Professional perspectives

Survey of current sedation practices for interventional pain procedures: UK Pain Specialists

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Introduction

Aim

The aim of this survey was to obtain an understanding of the current sedation practices among UK-based Pain Specialists. This is the first published, nationwide survey looking into sedation practices among Pain Specialists in the United Kingdom and the first discussion within existing literature guidelines.

Methods

A national survey of Pain Specialists in the United Kingdom was carried out using an online questionnaire. Respondents were identified using the UK Pain Specialists' network group, which has more than 450 members.

The survey contained 10 questions and pertained to current practices by Pain Specialists with regard to sedation during any interventional pain procedure. The survey contained a combination of free text responses and discrete options for various questions. The survey was accessed via an online webpage, with all the responses anonymised. The investigators only had access to the collated final data, with no demographic or geographic data about the respondents collected. This was to reduce responder bias.

Table 1 outlines the 10 questions that were used.

The aim of this study was to obtain results from 100 clinicians around the United Kingdom. The responses were collated using a web-based database and transferred to Microsoft Excel 360 for analysis and drawing graphs.

Results

A total of 100 responses were collected from June 2018 to July 2018. The respondents included 94 Consultants and 6 senior trainees undergoing Pain Fellowships. The results for each question are summarised (see Table 1).

Table 1. Summary of the 10 questions used in the survey.

Question no.	Question
1	Do you discuss sedation when consenting patients for procedures?
2	Do you provide sedation for patients undergoing pain procedures?
3	Who provides the sedation?
4	What drugs are used for sedation?
5	Is there an anaesthetic machine in the procedure room?
6	What monitoring is available during sedation?
7	Is supplemental oxygen provided during sedation?
8	Do you believe sedation improves the outcomes of pain procedures?
9	Which cases is sedation offered for?
10	What grade is the person performing the interventional pain procedure?

Figure 1. The number of respondents who provide sedation for interventional pain procedures.

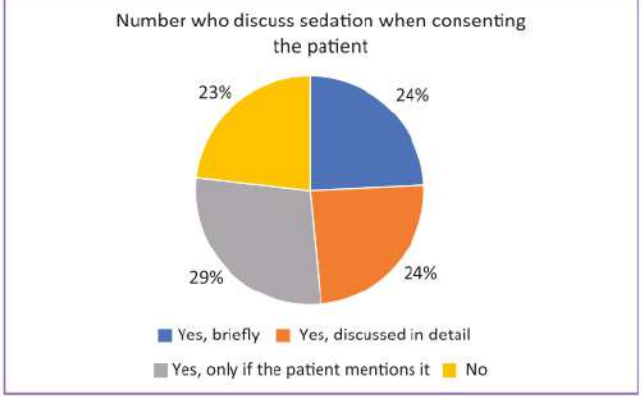
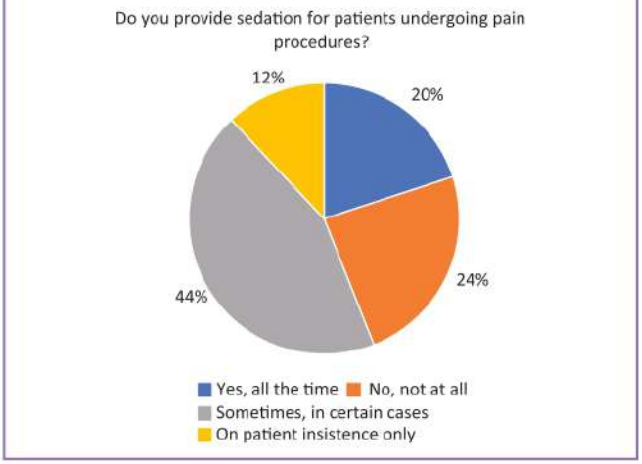


Figure 2. What proportion of responders provide sedation for patients undergoing interventional pain procedures.



Question 1: the number of respondents who consented for sedation

In total 98% of respondents answered the question about consenting for sedation while discussing the interventional pain procedure.

Only a quarter of respondents discussed sedation in detail as a matter of routine. Roughly a half of Pain Physicians either discussed sedation briefly or only if the patient mentioned it, and the remaining quarter did not discuss sedation at all (see Figure 1).

Question 2: the number of respondents who provide sedation for pain procedures

In total, 24% of respondents did not provide sedation for their interventional procedures. This corresponds with the 24% who did not discuss sedation with their patients prior to the procedure.

Interestingly, 12% of responders only provided sedation if their patients insisted on it. Almost half of the clinicians were flexible with sedation, providing it for some but not all interventional procedures. Conversely, 20% provided sedation for all interventional pain procedures (see Figure 2).

Question 3: who provides the sedation?

When sedation was provided to patients, there seemed to be a range of people providing it. The majority (40%) appeared to be given by the Pain Specialists who were also performing the procedure. Of the other people providing sedation, Consultant Anaesthetists (17%) and Operating department practitioner (ODP)/Anaesthetic nurses (14%) provided the remainder, with Trainee Anaesthetists only giving 6% of sedation.

Question 4: what drugs are used for sedation?

A variety of drug combinations were described for sedation during interventional procedures. It should be noted that almost half of respondents used other combinations of drugs. The other predominant combinations used were propofol or midazolam with fentanyl followed by midazolam only (see Figure 3).

Question 5: presence of an anaesthetic machine in the procedure room

A quarter of procedure rooms did not contain an anaesthetic machine. The remaining 75% reported an anaesthetic machine in their procedure room.

Question 6: monitoring for sedation

It was noted that only half of the patients had a full complement of saturations (SpO_2), blood pressure (BP) and electrocardiography (ECG) applied to them while a quarter of the patients had either SpO_2 or BP monitored. There were no data regarding the monitoring of end-tidal CO_2 ($EtCO_2$; see Figure 4).

Question 7: provision of supplemental oxygen with sedation

Only half of the patients had supplemental oxygen routinely applied if they were undergoing sedation. A total of 2% of patients did not have oxygen applied at all. A further 28% were given oxygen only if they desaturated.

Figure 3. The choice of sedative drugs used during interventional pain procedures.

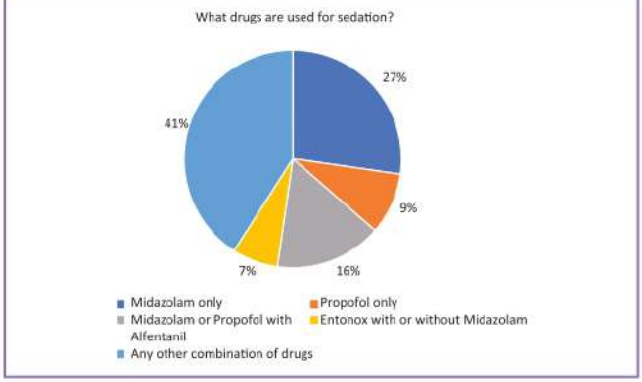
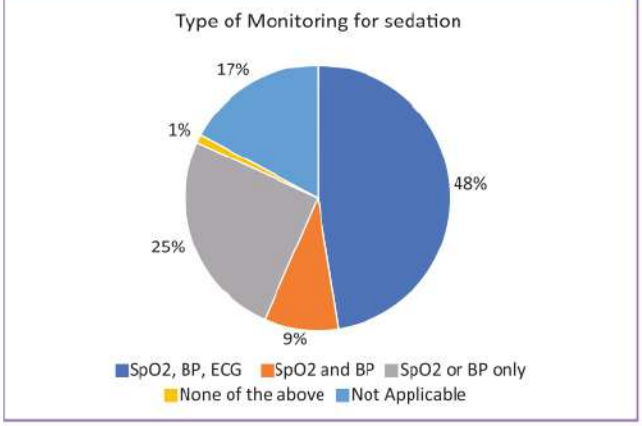


Figure 4. The types of monitoring applied to patients who have undergone sedation.



