JUNE 2019 VOLUME 17 ISSUE 2

PAIN NEWS

A PUBLICATION OF THE BRITISH PAIN SOCIETY

Credit: Bortonia

Cannabis in Canada what can their experience teach us?

The psychological origins of CRPS?

Treating patients with persistent pain and addiction

Pain and suffering and the duty of care

How not to be a doctor

ISSN 2050–4497

THE BRITISH PAIN SOCIETY
EXPERTISE WHERE IT MATTERS

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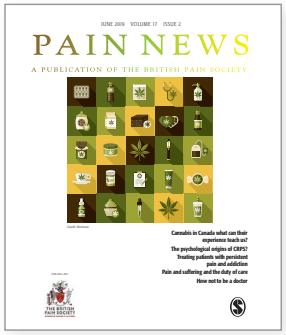

The Editor welcomes contributions including letters, short clinical reports and news of interest to members, including notice of meetings.

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Editorials

Regulars

Professional Perspectives

Informing practice

End Stuff

https://www.britishpainsociety.org/for-members/pain-news/

Have your say and contribute to *Pain News* today

Pain News is the newsletter for members of the British Pain Society and we welcome member and non-member contributions to share your news with the wider membership and beyond.

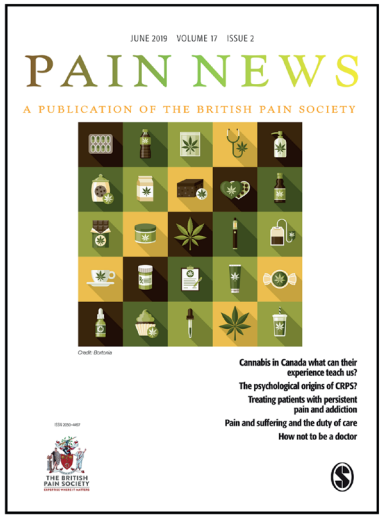
Do you have a news item to share?

Perhaps a professional perspective, or informing practice piece?

We'd love to hear from you so drop the Editor an email today at: rajeshmunglani@gmail.com

Pain News is published four times per year.

The Editor, Dr Rajesh Munglani, Associate Editor, Dr Margaret Dunham and Jenny Nicholas, CEO, welcome submissions for consideration of inclusion. Thoughtful pieces on Pain Medicine and related subjects including personal opinions, original work and reviews will be considered. By submitting an article, potential authors are agreeing to active editorial input to ensure conformity to house style, clarity and reasoned debate.



The British Pain Society
2019 Meetings & Events

THE BRITISH PAIN SOCIETY
EXPERTISE WHERE IT MATTERS

Philosophy & Ethics SIG Annual Meeting
"Exploring the Way Ahead For Pain Medicine: Caring For the Patient and the Clinician"
23rd – 26th June 2019
Rydal Hall, Cumbria

We are looking forward to another great meeting of the Philosophy and Ethics SIG taking place at Rydal Hall in Cumbria, Monday 24th -Weds 26th June. People usually travel to the venue on the afternoon of Sunday 23rd. Talks and discussion take place on the Monday and Tuesday with a break for relaxation or walking in the gardens and surrounding hills and lakes. Further talks take place on Wednesday morning and the meeting ends after lunch.

Pain Management Programmes SIG Bi-ennial Conference "Placing the Spotlight on the BiopsychoSOCIAL"
11th & 12th September 2019
Bristol

The theme of the conference is "Placing the Spotlight on the BiopsychoSOCIAL" and will feature excellent plenary talks and a diverse choice of workshops.

A social event including drinks and canapés will be held at Bristol Museum & Art Gallery on Wednesday 11th September between 18:00-21:00.

Headache SIG Annual Meeting
7th October 2019
London

The British Pain Society will be hosting the 3rd Headache Special Interest Group Meeting on Wednesday 5th June 2019 in London.

Talks include **"How important is psychology in management of headache symptoms" "CGRP receptors, what's new?"** and a session focusing on **"Neuromodulation in Headache".**

The meeting will feature also feature a Multidisciplinary workshop as well as a Hands on Botox workshop.

Cancer Pain Study Day
8th October 2019
London

This will be the fourth time that the Society has held a study day on this important and diverse topic. Previously we have explored basic science, oncology, pain/palliative medicine, mechanisms of cancer pain, and the role of the WHO ladder. Join us once again for further discussion as we continue to explore the many facets of cancer pain.

Further details for all our meetings can be found on our events listing page:
www.britishpainsociety.org/mediacentre/events/

Editorial

Why never to put marbles in the bottom of your chunky soup: (or how not to talk to your patients)

Dr Rajesh Munglani *Editor, Pain News*

Pain News
2019, Vol 17(2) 61–63

In 1968, Robson Ballantine, working at BBDO, an advertising agency in New York, had a problem.[2] He needed to photograph Campbell's recently released Chicken & Stars soup. The problem was that the chicken and pasta sank to the bottom of the bowl as it was poured. Ballantine's solution was to put glass marbles in the bowl to displace the chicken and pasta back to the surface. The US Federal Trade Commission, who regulated advertising, took a very dim view of this and in April 1968 launched an investigation into the 'deception' by Ballantine. This culminated in a formal complaint against both Campbell's and BBDO and resulted in a case which eventually dragged in 14 federal judges before it was finally dismissed in 1972.

