in general internal medicine practice. West. J. Med. 1989; 151: 157–160.
9. Antoine J.C., Camdessanche J.P. Peripheral nervous system involvement in patients with cancer. Lancet Neurol. 2007; 6: 75–86.
10. Vinik A., Mehrabyan A., Colen L., Boulton A. Focal entrapment neuropathies in diabetes. Diabetes Care 2004; 27: 1783—1788.
11. Koehler P.J., Buscher M., Rozeman C.A., Leffers P., Twijnstra A. Peroneal nerve neuropathy in cancer patients: a paraneoplastic syndrome? J. Neurol. 1997; 244: 328––332.
12. Penn J., Zylicz Z. Could shoulder pain be suprascapular nerve entrapment? Eur. J. Pall. Care 2006; 13: 98–100.
13. Fischer A.A. Pressure algometry over normal muscles. Standard values, validity and reproducibility of pressure threshold. Pain 1987; 30: 115–126.
14. Oldenmenger W.H., Stronks, D.L. Naar een landelijke, uniforme verpleegkundige pijnanamnese [Dutch]. Ned Tijdsch Pijn Pijnbestrijding 2006; 25: 6–12.
15. Reeves J.L., Jaeger B., Graff-Radford S.B. Reliability of the pressure algometer as a measure of myofascial trigger point sensitivity. Pain 1986; 24: 313–321.
16. Garcia E., Godoy-Izquierdo D., Godoy J.F., Perez M., Lopez-Chicheri I. Gender differences in pressure pain threshold in a repeated measures assessment. Psychol Health Med. 2007; 12: 567–579.
17. Alvarez D.J., Rockwell P.G. Trigger points: diagnosis and management. Am. Fam. Physician 2002; 65: 653–660.
18. Travell J.G., Simons D.G. Myofascial Pain and Dysfunction. The Trigger Point Manual. 1st ed. Williams & Willkins, Baltimore 1983.
19. Sciotti V.M., Mittak V.L., DiMarco L. et al. Clinical precision of myofascial trigger point location in the trapezius muscle. Pain 2001; 93: 259–266.
20. Tough E.A., White A.R., Richards S., Campbell J. Variability of criteria used to diagnose myofascial trigger point pain syndrome. Evidence from a review of the literature. Clin J. Pain 2007; 23: 278–286.
21. Fernandez-de-las-Penas C., Alonso-Blanco C., Cuadrado M.L., Gerwin R.D., Pareja J.A. Trigger points in the suboccipital muscles and forward head posture in tension-type headache. Headache 2006; 46: 454–460.
22. Melville K., Schultz E.A., Dougherty J.M. Ilionguinal-iliohypogastric nerve entrapment. Ann. Emerg. Med. 1990; 19: 925–929.
23. Haim A., Pritsch T., Ben-Galim P., Dekel S. Meralgia paresthetica: A retrospective analysis of 79 patients evaluated and treated according to a standard algorithm. Acta Orthop. 2006; 77: 482–486.
24. Grossman M.G., Ducey S.A., Nadler S.S., Levy A.S. Meralgia paresthetica: diagnosis and treatment. J. Am. Acad. Orthop. Surg. 2001; 9: 336–344.
25. Zylicz Z., Haaijman J. Suprascapular nerve entrapment: a neglected cause of shoulder pain in cachectic patients? J. Pain Symptom Manage 2000; 20: 315–317.
26. Cimen A., Celik M., Erdine S. Myofascial pain syndrome in the differential diagnosis of chronic abdominal pain. Agri. 2004; 16: 45–47.
27. Doggweiler-Wiygul R. Urologic myofascial pain syndromes. Curr. Pain Headache Rep. 2004; 8: 445–451.
28. Simons D.G. Myofascial pain syndromes: where are we? Where are we going? Arch. Phys. Med. Rehabil. 1988; 69: 207–212.

29. Nelemans PJ, de Bie RA, de Vet, H.C.W. Injection therapy for subacute and chronic benign low back pain. Spine 2001; 26: 501–515.
30. D'Mello R., Dickenson A.H. Spinal cord mechanisms of pain. Br. J. Anaesth 2008.
31. Giesbrecht R.J., Battie M.C. A comparison of pressure pain detection thresholds in people with chronic low back pain and volunteers without pain. Phys. Ther. 2005; 85: 1085–1092.
32. Angst M.S., Clark J.D. Opioid-induced hyperalgesia: a qualitative systematic review. Anesthesiology 2006; 104: 570–587.
33. Zylicz Z., Twycross R. Opioid-induced hyperalgesia may be more frequent than previously thought. J. Clin. Oncol. 2008; 26: 1564.
34. Sterling M., Treleaven J., Edwards S., Jull G. Pressure pain thresholds of upper limb peripheral nerve trunks in asymptomatic subjects. Physiother. Res. Int. 2000; 5: 220––229.
35. Maigne J.Y., Lazareth J.P., Guerin Surville H., Maigne R. The lateral cutaneous branches of the dorsal rami of the thoraco-lumbar junction. An anatomical study on 37 dissections. Surg. Radiol. Anat. 1989; 11: 289–293.