Question 8: do Pain Specialists believe sedation improves outcome?

Only 14% of respondents felt that sedation improved the outcome of pain interventions. Over half of the respondents did not believe that sedation improved the outcome of interventional pain procedures. A quarter of respondents were not sure whether sedation helped with the outcome of interventional pain procedures.

Question 9: the procedures that patients would be given sedation for

Patient request for sedation irrespective of the procedure undertaken was the main reason. This was closely followed by radiofrequency procedures and anxious or needle-phobic patients undergoing a procedure (see Figure 5).

Sedation was also offered to the patients to prevent pain during positioning.

Question 10: who undertakes the interventional pain procedure?

The majority of procedures (94%) were carried out by the Consultants while the remaining 6% were undertaken by trainees.

Discussion

There appears to be a wide variation in the sedation practices of interventional Pain Specialists in the United Kingdom. Only a quarter of respondents discussed sedation in detail.

There is increasing pressure on sedationists to obtain written consent before sedation, to ensure documented proof of valid consent. Sedation helps with allaying anxiety, reducing movement and facilitating cooperation during the procedure, and when combined with analgesics, it can reduce the discomfort during injections. However, it could lead to airway compromise and arrhythmias from hypercapnia (from hypoventilation), which could lead to potentially fatal consequences. In addition, as with any drug administered, there is always the risk of an allergic reaction or adverse drug reaction such as nausea and vomiting (e.g. from opiates).

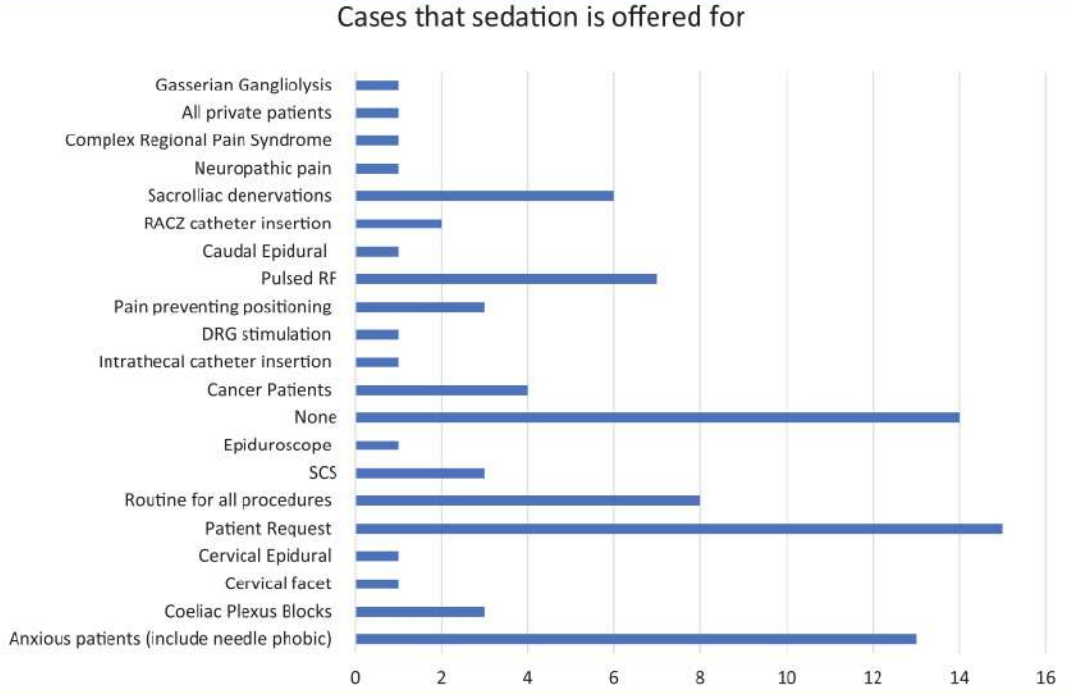
Sedation could also lead to potential false-positive results with diagnostic pain interventions since some sedatives have analgesic properties (e.g. opiate medications). Some studies have indicated an association between sedation and increased risk of nerve damage as the patient is unable to feed back to the interventionist in the same way as an unsedated patient. There may be legal repercussions from providing sedation without adequate proof of consent if there are complications.

According to the survey, some clinicians are only taking written consent for sedation if the patient asks for sedation. Interestingly, there is some evidence that sedation does not affect patient comfort during interventional pain procedures.[1]

It is of some concern that the Pain Specialists are both providing sedation and doing the procedure in 40% of cases. There are guidelines that suggest we should have a dedicated sedationist. However, the guidelines do seem variable depending on the area of the procedure (endoscopy or interventional radiology sedation is often given without a dedicated sedationist).

In the cases where a trainee or ODP/Anaesthetic nurse is providing the sedation, there is a question as to who holds ultimate responsibility for the sedation and management of any complications. This may be the Pain Specialists again, which

Figure 5. Procedures for which sedation is provided.



raises the concerns outlined above regarding management of complications.

Roughly a quarter of Pain Specialists who responded to the questionnaire do not provide sedation for any procedures. This may account for the fact that no anaesthetic machine is present in 25% of procedure rooms. It is assumed that all the people providing sedation either routinely or occasionally had access to an anaesthetic machine. The Royal College has guidelines for the administration of sedation with access to adequate airway and ventilation equipment.[2]

There does not seem to be a consensus on which sedative drugs are used. The majority (44%) described using a tailored combination of other drugs. The main deviation from the established use of midazolam or propofol with or without alfentanil seems to be superseded in some cases by fentanyl (in combination with either midazolam or propofol). There is a subsection (7%) who stated they used Entonox. This may be the ODP/Anaesthetic nurses using Entonox, as it has a lower risk of airway loss.

Only half of the patients having sedation had their oxygen saturations, BP and ECG monitored. In total, 1% of the patients did not have any monitoring applied. The Association of Anaesthetists of Great Britain and Ireland (AAGBI), Royal College of Anaesthetists (RCoA) and Faculty of Pain Medicine have issued guidance regarding monitoring during sedation.[2] It would have been interesting to see how many patients had access to $EtCO_2$ measurement. In those patients having a combination of midazolam or propofol with an opiate like alfentanil or fentanyl, there is a high risk of apnoea and potentially hypoxia. Difficulties with observing airway patency are compounded with the majority of procedures being performed in the prone position.

Only 50% of patients had supplemental oxygen given as a matter of routine. In 28% of patients, they only received oxygen if they desaturated. This is not ideal as the main reason for desaturation during sedation is hypoventilation or apnoea. Desaturation in these cases is a late sign. In addition, once a patient desaturates it can take a while for the patient to be re-oxygenated. By providing routine oxygenation to patients with sedation, one increases the oxygen reservoir in the functional residual capacity of the lungs. This will reduce the risk of desaturations. If the patient does have an apnoeic period, pre-oxygenation allows longer for the patient to recover their respiratory rate before there is a desaturation.

In total, 2% of respondents never provided oxygen. It is hard to believe this is the case if the patient has desaturated. There

Figure 6. A summary of the procedures outlined in the 2010 American Society of Anaesthesiology statement.[5]

- Epidural Steroid Injections
- Trigger Point Injections
- Epidural Blood Patches
- Sacroiliac Joint Injections
- Bursa Injections
- Occipital Nerve Blocks
- Facet Joint Injections

are two possible explanations for this: one is that they do not provide oxygen as they have a separate sedationist who manages the patient's sedation; the other is that 1% of respondents do not place any monitoring on the patient during sedation. Therefore, they may be missing the hypoxic event that would normally trigger supplemental oxygen provision.