There can be a similar dissonance between what is anticipated and what is experienced in life. For me, why don't burgers served in fast food outlets *ever* look like their pictures above the service counter?

Are we making similar misrepresentations in our medical practice? Despite all the advances that have been made in medical treatments, we can currently expect a significant proportion of our lives to be lived in ill health. The Office for National Statistics (ONS) reports that healthy life expectancy has failed to keep pace with life expectancy for males and females.[3] The ONS goes on to say, currently males could expect to live 63.1 years in good health (79.7% of their life) and females 63.6 years (76.7% of their life). For each sex, the years lived in 'Not Good' health has increased both in relative and in absolute terms, because life expectancy has risen more quickly than healthy life expectancy.

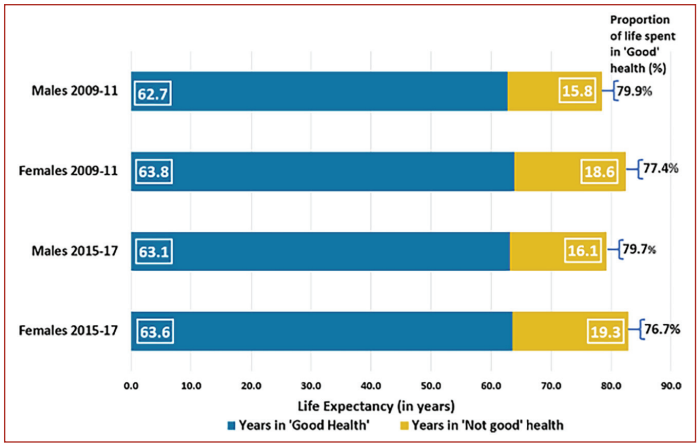
The implications for medicine in general are obvious. We may have (medically) contributed to increased longevity of life of people generally, but seemingly we have not added to the quality of the last two decades of life. So, despite all interventions, all of us can expect to spend the last 20%–25% of our latter years in serious ill health.

Photography by Navada Ra. Copyright free. Pexels.com.[i]

Pain in particular has been shown to be a profoundly negative factor for quality of life (QOL). For many, the suffering of severe pain can destroy any meaningful value from a remaining life. Fayaz et al.[10] recently reported that the prevalence of chronic pain in the United Kingdom ranged from 35% to 50% and that the prevalence in the United Kingdom of moderate to severely disabling chronic pain ranged from 10% to 15%. It was also demonstrated that pain prevalence increased inexorably from 15% in 18–25 year olds to 62% in the above-75 age group. A further study in the United States on over 7,000 elderly (>65 years old) showed that over half experienced pain that was intrusive and 75% reported multisite pain.[11]

It needs to be stressed that the figures given above are currently reported with current levels of access to medical care. This means that these figures are in patients who have access to primary care and medication including nonsteroidal anti-inflammatory drugs (NSAIDS) and opioids, as well as some access to specialist services. The figures are also similar worldwide.

Why never to put marbles in the bottom of your chunky soup: (or how not to talk to your patients)



Healthy life expectancy at birth, years lived in 'Not Good' health and the proportion of life spent healthy, by sex: UK, 2009 to 2011 and 2015 to 2017.
Source: Annual Population Survey, Office for National Statistics reproduced under http://www.nationalarchives.gov.uk/doc/open-government-licence/version/3/

'Any man's death diminishes me, because I am involved in mankind; and therefore never send to know **for whom the bell tolls; it tolls for thee**'.[5] Unattributed Picture. Copyright free. Pexels.com.[6]

In response to our gloomy future, we may attempt to mitigate the effects of ageing by trying low-carb diets, to exercise more, to stress less and to live cleanly. This approach is in fact not new. In 1624, John Donne, the English metaphysical poet and cleric, published his *Devotions Upon Emergent Occasions, and severall steps in my Sicknes*.[4] It is a profound work which covers death, rebirth and the Elizabethan concept of sickness as a visit from God, reflecting internal sinfulness.
The *Devotions* were written in December 1623 as Donne recovered from a serious but unknown illness – believed to be relapsing fever or typhus. As well as being the author of such quotes as '*No man is an island*' and '*and for whom the bell tolls*' (the latter became the title of a book by Hemingway in 1924), Donne expressed his frustration at the inevitability of sickness and pain in the following paragraphs from *Devotions*.