The number of interventional pain procedures being performed each year is increasing as the patient population rises and new techniques are developed. According to the 2014–2015 UK Hospital Episodes Statistics (HES) data, there were 82,188 therapeutic epidurals, 13,796 facet joint denervations and 83,308 'other procedures' around the spine performed in the United Kingdom.[3] In the United States, there has been an 11% annual increase in select Medicare service beneficiaries, whereas facet and sacroiliac (SI) joint interventions increased by 313% in 10 years.[4]

With the rising numbers of these procedures being performed, there is no UK consensus on how the procedures should be performed, with regard to sedation or analgesia during the intervention. In 2010, the American Society of Anaesthesiology stated, 'the majority of minor pain procedures, under most routine circumstances, do not require anaesthesia other than local anaesthetic'.[5] The procedures encompassed in this statement are summarised in Figure 6.

Cucuzzella et al.[6] performed a retrospective survey of 500 patients who underwent cervical, thoracic, lumbar epidural or facet joint injections. They found that only 17% requested sedation if given the choice;[6] about half of the patients had sedation for their procedure out of the 500 surveyed. In a subsequent follow-up study of the 500 patients, 93% who did not have sedation were happy with their decision to not have sedation.[7] Only 1.5% of the total said that they would have liked sedation.

There is evidence that moderate to heavy sedation is associated with an increased risk of neurological damage. The risk of spinal cord injury during cervical procedures has been shown to be much higher with general versus local anaesthetics.[8,9] Gajraj[10] suggested that sedated patients were unable to report paraesthesia, perhaps significantly increasing the risk of spinal and nerve damage during cervical injections.

Smith et al.[1] discussed the potential drawback of using sedation in diagnostic blocks. Depending on the type of sedation used, if it had analgesic effects itself (such as an opiate – fentanyl being a common option), it may make it difficult to assess the actual effect of the block.

The commonest reason for offering sedation to patients undergoing interventional pain procedures is for patient comfort and satisfaction. However, there is little evidence that using sedation improves patient satisfaction. Diehn et al.[11] surveyed patients undergoing transforaminal epidural steroid injections without sedation. They found that the vast majority rated their experience as either good (15%), very good (30%) or excellent (51%). There was only a 0.4% incidence of vasovagal events. The authors argued that the high patient satisfaction rates, coupled with the low vasovagal events, indicated that the procedure could be performed without sedation. They hypothesised that the increased risk of sedation-related neurological injuries far outweighed any potential benefit from patient satisfaction achieved with providing sedation. Trentman et al.[12] had similar findings when looking specifically at rates of vasovagal episodes in patients having cervical and lumbar transforaminal epidural steroid injections.

Cohen et al. further confirmed the potential confounding effects of sedation for diagnostic pain procedures. In a randomised, controlled, crossover trial, they found lower pain scores in patient diaries for diagnostic SI joint injections or sympathetic nerve root blocks in the patients who received sedation versus those who did not.[13]

Overall, there is limited evidence for the use or not of sedation in interventional pain procedures. Although there are no cost analyses into sedation versus no sedation for interventional pain procedures, it would seem logical that offering sedation would increase the cost and complexity of the procedure. The potential for increased cost would be related to drugs, additional equipment and potentially additional personnel.

There are potential complications associated with the use of sedation. There are data that sedation for day-case procedures can increase the risk of falls, driving accidents and aspiration of gastric contents.[14,15]

One of the difficulties with research in this field is the definition of sedation. The AAGBI guidance on sedation refers to five types of anaesthetic administration. These are outlined in Figure 7.

There are a number of procedures for which the majority of Pain Physicians will routinely offer sedation. These are listed as follows:

Figure 7. The five forms of sedation outlined by the AAGBI.

None
Local Anaesthetic (including regional nerve block)
Light Plane of Sedation
Deep Plane of Sedation
General Anaesthetic

- Caudal;
- Radiofrequency ablation;
- Nerve root injection;
- RACZ catheter;
- Trigger point injections;
- SI joint injections;
- Epidural injections (transforaminal or interlaminar)
  - With local anaesthetic,
  - Without local anaesthetic,
- Diagnostic medial branch blocks;
- Regional nerve blocks;
- Plexus/ganglion blocks.

The aims of sedation are dependent on the procedure being performed. These can be divided into patient-specific and procedure-specific concerns. The procedure-specific issues include the necessity for the patient to stay still, being able to give appropriate feedback during the procedure (pain, paraesthesia, relief of pain) and duration of the procedure (longer procedures may be challenging for the patient to stay still). Patient-specific issues include anxiety, needle phobia, inability to stay still and discomfort being in particular positions.

There are specific considerations for the sedationist such as patient positioning (many interventional pain procedures are performed in the prone position, making access to the airway challenging) and use of special equipment (such as X-ray image intensifiers).

The RCoA has stipulated that even if no sedation is provided:

> *The following ancillary anaesthetic equipment must also be available at all sites where patients are undergoing any pain intervention procedure, even if no sedation or anaesthesia is being administered: Oxygen supply, facemasks, suction, airways (e.g. Guedel and laryngeal mask), tracheal tubes and intubation aids, self-inflating bag, trolley/bed/operating table that can be tilted head-down rapidly.*[16]

The General Medical Council (GMC) guidance of Good Practice and Managing Medicines and Devices does stipulate that any procedure or intervention that causes significant levels of pain or distress should be performed under sedation or a general anaesthetic.[17]

There are a number of drugs that can be used for sedation. The options are summarised below.

The commonly used drugs for sedation are as follows:

- Midazolam,
- Clonidine,
- Propofol,
- Fentanyl,
- Alfentanil,
- Remifentanil,
- Entonox (nitrous oxide with oxygen in a 50:50 ratio),
- Sevoflurane.

It should be noted that there are alternatives to sedation. These can include psychological support and the use of local anaesthetics.

To date, there have been no national or international guidelines pertaining to the conduct of interventional pain procedures, especially with regard to sedation for the interventions. There are, therefore, questions about where it should be performed, what equipment should be available, who should perform the sedation and who should supervise the sedationist (if the sedationist is a trainee). There is some general guidance published from the Royal College of Radiologists and Anaesthetists about interventional procedures in general (not specifically related to interventional pain procedures).[2,16]

There are very few studies that have outlined current practice, and none to date in the United Kingdom have been published. Kohan et al.[18] published an American survey of 337 physicians (out of 4,037 members – 8.4% response rate). They found that 82% of patients had sedation, and most needed a driver post-procedure.

The heterogeneity of practice among interventional Pain Specialists in the United Kingdom highlights the necessity of clear national guidelines. Any such guidelines should be flexible enough to allow individual practitioners to tailor their treatments to their patients and their individual practice. However, there should always be an emphasis on safe sedation. This may require a separate sedationist for the intervention list who can either be a Consultant Anaesthetist, Trainee Anaesthetist undergoing sedation module or trained ODP/Anaesthetic nurse. The presence of a separate sedationist might entail the use of routine monitoring as described by the AAGBI. This would include the use of oxygen saturation probes, ECG, BP and $EtCO_2$ monitors.

Conclusion

Further study is required to help standardise practice and ensure the safe management of sedation. Part of the proposed

guidelines would be to highlight the cases where sedation may not be necessary. Although individual clinician discretion should always be respected, the less sedation that is provided, the lower the risk of sedation-related adverse events.

Standardising the equipment and protocols required for sedation would be important for any protocols and guidance produced for sedation relating to interventional pain procedures. These protocols could be built on similar protocols and guidelines developed by the RCoA and AAGBI for sedation in other specialties.

Author contributions

M.S. created, designed and distributed the survey, interpreted the results and performed literature search, compilation writing and proofreading; G.R. interpreted the results and performed literature search, compilation writing and proofreading; F.N. designed the survey, interpreted the results and performed proofreading; H.A.-S. designed and distributed the survey, interpreted the results and performed proofreading; A.D. performed proofreading and compilation writing.

References

1. Smith HS, Colson J, and Sehgal N. An update of evaluation of intravenous sedation on diagnostic spinal injection procedures. *Pain Physician* 2013; 16: SE217–28.
2. Safe sedation analgesia anaesthesia with the radiology department, 2003. Available online at http://www.rcr.ac.uk/publications.aspx?PageID=310&PublicationID=186
3. Available online at https://data.gov.uk/dataset/hospital_episode_statistics_admitted_patient_care (accessed 5 November 2018).
4. Manchikanti L, Pampati V, and Hirsch JA. Utilization of interventional techniques in managing chronic pain in Medicare population from 2000 to 2014: An analysis of patterns of utilization. *Pain Physician* 2016; 19: E531–46.
5. ASAHQ statement on anaesthetic care during interventional pain. Available online at http://www.asahq.org/~/media/Sites/ASAHQ/Files/Public/Resources/standards-guidelines/statement-on-anesthetic-care-during-interventional-pain-procedures-for-adults.pdf (accessed 5 December 2018).
6. Cucuzzella TR, Delport EG, Kim N, et al. A survey: Conscious sedation with epidural and zygapophyseal injections: Is it necessary? *The Spine Journal* 2006; 6(4): 364–9.
7. Kim N, Delport E, Cucuzzella T, et al. Is sedation indicated before spinal injections? *Spine* 2007; 32(25): E748–52.
8. Fitzgibbon DR, Rathmell JP, Michna E, et al. Malpractice claims associated with medication management for chronic pain. *Anesthesiology* 2010; 112(4): 948–56.
9. Rathmell JP, Michna E, Fitzgibbon DR, et al. Injury and liability associated with cervical procedures for chronic pain. *Anesthesiology* 2011; 114(4): 918–26.
10. Gajraj NM. Avoid excessive sedation during cervical injections. *Anesthesiology* 2005; 102(4): 869.
11. Diehn FE, Geske JR, Amrami KK, et al. An audit of transforaminal epidural steroid injections without sedation: Low patient dissatisfaction and low vasovagal rates. *Pain Medicine* 2013; 14(7): 994–8.
12. Trentman TL, Rosenfeld DM, Seamans DP, et al. Vasovagal reactions and other complications of cervical vs. lumbar translaminar epidural steroid injections. *Pain Practice* 2009; 9(1): 59–64.
13. Cohen SP, Hameed H, Kurihara C, et al. The effect of sedation on the accuracy and treatment outcomes for diagnostic injections: A randomized, controlled, crossover study. *Pain Medicine* 2014; 15(4): 588–602.
14. Over sedation for emergency procedures in isolated locations, 2011. Available online at http://www.nrls.npsa.nhs.uk/resources/type/signals/?entryid45=94848
15. Malviya S, Voepel-Lewis T, and Tait AR. Adverse events and risks of sedation and general anaesthesia in children undergoing MRI and CT scans. *Anesthesia & Analgesia* 1998; 86(2S): 410S.
16. Available online at https://www.rcoa.ac.uk/document-store/safe-sedation-practice-healthcare-procedures-standards-and-guidance (accessed 5 December 2018).
17. GMC. Good practice in prescribing and managing medicines and devices, 2013. Available online at http://www.gmc-uk.org/guidance/ethical_guidance/14316.asp (accessed 20 December 2018).
18. Kohan L, Salajegheh R, Hamill-Ruth RJ, et al. A review and survey of policies utilized for interventional pain procedures: A need for consensus. *Journal of Pain Research* 2017; 10: 625–34.

conditions.[41–52] These contradictory findings are likely due to the following differences in practice between USGDN and DN.

Number of needles

3D ultrasound studies have shown that MTrPs occur in clusters,[35] and the use of only a limited number (up to 6) of needles in DN may leave many MTrPs in a muscle untreated. Our experience with USGDN (which utilises 30–60 needles per session, Table 1) suggests that the number of active MTrPs causing spontaneous pain or even latent MTrPs form only the tip of the iceberg of MPS – the majority of the problem may be attributable to a subclinical contribution of asymptomatic MTrPs or their predecessor abnormalities in the muscle.

Needle length

Blindly performed DN usually utilises short 25–50 mm or occasionally 75 mm needles that may not be able to reach deep-seated MTrPs, particularly in obese patients. It is our routine observation with USGDN (where needles as long as 120 mm are used) that it is the deepest layers of muscle (e.g. multifidus in back pain or vastus intermedius juxtaposed on femur or serratus anterior just superficial to ribs and intercostal muscles) that seem to have the most taut bands that exhibit distinct LTRs and a perceptible resistance to needle passage, as well as cause most pain to the patient on needling.

Duration of needling

In DN and in IMS, the needle is rapidly inserted with a pumping motion into the MTrP and kept in situ for only a few seconds before removal. In contrast, during USGDN, they are smoothly inserted till a resistance is encountered or patient reports pain, when the introduction is halted for a few seconds and then advanced slowly and gradually into the muscle as the muscle relaxes. Needles are maintained in situ for 20–30 minutes and come out easily and painlessly, compared to the resistance to needle passage at insertion, or after maintenance for a shorter period (e.g. 10 minutes). We have observed repeatedly that early removal of needles results in greater pain during needle removal and is also far less effective at resolving the original pain, as shorter needle maintenance in situ is often insufficient for MTrP deactivation. This difference in needle maintenance time between DN and USGDN may be highly relevant: using ultrasound monitoring, we have observed that cessation of LTRs (which indicates deactivation of an MTrP) can require up to 20–30 minutes of needle maintenance. Clinically, needle introduction into an active MTrP produces a gripping of the needle by the muscle with intense pain and any attempt to redirect the needle (away from a vessel) at this time is painful to the patient. We believe the immediate removal of the needle after eliciting the LTR precludes the wind down of the natural stimulation-relaxation induced by the needling observed under ultrasound visualisation. Therefore, we surmise that the routine practice among DN and IMS practitioners of rapidly pumping the needle in an attempt to elicit a clinically visible LTR may fail to fully deactivate the MTrP.

The practitioner effect

Currently, DN practised by physiotherapists as the sole treatment modality involves targeting a few painful spots in the muscle, with pain relief as the main goal. Disability relief is not targeted. USGDN practised by pain physicians has the flexibility of serving as a sole modality, or as a follow-up to neural interventions, depending on the severity of clinical presentation. USGDN at our centre aims as much for disability relief as pain relief, based on the theory that myofascial pain and functional impairment are two aspects of the same pathology. To this end the agonist, antagonist and synergists are comprehensively addressed. However, the effectiveness of DN versus USGDN has not been explored in a study.

The MTrP and motor neuropathy – the connection

While the role of the somatosensory nervous system in the genesis and propagation of pain is well established, the possibility that motor nerves are as vulnerable to being affected by neuropathy as sensory nerves has not been considered. Based on the effect of USGDN in multiple pain conditions considered to be purely neuropathic,[41,46,47,50,51] we have come to refer to this motor neuropathy as neur*omyo*pathy, because we believe that not only is the motor nerve involved in the neuropathic process, but it also produces significant changes in the muscle by way of MTrP generation and taut bands, culminating in MPS.[2,10,19,29,31–38,53–56] Simons et al. have proposed the integrated trigger point hypothesis incorporating the concepts of the local ischaemia in an energy crisis (the Cinderella hypothesis)[53–55] and this has been further expanded by Gerwin et al.[56] to explain MTrP generation: briefly, increased discharge of acetylcholine at the motor end plate or the neuromuscular junction produces recordable electromyogram changes in the end plate zone near MTrPs. Electrical discharges that occur with frequencies that are 10–1,000 times that of normal end plate potentials have been shown in humans, presumably as a result of increased discharge of acetylcholine.[57] This crescendo of miniature end plate potentials leads to a muscle contracture, wherein myosin filaments get stuck at the Z band. The lack of ATP (and perhaps oxygen), which is required to break the cross-bridges between actin and myosin filaments, leads to the formation of an MTrP. We propose that if these theories, which pertain to the downstream effects of increased acetylcholine discharge at the motor end plate or neuromuscular junction, were to be extended a little more proximally from the neuromuscular junction to the motor nerve,

it would form the missing connection between motor nerve neuropathy and MTrP production.