> *We study health, and we deliberate upon our meats and drink and air and exercises, and we hew and we polish every stone that goes to that building – and so our health is a long and a regular work, but in a minute a cannon batters all, overthrows all, demolishes all. A sickness unprevented for all our diligence, unsuspected for all our curiosity – nay, undeserved, if we consider only disorder – summons us, seizes us, possesses us, destroys us in an instant. O miserable condition of man!*
>
> *We beggared ourselves by hearkening after false riches and infatuated ourselves by hearkening after false knowledge. So that now we do not only die, but die upon the rack, die by the torment of sickness – nor that only, but are pre-afflicted, superafflicted with these jealousies and suspicions and apprehensions of sickness before we can call it a sickness.*
>
> *We are not sure we are ill; one hand asks the other by the pulse, and our eye asks our own urine how we do? Is he a world to himself only therefore, that he hath enough in himself not only to destroy and execute himself but to presage that execution upon himself – to assist the sickness, to antedate the sickness, to make the sickness the more irremediable by sad apprehensions, and – as if he would make a fire the more vehement by sprinkling water upon the coals – so to wrap a hot fever in cold melancholy, lest the fever alone should not destroy fast enough without this contribution, nor perfect the work (which is destruction) except we joined an artificial sickness of our own melancholy, to our natural, our unnatural fever? O perplexed discomposition, O riddling distemper, O miserable condition of man!*[7]

Need we go any further to find such an eloquent description of health anxiety?
The medical profession has to be realistic about we can do. Yes we do have antibiotics (for a while longer at least), stem cell injections may be around the corner but pain and senescence[8] is the ultimate fate of us all. The early Stoics urged as to be realistic about outcome which is captured well in the subsequent Latin phrase '**Memento mori**' (remember (that) you will die) especially as a means of considering the vanity of earthly life and the transient nature of all earthly goods and pursuits.

King Solomon considered the same in the work he wrote as he grew old. In Ecclesiastes (8:15), he asked to consider the fleeting glory that is our life and commended the following approach to us:

> *Then I commended mirth, because a man hath no better thing under the sun, than to eat, and to drink, and to be merry: for that shall abide with him of his labour the days of his life, which God giveth him under the sun.*

So, are we guilty of false or unrealistic advertising in medicine also? Even if it is not explicit in our conversations with patients, do we make the assumption that ultimately we can cure or patch up to an extent which allows patients to continue in their mad rush to nowhere in particular (apart from logging their unnaturally bronze smooth and toned photoshopped bodies on Instagram on the way) but ultimately to a destination we will all share?

In 2003, Allen Roses, the then worldwide president of genetics at GlaxoSmithKline (GSK), stated that 'The vast majority of drugs — more than 90 per cent — only work in 30 to 50 per cent of the people.[9] Moore et al.[12] in 2013 wrote in the *British Medical Journal* (BMJ) to 'expect analgesic failure … most of our drugs will work well in a small percentage of people'. Most of our drugs don't work in most people'.

The best predictor of pain is a past history of the same. The natural exuberance and optimism of doctors to over-emphasise the positives and minimise the negative of treatment is no longer acceptable in the new post-Montgomery world of how we talk to and consent our patients. Our previous series of articles in *Pain News* have highlighted that, in 2014, Fiona Godlee[13] wrote about unwarranted optimism and the implication for consent to treatment.

In Pain Medicine, we need to be especially careful about informing our patients about the limits of what we can do. Ultimately, we may only be able to do only a little for most of our patients, but our relationship with them is likely to be long one and building trust about realistic outcomes is important (even if we can't keep following them up, they will invariably be referred back). We have to be honest and help them to expect and indeed accept (Acceptance Therapy?) that the chicken and pasta will invariably sink rather than rise to the top in the real world soup.

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In this issue

Jenny Nicholas

THE BRITISH PAIN SOCIETY
EXPERTISE WHERE IT MATTERS

Pain News
2019, Vol 17(2) 64

SAGE

Welcome to our summer issue of *Pain News* and what an issue it is!

With some returning themes, and new articles to give food for thought, here are a few highlights of what's in store to whet the appetite …

Where we left off...

We continue the discussion on Peripheral Neuromodulation in part 2 of this three-part series titled 'Somatosensory, head and facial pain'. In this article, the authors discuss the role of peripheral modulation for specific clinical indications including brachial plexus stimulation (BPS), stimulation of the lumbar plexus/paravertebral stimulation and nerve stimulation for headache and facial pain.

We also pick up again with Dr Lesley Haines, Consultant Psychiatrist with her second of two articles 'Treating pain and drug dependence: getting it "least wrong"'. In this piece she talks us through difficulties in managing patients with acute and chronic pain with co-morbid opioid dependence.

What's new?...

There continues to be much discussion in the media on medicinal cannabis for pain. In this issue, we have an interesting article from our colleagues overseas in Canada where medicinal cannabis for pain has been prescribed for some time. In this piece 'Medicinal Cannabis in Canada: Potential lessons from across the pond' they share their experiences and potential lessons for the United Kingdom.

In the article, 'An integrated medical, psychiatric and behavioural perspective on CRPS' by George Ikkos, Helen Cohen and Andrew Lucas, the authors reflect upon their experience of the assessment and treatment of complex regional pain syndrome (CRPS) in relation to the current diagnostic criteria and the emerging psycho-neuro-biology and behavioural literature and offer an alternative integrated and dynamic perspective on predisposition to, prevention, evolution and management of CRPS.

This is only a sample of what's to come – happy reading!