While there is reporting of MPS in neuropathic pain,[59–61] these associations have been dismissed as secondary musculoskeletal issues unconnected to the main pathology. We have proposed that MTrPs are generated in neuropathic conditions as an end result of neuropathy of motor nerve. Thus, many pain syndromes, instead of being described as a neuropathy, would be better described as a neuromyopathy, which is an all-encompassing terminology that describes disorders of peripheral nerve or lower motor neuron that directly produce muscle changes that become independent pain generators. In our clinical experience, residual pains in multiple neuropathic conditions have been unequivocally relieved by USGDN,[46,47,49–51] warranting a serious consideration of the possibility that the muscle is actually an expressor of neural pathology.

The present description of neuropathic pain as pain arising as a direct consequence of a lesion or disease affecting the *somatosensory* system[1] is less than comprehensive. While the division of chronic pain into secluded chapters in text books such as neuropathic pain and myofascial pain makes it easy for physicians to neatly compartmentalise their understanding of this condition, neuropathy in real-life situations appears to have no special preferences for sensory nerves one way or the other, and motor and autonomic nerves may be equally involved in the neuropathic process. Moreover, muscles seldom act in isolation; MTrPs in flexor muscles can cause strain in other agonists, antagonists, synergists and fixators to give rise to MTrPs in these muscles. Furthermore, MTrPs can refer pain by forming secondary satellite MTrPs at other distal sites along the kinetic chain of muscles involved in complex movements across many joints. Muscle kinetic chains are combinations of several successively arranged joints constituting a complex motor unit: for example, the act of picking up an object involves several muscles acting across the shoulder, elbow, wrist and the small joints of the hand as well as the neck. An MTrP in one group of muscles (biceps) not only compromises the movement of that muscle group but also places an extra strain on the other muscles and joints required to achieve the function. Thus, we believe that muscles are not just passive expressors, but are also the perpetrators, facilitators, sustainers and amplifiers of the pathogenic process responsible for pain generation. The sheer interdependent complexity of muscle function ensures the production of myriad bizarre symptoms, which are the hallmark of many neuropathic pain syndromes that remain unresponsive to opioids. Once formed, the MTrPs become the autonomous source of pain, inflammation, peripheral and central sensitisation, all of which persist even after treatment with spinal or peripheral nerve blocks, radiofrequency procedures, and even intrathecal drug delivery systems and spinal cord stimulation. Pain from persistent MTrPs might well explain the conclusions of the Mint trial[62] and the second ASBMR task force report on vertebral augmentation[63] that opined that radiofrequency denervation and vertebroplasty procedures were not useful in relieving pain.

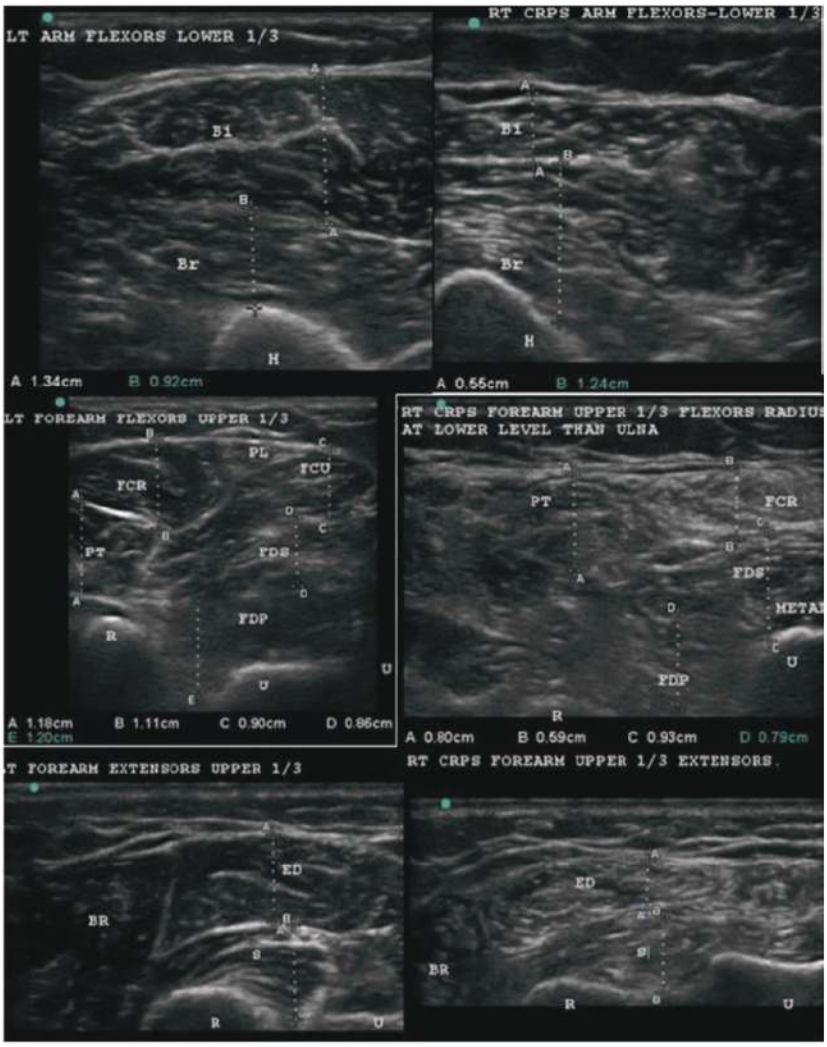
Effectiveness of USGDN in current clinical pain practice

Currently, interventional pain management procedures address the nerves affected by neuropathy and then follow up with physiotherapy referrals and opioid prescription for residual pain. Our clinic is probably the only one (to our knowledge) to take an integrative approach, treating pain syndromes not as a neuropathy, but rather a neuromyopathy: the *neural* component is usually first addressed in patients with severe pain with interventions such as transforaminal epidural injection, cervical interlaminar epidural, radiofrequency procedures (both thermal radiofrequency (TRF) and pulsed radiofrequency (PRF)), continuous nerve or plexus infusions or intravenous lignocaine/ketamine infusions. The residual pains persisting after these neural interventions are addressed by systematic USGDN, which routinely achieves a dramatic reduction in pain and also disability. Strikingly, in many patients, USGDN is effective as the sole modality of treatment. Furthermore, neuropathic symptoms such as burning, allodynia, and hyperalgesia and hyperaesthesia (seen in herpes and post-herpetic neuralgia, brachial plexus injuries, CRPS and other severe neuropathic conditions) are routinely and predictably relieved with 2–3 sessions of USGDN. After initially puzzling over why such 'sensory' symptoms were relieved by USGDN, a treatment that patently and exclusively addresses muscles, we came to the realisation that these so-called sensory symptoms could actually be the result of an intense spasm of erector pili muscles in the dermis.[47] USGDN results in a relaxation of these dermal muscle fibres while also deactivating MTrPs in much deeper-seated muscles.