From the President

Dr Arun Bhaskar

Pain News
2019, Vol 17(2) 65–66

Dear Friends

I trust this finds you well.

This is my first column in *Pain News* as President of The British Pain Society, a role which I have accepted with great humility. I understand the challenges the Society is currently facing and it is our responsibility to ensure that we as elected and co-opted office bearers work together to address the need of the hour and deliver an organisation that stands up for each and every one of you as members of the largest multidisciplinary society of Pain Medicine in the United Kingdom. I would like to take this opportunity to outline some of my thoughts and vision for the future. I am aware that I am writing this in April and many things would have happened by the time you receive your copy of the summer edition of *Pain News* in June.

First of all, I would like to thank Dr Andrew Baranowski and Dr Heather Cameron who had completed their term of office as the President and Hon. Treasurer of our esteemed Society. Andrew has been very generous in imparting advice and sharing his wealth of experience in dealing with the various machinations of the government and Department of Health. He has advised me of the responsibilities of the office of President. Andrew will continue to support me and the Council in his role as Immediate Past-President. I would also like to thank Heather for her services to the British Pain Society. Prof Roger Knaggs has always been a guiding light with his measured words of wisdom as Hon Secretary and I am delighted that he has been unanimously elected as the Vice-President. Roger is heavily involved in various aspects of the Society and will work hard to serve the best interests of all professionals involved in Pain Management. I would like to reassure you that Dr Ayman Eissa (Hon Secretary), the Interim Hon Treasurer, Dr Ashish Gulve, Roger Knaggs and I will be working with the Council Members to engage with the membership and address the issues relevant to us and the Society.

I would also take this opportunity to thank Dr Tim Johnson and Dr Zoey Malpus who have completed their term of office as Elected Council Members. Tim has played a stellar role in ensuring that the British Pain Society was involved as a stakeholder in all the relevant NICE guidance and also supported the Patient Liaison Committee and other SIGs during his tenure. Zoey has been instrumental in taking forward PMP SIG and was also involved in various other projects. I would like to welcome Dr Amelia Swift (Head of Nursing Education, University of Birmingham) and Mr Martin Hey (Consultant Physiotherapist, Mid Yorkshire Hospitals NHS Trust) as Elected Members of the Council of the British Pain Society. They join a dynamic group of colleagues who are committed to work towards maintaining the standards and ethos of the Society. Currently as Zoey has completed her term, we do not have an elected Council member representing our colleagues in clinical psychology and we should have a co-opted member in place by the time you are reading this edition of *Pain News*.

I had written to you earlier this year about the financial challenges faced by the organisation. You will have have seen from the Annual Report and Accounts, a consistent loss of several thousands of pounds per annum. The major sources of income to the Society are from the membership fees and the ASM. Over the past few years, there has been a steady decline in membership for a variety of reasons and it is our priority to address that; we are stronger when we are together as a multidisciplinary organisation. I would ask all of you to encourage your colleagues to join and become part of the Society; this applies to new members as well as those who were previously members and have not renewed their membership in the past few years. We are also trying to look at increasing our international membership.

One of the issues that has come to our attention is that many of the members are yet to update their banding to reflect their current income. This is having an adverse effect on the membership dues and the Society's income. Members who have the ability to pay should contribute correctly so that it can support our members who are not yet in a position at their early stages of their careers to contribute financially as some of us, but we should continue to encourage future generations of professionals involved in pain clinics and pain research. We are also looking at improving our relationship with our industry

Dr Arun Bhaskar

partners so that we are supported better in our educational and research activities.

The other major income generator as well as the focal point of our annual meeting is the ASM. Following the rather dismal result in 2018 at Brighton, we had conducted a risk assessment against holding it in Belfast in 2019 and Aberdeen in 2020 involving members, industry partners and other relevant stakeholders. Unfortunately, due to various operational reasons, the process was delayed till early 2019, but we managed to put together a meeting in London. I would like to thank Prof David Walsh as Chair of the Scientific Programme Committee as well as Mr Ciaran Wazir for helping organise the ASM in challenging circumstances.

One of the strongest feedbacks we received after the 2018 ASM was to look at changing the format to suit the needs of the delegates and showcase cutting edge research as well as submit clinically relevant topics in the ASM to benefit the vast majority of the membership. This would be fully addressed in the ASM 2020 and the aim is to have an event that would raise the profile of The British Pain Society; you shall be hearing more about this soon as I am writing this before the ASM 2019 and we would like to hear the feedback before planning ahead for 2020. Prof Sam Ahmedzai as Chair of the Education Committee is planning a series of study days and regional meetings throughout this year, covering various topics relevant to pain management.

The British Pain Society has several projects in place for 2019–2020 including Guidelines Development for Cancer pain and also Neuromodulation in Pain. These are in collaboration with the Faculty of Pain Medicine and other relevant organisations in the United Kingdom to have a consensus among various disciplines involved in patient care of people living with pain. In my role as President, I will also be representing the British Pain Society as Councillor for the European Pain Federation and we would be looking at collaborative working with EFIC as well as other global organisations involved in pain management. May I kindly request you to get in touch with me or any one of my colleagues in the Council if you have an idea for a project or anything which you feel that we as a Society should get involved?