Effectiveness of USGDN in CRPS

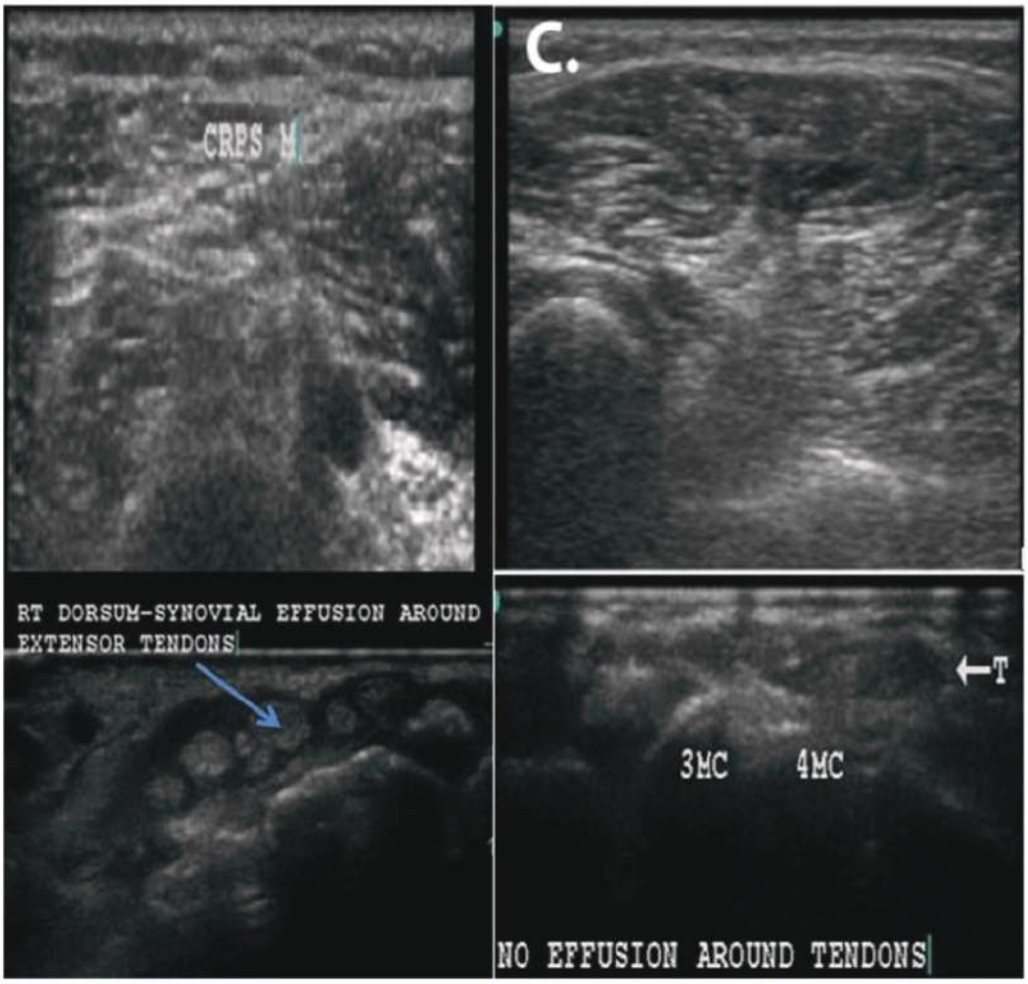
Our experience with CRPS has been in stark contrast to the world literature, in that complete reversal of CRPS has been routinely achieved.[42–45,47,52,64] To date, CRPS has been reversed in 204 consecutive patients, including 2 paediatric patients, 5 cases of bilateral CRPS,[42] 1 case of recurrent CRPS[43] and 4 cases of chest wall CRPS that had developed after coronary bypass surgery. There were 155 cases of upper extremity CRPS, of which 149 had CRPS-1 and 6 had CRPS-2. There were 45 patients with lower extremity CRPS, of which 41 had CRPS-1 and 4 had CRPS-2[42–44,47,52,64] (unpublished data). Given the overwhelming incidence of disability in CRPS and its relief by USGDN, a treatment that only deactivates MTrPs, we have hypothesised that the primary pathology of CRPS is actually motor impairment: formation of abundant MTrPs and

Figure 3. Muscle ultrasonography is a novel diagnostic investigation for CRPS. Ultrasonographic images of digital flexor and extensor muscles of the normal left arm (left-hand side panels) compared with CRPS-affected arm (right-hand side panels). Muscles in the normal arm (left panels) show clear demarcation of muscles and well-defined muscle outlines, with the hypoechoic (dark) background representing muscle fibres, and bright curvilinear echoes representing the connective tissue frame work of the perimysium. In the CRPS-affected arm (right panels), muscle outlines are lost and there is a predominance of uniform hyperechoic fibrous tissue, with loss in muscle bulk. BI: biceps; BR: brachialis; H: humerus; R: radius; U: ulna; PT: pronator teres; FCR: flexor carpi radialis; PL: palmaris longus; FDS: flexor digitorum superficialis; FDP: flexor digitorum profundus; BR: brachioradialis; ED: extensor digitorum; S: supinator.



taut bands in the agonist/antagonist muscles such as flexor/extensors and supinator/pronators causes an impaired reciprocal inhibition that culminates in an abnormal co-contraction that severely impedes all extremity movements. Attempted movements of muscles tethered by constant co-contraction lead to friction and inflammation at the digital tenosynovial sheaths (demonstrable on ultrasound, Figure 4) similar to that seen in de Quervain's tenosynovitis.

Figure 4. Muscle ultrasonography as a prognostic tool in CRPS. Ultrasound images obtained before (right panel) and after (left panel) USGDN of the CRPS-affected hand. *Top row*: Ultrasound images of the forearm just below the elbow before and after USGDN shows the return of normal outlines as well as return of hypoechoic muscle fibres in the muscles (right panel). The bony outlines of radius and ulna obscured by the hyperechoic echoes pre-USGDN (left panels) become clearer after treatment (right panels). There is also an increase in muscle bulk in the right panels, compared to left. *Bottom row*: Images show the tenosynovial effusion around the digital extensor tendons before USGDN (left panel) which is completely resolved post-USGDN, suggesting that USGDN of the digital extensor and the flexor muscles relieves the co-contraction and the consequent tenosynovial inflammation and effusion. T: tendons; MC: metacarpal bone.



We have proposed that the inflammation seen in CRPS is secondary to a global tenosynovitis, rather than a neurogenic inflammation that has been proposed by other authors. The unrelenting co-contraction is likely responsible for the resource depletion, which causes the hypoxic changes like wasting and fibrosis with the dystrophic and atrophic manifestations of later CRPS. We have consistently observed that this co-contraction responds with exquisite sensitivity to USGDN. Relaxation of the co-contracted agonist/antagonist muscles of the CRPS-affected limb automatically reduces the synovial friction and

resolves the inflammatory tendinoses in the hand, thereby reversing the pain, vasomotor, sudomotor and sensory features forming the Budapest criteria. Relaxation of muscles also allows a return of the normal coordination between the flexor (agonist) and extensor (antagonist) muscles with dramatic improvement of stiffness, weakness and disability. Ultrasound documentation of changes in CRPS-affected muscles, as well as their reversal after USGDN, supports this theory[45,47,52,64] (Figures 3 and 4).