One of my personal observations over the past few years is that somewhere along the way a disconnect has developed between the Society and the membership. This disconnect resulted in the lack of engagement between the Society and its members and it is a priority for us to address this issue. We would like you to come forward and get involved in the numerous projects and activities of the Society; if you have something which you feel is important to you and the Society, please bring it forward and we shall do our utmost to consider and support it. Our media team led by Dr Sam Ahmedzai and Dr Stephen Humble will continue to monitor what is happening in the public domain and will represent the views of the Society and the membership in both mainstream and social media platforms. I aim to inform you on a regular basis through various means on the developments and activities of the Society so that you are aware of what is currently happening within the Society and how it is engaging with other stakeholders. More importantly, we would like to listen to your views, opinions and suggestions in these matters.

Finally, I would like to thank on behalf of the membership, the Council and also personally Ms Jenny Nicholas, Ms Dina Almuli and Mr Dylan Taylor who work incredibly hard behind the scenes within the BPS Secretariat. One of the decisions we had taken was to outsource the organisation of the ASM so that the Secretariat can focus on better engagement with the membership as well as devote more resources towards ensuring that the various projects undertaken by the Society are completed in a timely manner. By the time you are reading this, you would have realised that I had been trying to meet and directly take feedback from as many people during the ASM and otherwise, so that I understand what are the priorities I should address for the membership. Needless to say, loss mitigation and ensuring financial stability so that we can continue as the MDT organisation we are is my overarching priority at the moment. I am looking forward to sitting down with my colleagues in Council and work on the various projects and activities. I would like to thank you for all the support and guidance you have given me so far and I would request you to comment, critique and engage in conversation with me and your Council members so that we can deliver a vibrant, dynamic and engaging Society that puts your interests at its forefront.

I look forward to hear from you.

Thank you.
Yours sincerely
Arun

From the Honorary Secretary

Dr Ayman Eissa

Pain News
2019, Vol 17(2) 67

Dear Members,

I would like to introduce myself as the Honorary Secretary of the British Pain Society (BPS).

I have been a consultant in anaesthesia and pain medicine at Sheffield Children's Health trust for about 20 years now, and I practice adult and paediatric pain management on a regular basis. I am interested in pain medicine research and won the DOH research award 2016 in paediatric pain research.

I have had the pleasure of working closely with Prof. Roger Knaggs for the last couple of years, and I am very honoured to follow him as the Honorary Secretary of the BPS. I am sure you are aware of the challenges we are facing as a Society, and I am sure Brexit will add more serious challenges. We have been in intense discussions in the last 12 month, looking back to our performances and difficulties we faced over the years, and I believe we now have a clear vision – led by Arun – where we need to go and where we need to be in 3 years' time. We are making some big changes in our strategy; level of members involvement in running the show, relations with national and international Societies, and our relations with industry and sponsors.

Our emphasis at this stage will be centred on a bespoke ASM that meets the need of the members including all disciplines as an MDT Society, full support to SIGs and regional pain groups and forums, and a close interactive relation with industry and other societies. We have started very promising talks with different spine surgical groups for a possible joint meeting with surgeons in the near future to exchange and debate experiences in spine pain management. We are taking steps to make the BPS a bank of data for all clinical, educational and industrial information that will serve all parties involved.

You are aware of the decline in membership in the last few years; simply we cannot survive with this trend. We are looking to all options and reviewing our policies to tackle this problem at a national and international level.

Before I finish, I would like to thank Andrew, Roger and Heather for their immense input and efforts over the last few years.

BPS is for you by you!

Professional perspectives

Medicinal cannabis in Canada: potential lessons from across the pond

THE BRITISH PAIN SOCIETY
EXPERTISE WHERE IT MATTERS

Pain News
2019, Vol 17(2) 68–71

SAGE

Amol Deshpande * *Toronto Rehabilitation Institute; Assistant Professor, Quality and Innovation, University of Toronto*

Athanasia Chatziperi *Fellow in Chronic Pain Medicine; Department of Anesthesia, University of Toronto; St. Michael's Hospital*

Credit: UrosPoteko.

Amol Deshpande

In October 2018, the Cannabis Act came into effect, thrusting Canada into the spotlight as the first G-7 country to legalise cannabis.[1] In fact, the reality is that access to the medicinal use of cannabis in Canada has been available for almost two decades. Canada's journey may provide some insight to those in the United Kingdom as they attempt to determine the benefits and risks of the medicinal use of cannabis.

In the United Kingdom, the Home Office[2] launched a review into the scheduling of cannabis and cannabis-based products for medicinal purposes in June 2018. Professor Dame Sally Davies, Chief Medical Officer (CMO) for England and Chief Medical Advisor to the UK Government, assessed the therapeutic and medicinal benefits of cannabis-based products and found that there was conclusive or substantial evidence of therapeutic benefit for the treatment of chronic pain in adults and moderate to limited evidence for various other medical conditions. Based on this review, the CMO for

Athanasia Chatziperi

*Amol Deshpande - Scientific Advisory Board, Resolve Digital Health.

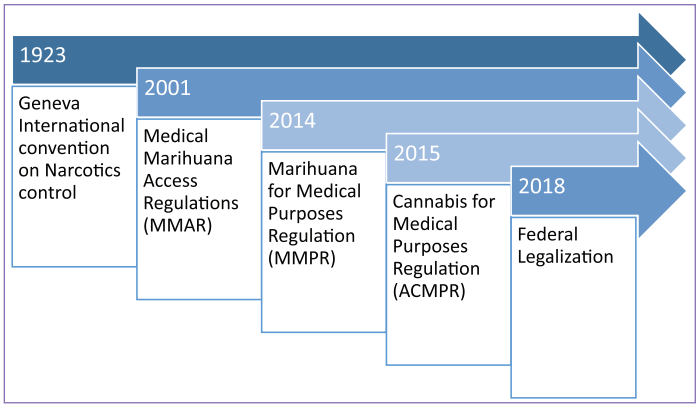
England recommended that the whole class of cannabis-based products for medicinal use be moved from Schedule 1 of the Misuse of Drugs Regulations 2001 (MDR)1 into Schedule 2.[3]

In contrast to the CMO's position, the British Pain Society (BPS) offered an alternative stance.[4] The BPS noted '… clinical studies on cannabinoids for the management of pain conclude that there is no positive evidence to support routine use in pain management'. If the evidence is so clear-cut, how did Canada enable the medical use of cannabis?

Foremost, Canada's journey with cannabis has never been based on the balance of medical evidence but rather has been more akin to a political trek. Banned since 1923, cannabis resurfaced in 2001 under the *Medical Marihuana Access Regulations (MMAR)* after the courts determined it unconstitutional to prevent individuals from using cannabis to control debilitating symptoms.[5] The MMAR set out guidelines for use in compassionate care. With a physician's authorisation, access to cannabis was allowed through either a personal use production licence or Health Canada's approved licenced provider, which offered access to 12.5% THC (Δ^9-tetrahydrocannabinol – the intoxicating and addictive component) herbal cannabis. In 2014, due to the burgeoning number of personal licences in the absence of close monitoring and a growing grey/black market for cannabis, Health Canada enacted the *Marihuana for Medical Purposes Regulation (MMPR)*. These new regulations imposed more stringent controls and were created to treat cannabis more like narcotics.[6] The new regulations included instituting a ban on personal grow licences, opening the market to licenced commercial growers and allowing any physician (not only consultants) the ability to authorise the use of dried cannabis only.

Most importantly, however, MMPR absolved Health Canada from their role as custodian of registering and maintaining a database of authorised cannabis users in Canada. This responsibility was devolved to the commercial licenced producers. The MMPR was short-lived and subject to court challenge regarding the right to grow and the ability to use cannabis in forms other than dried flower (e.g. edibles, oils, etc.). In 2015, Health Canada launched the Access to Cannabis for Medical Purposes Regulation (ACMPR) to accommodate personal grow licences and allow for patients to consume cannabis in a variety of forms. The ACMPR has now been subsumed under the Cannabis Act and stipulates specific regulations for the medicinal use of cannabis. Given the October 2018 legalisation, the future of ACMPR is uncertain, with some health care groups suggesting that its existence is redundant.[7]

The actual medical use of cannabis in Canada has not followed such a steady trend. Under the original MMAR, the



number of registrants grew from less than 500 in 2001 to almost 38,000 in 2014. Since MMPR, however, and leading up to October 2018, the number of registrants for the medicinal use of cannabis exploded by almost 10-fold to over 340,000. Personal grow licences (or those designated to grow) under this regime add an additional 25,000 registrants. For their part, Canadian health care practitioners seem to have readily adopted the notion of medicinal cannabis. Physicians who have ever provided a medical document for a client registration with a licenced provider almost doubled from 9,726 to 18,086 in the short span of 13 months. The overwhelming majority (91%) of patients sought out the use of cannabis for the management of pain.[8] So, has this major increase in cannabis use resulted in better pain management?

Canada has the second highest per capita opioid consumption in the world, second only to the United States.[9] One of the main theoretical reasons cited to support the medicinal use of cannabis in pain medicine is its synergistic or even substitutive effect for opioids.[10] The Canadian experience appears consistent with this narrative. A recent survey of medical cannabis users found 32% substitute cannabis for opioids and up to 40% do so when cannabis is taken in the context of pain.[11] This reduction in opioid use with cannabis has also been demonstrated in larger epidemiological studies.[12] However, while the preclinical data, anecdotal surveys and epidemiological studies are supportive of this notion, clinical studies have so far failed to substantiate this phenomenon.[13] Others have also pointed out, citing equally compelling studies, that this concept may be seriously flawed and even produce the opposite long-term effect.[14]

There is no doubt that the evidence base for the medical use of cannabis in pain management is mixed at best. In Canada, pain physicians have determined that ample evidence exists to consider cannabinoids a third-line intervention in the relief of neuropathic pain.[15] In contrast, the BPS based their decision on a more recent systematic review.[16] The review determined that it is unlikely that cannabinoids are highly effective

medicines for chronic noncancer pain (CNCP), despite documenting significant results favouring cannabinoids for studies reporting outcomes of 30% pain relief in the context of neuropathic pain. These results, as the authors stated, were similar to a recent Cochrane review assessing cannabinoids in neuropathic pain.[17] The Cochrane authors, however, had a more measured conclusion, stating that there may be at best a small effect for a few neuropathic pain patients.