USGDN has also proven effective in numerous other pain conditions, alone or in combination with modalities such as PRF, ultra-low dose botox and trigger injection of ultra low-dose steroids. An incomplete list of conditions that have been improved includes various neuropathic pains, including post-surgical pains,[41,65] post-herpetic neuralgia (manuscript under preparation), diabetic neuropathy, brachial plexus injury, spinal cord injury with causalgia (unpublished data); central pains such as post-stroke pain, and deafferentation pains (unpublished data); trigeminal neuralgia (manuscript under preparation); migraines (unpublished data); lower back pain (discogenic, facetogenic, spondylolytic and spondylolisthetic (unpublished data)); failed back surgery syndrome;[49] arthritis of knee (both osteoarthritis[66] and rheumatoid arthritis (unpublished data)); writer's cramp;[48] shoulder pains (frozen shoulder (unpublished data)); chronic pelvic pain;[50] and cancer pains.[46,51] USGDN is also used as an add-on therapy after transforaminal epidural and pars injections and after RF denervation of the medial branch to the facet joint.[65]

Conclusion

USGDN is a low-cost, simple yet safe technique that simultaneously addresses pain and disability across a wide range of chronic pains with a specific and predictable accuracy. For its widespread dissemination, the following are needed:

1. The necessity to sensitise pain physicians to the concept that neuromyopathy is operative in most chronic pain conditions.
2. Training pain physicians in muscle anatomy via cadaveric dissection workshops and educating in musculoskeletal ultrasound use.
3. Well-designed clinical trials to determine its superiority over opioid prescription.
4. Basic science research to explore the concept of neuromyopathy as a causative factor in various chronic pain conditions.

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References

1. Treede RD, Jensen TS, Campbell JN, et al. Neuropathic pain: Redefinition and a grading system for clinical and research purposes. *Neurology* 2008; 70(18): 1630–5.
2. Dommerholt J, Bron C, and Franssen J. Myofascial trigger points: An evidence-informed review. *Journal of Manual & Manipulative Therapy* 2006; 14(4): 203–21.
3. Fishbain DA, Goldberg M, Meagher BR, et al. Male and female chronic pain patients categorized by DSM-III psychiatric diagnostic criteria. *Pain* 1986; 26(2): 181–97.
4. Fleckenstein J, Zaps D, Ruger LJ, et al. Discrepancy between prevalence and perceived effectiveness of treatment methods in myofascial pain syndrome: Results of a cross-sectional, nationwide survey. *BMC Musculoskeletal Disorders* 2010; 11: 32.
5. Gerwin RD. Classification, epidemiology, and natural history of myofascial pain syndrome. *Current Pain and Headache Reports* 2001; 5(5): 412–20.
6. Malanga GA, and Cruz Colon EJ. Myofascial low back pain: A review. *Physical Medicine and Rehabilitation Clinics of North America* 2010; 21(4): 711–24.
7. Skootsky SA, Jaeger B, and Oye RK. Prevalence of myofascial pain in general internal medicine practice. *The Western Journal of Medicine* 1989; 151(2): 157–60.
8. Travell JG, and Simons DG. *Myofascial Pain and Dysfunction: The Trigger Point Manual*. Philadelphia, PA: Lippincott Williams & Wilkins, 1983.
9. Arendt-Nielsen L, and Castaldo M. MTPs are a peripheral source of nociception. *Pain Medicine* 2015; 16(4): 625–7.
10. Lucas KR, Polus BI, and Rich PA. Latent myofascial trigger points: Their effects on muscle activation and movement efficiency. *Journal of Bodywork and Movement Therapies* 2004; 8(3): 160–6.
11. Kelgren JH. Referred pains from muscle. *British Medical Journal* 1938; 1(4023): 325–7.
12. Legge D. A history of dry needling. *Journal of Musculoskeletal Pain* 2014; 22(3): 301–7.
13. Harman J, and Young R. Muscle lesions simulating visceral disease. *The Lancet* 1940; 235(6095): 1111–3.
14. Kelly M. Muscular lesions in somatic pain. *British Medical Journal* 1941; 1(4183): 379.
15. Brav EA, and Sigmond H. The local and regional injection treatment of low back pain and sciatica. *Annals of Internal Medicine* 1941; 15(5): 840–52.
16. Paulett JD. Low back pain. *The Lancet* 1947; 250(6469): 272–76.
17. Travell J, and Rinzler SH. The myofascial genesis of pain. *Postgraduate Medicine* 1952; 11(5): 425–34.
18. Lewit K. The needle effect in the relief of myofascial pain. *Pain* 1979; 6(1): 83–90.
19. Gunn CC, and Wall PD. *The Gunn Approach to the Treatment of Chronic Pain: Intramuscular Stimulation for Myofascial Pain of Radiculopathic Origin*. London: Churchill Livingstone, 1996.
20. Espí-López GV, Serra-Añó P, Vicent-Ferrando J, et al. Effectiveness of inclusion of dry needling in a multimodal therapy program for patellofemoral pain: A randomized parallel-group trial. *The Journal of Orthopaedic and Sports Physical Therapy* 2017; 47(6): 392–401.
21. Tejera-Falcon E, Toledo-Martel NDC, Sosa-Medina FM, et al. Dry needling in a manual physiotherapy and therapeutic exercise protocol for patients with chronic mechanical shoulder pain of unspecific origin: A protocol for a randomized control trial. *BMC Musculoskeletal Disorders* 2017; 18(1): 400.
22. Gattie ER, Cleland JA, and Snodgrass SJ. Dry needling for patients with neck pain: Protocol of a randomized clinical trial. *JMIR Research Protocols* 2017; 6(11): e227.
23. Cummings M, Ross-Marrs R, and Gerwin R. Pneumothorax complication of deep dry needling demonstration. *Acupuncture in Medicine* 2014; 32(6): 517–9.
24. Lee JH, Lee H, and Jo DJ. An acute cervical epidural hematoma as a complication of dry needling. *Spine* 2011; 36(13): E891–3.
25. Ullah W, Ahmad A, Mukhtar M, et al. Acupuncture-related cardiac complications: A systematic review. *The Journal of Invasive Cardiology* 2019; 31(4): E69–72.
26. Zhou K, Ma Y, and Brogan MS. Dry needling versus acupuncture: The ongoing debate. *Acupuncture in Medicine* 2015; 33(6): 485–90.
27. Tough EA, White AR, Cummings TM, et al. Acupuncture and dry needling in the management of myofascial trigger point pain: A systematic review and meta-analysis of randomised controlled trials. *European Journal of Pain* 2009; 13(1): 3–10.