How is it that similar high-quality systematic reviews, both rightly acknowledging the low-quality of primary studies, can reach, albeit not drastic, but arguably distinct conclusions? As an observation, the authors with the more conservative conclusion were associated with institutions related to substance use disorders, while the other authors were linked to pain medicine. We suggest that academics may choose to review the extant evidence base through the lens of their respective patient populations. In politics, the notion of '… where you stand is a function of where you sit' has been coined 'Miles Law'. Maybe this concept exists in medicine as well? At this juncture, the only aspect that all academics and clinicians seem to agree on is that more high-quality primary research is required.

At the crux of the debate, in addition to the lack of high-quality evidence for efficacy, is the long history of documented potential for harm in the context of recreational cannabis use.[18–21] From the literature, studies based on recreational use have identified a 9% chance of developing cannabis use disorder, an increased risk of psychosis and a twofold increase in road traffic accidents.[22] Canadian rates of cannabis substance use are in line with these, noting that risks increase with earlier age of onset.[23] Given that Canadian guidelines and regulations espouse a minimum age of 18 (or older) for the use of cannabis, it seems reasonable to assume that this rate of substance use disorder may at worst represent an upper boundary for authorised users in the medical context. In terms of psychosis, some authors argue that many studies fail to account for reverse causality and possible confounders, overestimating the relationship to cannabis. The same authors estimate that in Canada 2%–3% of psychosis cases can be attributable to cannabis use.[24] The ongoing challenge with any of these epidemiologically based studies is that it is difficult to determine whether the level of risk with recreational use will directly translate to medical cannabis users based on amount, type and frequency of use.

One of the main challenges still unresolved is the use of cannabis and its relation to road traffic accidents. In 2000, more than one-third (34.8%) of fatally injured drivers tested positive for alcohol compared to just 15.9% who tested positive for cannabis. However, from 2010 to 2015, the percentage of fatally injured drivers who tested positive for alcohol decreased (from 37.6% to 30.9%), while the percentage of those drivers who tested positive for cannabis increased (from 15.9% to 20.9%).[25] It is difficult from these data to determine the nature of cannabis use (i.e. medical or recreational). A direct cause and effect relation to cannabis is also spurious since many individuals test positive for multiple psychoactive drugs. A group (>45 years old) more likely to access cannabis for medical use, however, does highlight a cautionary note. Already testing positive in fatal crashes for central nervous system (CNS) depressants and opioids, cannabis may only contribute to their fatality.[25] Irrespective of cause, the trend is highly concerning.

Despite several legal challenges since 2001, Canada has had a mostly uneventful two decades of medicinal cannabis use. At the same time, several mis-steps have unfortunately led to a strong commercial interest and persuasive public opinion overseeing the cannabis agenda. Health Canada, in abdicating its custodial role and the opening of doors to recreational use, have likely tipped the balance away from a more measured approach to properly understanding the nuances of cannabis for medical use.

The basic science of cannabis and cannabinoids has been well studied.[26] On the other hand, the complexities of cannabis chemistry in clinical medicine have been well documented. O'Shaughnessy introduced the medical use of cannabis to Western medicine more than a hundred years ago, absent the scientific knowledge we possess today. His early work to treat a variety of medical conditions with cannabis was met with mixed results, perplexing physicians as far back as 1883.[27] The intricacies of multiple bioactive cannabinoids, terpenoids and flavonoids may continue to limit the impact of traditional high-quality evidence, typically designed to assess single molecules. Currently, there is ample noise, but also occasional signals for the potential benefits for the medicinal use of cannabis. The BPS, Faculty of Pain Medicine and the Royal College of Physicians are in a position to take a more proactive approach to help guide the public and policymakers and advocate for the proper and safe design of a system to monitor and investigate the medical use of cannabis as we enter uncharted waters.[28] Globally, countries around the world in addition to Canada are acquiescing to the public's demand to access the potential benefits of cannabis. It is hard to imagine that the United Kingdom will be able to hold out for much longer. The tide is coming, whether we accept it or not. But until then, our task in pain medicine should be to utilise novel tools, such as big data collected through national registries, to continuously gather and objectively evaluate all types of evidence to better understand the role that therapeutic cannabis *could* play in pain management, even if it is only for a few.