28. Fan AY, Xu J, and Li YM. Evidence and expert opinions: Dry needling versus acupuncture (I). *Chinese Journal of Integrative Medicine* 2017; 23(1): 3–9.
29. Hong CZ, and Torigoe Y. Electrophysiological characteristics of localized twitch responses in responsive taut bands of rabbit skeletal muscle fibers. *Journal of Musculoskeletal Pain* 1994; 2(2): 17–43.
30. Gerwin R, and Duranleau D. Ultrasound identification of the myofacial trigger point. *Muscle & Nerve* 1997; 20(6): 767–8.
31. Shah JP, and Gilliams EA. Uncovering the biochemical milieu of myofascial trigger points using in vivo microdialysis: An application of muscle pain concepts to myofascial pain syndrome. *Journal of Bodywork and Movement Therapies* 2008; 12(4): 371–84.
32. Shah JP, Thaker N, Heimur J, et al. Myofascial trigger points then and now: A historical and scientific perspective. *PM & R: The Journal of Injury, Function, and Rehabilitation* 2015; 7(7): 746–61.
33. Kuan T-S, Hsieh YL, Chen SM, et al. The myofascial trigger point region: Correlation between the degree of irritability and the prevalence of endplate noise. *American Journal of Physical Medicine & Rehabilitation* 2007; 86(3): 183–9.
34. Chen Q, Wang HJ, Gay RE, et al. Quantification of myofascial taut bands. *Archives of Physical Medicine and Rehabilitation* 2016; 97(1): 67–73.
35. Sikdar S, Shah JP, Gebreab T, et al. Novel applications of ultrasound technology to visualize and characterize myofascial trigger points and surrounding soft tissue. *Archives of Physical Medicine and Rehabilitation* 2009; 90(11): 1829–38.
36. Mense S. The pathogenesis of muscle pain. *Current Pain and Headache Reports* 2004; 7: 419–25.
37. Zhang H, Lu JJ, Huang QM, et al. Histopathological nature of myofascial trigger points at different stages of recovery from injury in a rat model. *Acupuncture in Medicine* 2017; 35(6): 445–51.
38. Shah JP, Phillips TM, Danoff JV, et al. An in vivo microanalytical technique for measuring the local biochemical milieu of human skeletal muscle. *Journal of Applied Physiology* 2005; 99(5): 1977–84.
39. Borg-Stein J, and Simons DG. Focused review: Myofascial pain. *Archives of Physical Medicine and Rehabilitation* 2002; 83(3 Suppl. 1): S40–7; S48–9s.
40. Gattie E, Cleland JA, and Snodgrass S. The effectiveness of trigger point dry needling for musculoskeletal conditions by physical therapists: A systematic review and meta-analysis. *The Journal of Orthopaedic and Sports Physical Therapy* 2017; 47(3): 133–49.
41. Vas L, Khandagale N, and Pai R. Successful management of chronic postsurgical pain following total knee replacement. *Pain Medicine* 2014; 15(10): 1781–5.
42. Vas L, and Pai R. Successful reversal of complex regional pain syndrome type 1 of both upper extremities in five patients. *Pain Medicine* 2012; 13(9): 1253–6.
43. Vas L, and Pai R. Reversal of complex regional pain syndrome type 2 and the subsequent management of complex regional pain syndrome type 1 occurring after corrective surgery for residual ulnar claw. *Pain Medicine* 2014; 15(6): 1059–63.
44. Vas L, and Pai R. Complex regional pain syndrome-type 1 presenting as deQuervain's stenosing tenosynovitis. *Pain Physician* 2016; 19(1): E227–34.
45. Vas L, and Pai R. Musculoskeletal ultrasonography to distinguish muscle changes in complex regional pain syndrome type 1 from those of neuropathic pain: An observational study. *Pain Practice* 2016; 16(1): E1–13.
46. Vas L, and Pai R. Ultrasound-guided dry needling as a treatment for postmastectomy pain syndrome: A case series of twenty patients. *Indian Journal of Palliative Care* 2019; 25(1): 93–102.
47. Vas L, Pai R, Geete D, et al. Improvement in CRPS after deep dry needling suggests a role in myofascial pain. *Pain Medicine* 2018; 19(1): 208–12.
48. Vas L, Pai R, Khandagale N, et al. Myofascial trigger points as a cause of abnormal cocontraction in writer's cramp. *Pain Medicine* 2015; 16(10): 2041–45.
49. Vas L, Pai R, Pawar KS, et al. 'Piriformis syndrome': Is it only piriformis. *Pain Medicine* 2016; 17(9): 1775–9.
50. Vas L, Pattanik M, and Titarmore V. Treatment of interstitial cystitis/painful bladder syndrome as a neuropathic pain condition. *Indian Journal of Urology* 2014; 30(3): 350–3.
51. Vas L, Phanse S, and Pai R. A new perspective of neuromyopathy to explain intractable pancreatic cancer pains; Dry needling as an effective adjunct to neurolytic blocks. *Indian Journal of Palliative Care* 2016; 22(1): 85–93.
52. Vas LC, Pai R, and Pattnaik M. Musculoskeletal ultrasonography in CRPS: Assessment of muscles before and after motor function recovery with dry needling as the sole treatment. *Pain Physician* 2016; 19(1): E163–79.
53. Hägg G. The Cinderella hypothesis. In: Johansson H, Windhorst U, Djupsjöbacka M, et al. (eds) *Chronic Work-related Myalgia: Neuromuscular Mechanisms Behind Work-Related Chronic Muscle Pain Syndromes*. Gävle: Gävle University Press, 2003, pp. 127–32.
54. Simons DG. Review of enigmatic MTrPs as a common cause of enigmatic musculoskeletal pain and dysfunction. *Journal of Electromyography and Kinesiology* 2004; 14: 95–107.
55. Simons DG. New views of myofascial trigger points: Etiology and diagnosis. *Archives of Physical Medicine and Rehabilitation* 2008; 89(1): 157–9.
56. Gerwin RD, Dommerholt J, and Shah JP. An expansion of Simons' integrated hypothesis of trigger point formation. *Current Pain and Headache Reports* 2004; 8(6): 468–75.
57. Simons DG, Hong CZ, and Simons LS. Endplate potentials are common to midfiber myofacial trigger points. *American Journal of Physical Medicine & Rehabilitation* 2002; 81(3): 212–22.
58. Schmader K, and Dworkin RH. Herpes zoster and postherpetic neuralgia. In: Hurley RW (ed.) *Essentials of Pain Medicine*. New York: Elsevier, 2018, pp. 233–240; e232.
59. Delpont B, Blanc C, Osseby GV, et al. Pain after stroke: A review. *Revue Neurologique* 2018; 174(10): 671–74.
60. Sirois DA, and Jorns TP. Trigeminal neuropathic pain and orofacial neuralgias. In: Sirois DA and Jorns TP (eds) *Orofacial Disorders*. New York: Springer, 2017, pp. 213–222.
61. Subedi A, Chaudakshetrin P, Chotisukarat H, et al. Effect of co-morbid conditions on persistent neuropathic pain after brachial plexus injury in adult patients. *Journal of Clinical Neurology* 2016; 12(4): 489–94.
62. Juch JN, Maas ET, Ostelo RW, et al. Effect of radiofrequency denervation on pain intensity among patients with chronic low back pain: The Mint randomized clinical trials. *JAMA* 2017; 318(1): 68–81.
63. Ebeling PR, Akesson K, Bauer DC, et al. The efficacy and safety of vertebral augmentation: A second ASBMR task force report. *Journal of Bone and Mineral Research* 2019; 34(1): 3–21.
64. Vas LC, Pai R, and Radhakrishnan M. Ultrasound appearance of forearm muscles in 18 patients with complex regional pain syndrome 1 of the upper extremity. *Pain Practice* 2013; 13(1): 76–88.
65. Vas L, Khandagale N, and Pai R. Report of an unusual complication of radiofrequency neurotomy of medial branches of dorsal rami. *Pain Physician* 2014; 17(5): E654–7.
66. Vas L, Pai R, Khandagale N, et al. Pulsed radiofrequency of the composite nerve supply to the knee joint as a new technique for relieving osteoarthritic pain: A preliminary report. *Pain Physician* 2014; 17(6): 493–506.
67. Steindler A. *Kinesiology of the Human Body Under Normal and Pathological Conditions*. Springfield, IL: Charles C Thomas, 1955 (reprint 1977).

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Evaluating the effectiveness of Essential Pain Management programme as a method for improving health care professionals' knowledge of pain assessment and management in a District General Hospital

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Credit: Tetiana Lazunova.

Introduction

Inadequate pain management continues to be a problem facing health care professionals globally.[1] Despite advances in technology and medicine, pain management continues to be inadequate.[2] Regardless of the reason for their admission, all health care professionals will encounter a patient reporting an episode of pain during their admission.[3,4] It is estimated that between 37% and 84% of patients will report pain during their hospital admission, with the prevalence of severe pain being reported by between 9% and 36% of those admitted.[5] This high prevalence of pain is not only restricted to the hospital population, but it is also estimated that the prevalence of chronic pain in the United Kingdom is between 33% and 50% in the general population.[6] This prevalence increases significantly