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THE BRITISH PAIN SOCIETY
EXPERTISE WHERE IT MATTERS

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SAGE

Assisted dying and the Hippocratic Oath: the relief of suffering is our duty of care

Daniel Sokol *Medical Ethicist and Barrister*

On 10 December 2008, a documentary by John Zaritsky showed the suicide of Craig Ewert, a 59-year-old man with motor neurone disease. We see Craig in an apartment in Zurich, surrounded by his wife and a social worker, switch off his time-controlled ventilator. Unable to press the switch with his fingers, he does so with his teeth. The social worker hands him a potion of sodium pentobarbital. 'Mr Ewert', he says, 'if you drink this you're going to die'. Craig sucks the liquid through a straw and grimaces. He asks for apple juice to wash away the unpleasant taste. At his request, the first movement of Beethoven's Ninth Symphony resounds around the room. 'Thank you', he says after finishing the cocktail. The camera is fixed on Craig. Gradually his eyes close. He falls asleep.

Since then, other high-profile 'right to die' cases have followed, notably Tony Nicklinson, who had locked-in syndrome, in 2014, and Noel Conway, the retired lecturer who suffered from motor neurone disease, in 2017. Recently, Geoff Whaley, another terminally ill man with motor neurone disease, travelled from the United Kingdom to a Dignitas facility in Switzerland to end his own life. This comes as The Royal College of Physicians is polling its members to gauge their views on assisted dying.

Of course, good palliative care can alleviate most types of pain and suffering; of course, assisted suicide must be a last resort after all reasonable alternatives have been considered; of course, procedural safeguards must be in place to avoid abuse and exploitation; of course, medicine is principally concerned with saving or prolonging life and not helping patients to die. However, even state-of-the-art palliative care is of limited effectiveness in rare cases (and targets the physical pain, not the existential anguish); all available alternatives may have been exhausted; procedures may be robust enough to indicate a patient's autonomous choice and suffering; and death can be a friend rather than a foe to the desperately sick patient.

The BMA has long been opposed to assisted dying because it violates 'the ethics of clinical practice, as the principal purpose of medicine is to improve patient's quality of life, not to foreshorten it'. Amy Proffitt, the Honorary Secretary of the Association for Palliative Medicine, believes assisted dying 'changes Hippocratic duties of doctors and goes to the heart of medicine'.

The Hippocratic Oath instructs doctors 'I will not give a drug that is deadly'. Contrary to first impressions, this is not in fact a prohibition of euthanasia. The lexicographer Littré and the physician–ethicist Miles argue instead that this was in fact a prohibition against doctors collaborating in murder. Political murder was common in Ancient Greece and doctors were sought after for their knowledge of poisons.

All doctors recognise that there are conditions worse than death. Every day, doctors make decisions knowing that they will shorten their patients' lives such as withdrawing life support from a terminally ill patient or deciding against an operation to remove a brain stem tumour because life after surgery would be terrible. Yet, most would not consider these actions to be unethical.

A key purpose of medicine is the relief of human suffering and, for many working in the field of pain, integral to their duty of care. Medical technology, ever more sophisticated, has allowed us to prolong life, but it can also unwittingly extend suffering. As the saying goes, 'just because we can, doesn't mean we should'.

The doctor who administers a fatal injection to a patient with end stage MND, who has clearly expressed a wish for an earlier death, is relieving human suffering and in my view, this doctor should be no more criticised than the doctor who switches off the ventilator from a patient with full capacity who decides that 'enough is enough'. Both are acting in accordance with the Hippocratic commitment to benefit the sick and alleviate suffering. So too was the medic in a true incident recounted to me by the late Rick Jolly, OBE, a trauma surgeon serving in the Falklands War.

An Argentine prisoner of war was carrying an unexploded bomb when it exploded and engulfed him in flames. A British medic tried to reach him but could not get close due to the intense heat. After 5 minutes, the burning prisoner was still moving. There was no longer any hope of survival. The medic grabbed his rifle and shot him four times to end his suffering. That act was not, in my view, contrary to the ethics of medicine.

The wrongness of medical assisted dying, if it is wrong, must be found somewhere other than the 'fundamental ethics of medicine'. Its use in certain circumstances may be entirely compatible with the primary duty of medicine to relieve suffering.

Dr Daniel Sokol is a medical ethicist and a barrister specialising in clinical negligence at 12 King's Bench Walk Chambers, London. He is an award-winning columnist for the *British Medical Journal* ('Ethics Man') and the author of three books on medical ethics and law. He has sat on various committees, including those of the Ministry of Defence, the Ministry of Justice and the Royal College of Surgeons. He has conducted advisory work, training, conference moderation and after-dinner speaking for leading companies and charities, and lectures in the United Kingdom and internationally on medical ethics and law.

This piece is based on the chapter on assisted suicide in his new book, 'Tough Choices: Stories from the Front Line of Medical Ethics' (Book Guild, 2018) and also 'Assisted dying is compatible with the Hippocratic Oath' published on 14 February 2019 (https://blogs.bmj.com/bmj/2019/02/14/daniel-sokol-assisted-dying-is-compatible-with-the-hippocratic-oath/).

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His book 'Tough Choices: Stories from the Front Line of Medical Ethics' (Book Guild, 2018) is now available.

Cannabidiol, the law and the evidence

THE BRITISH PAIN SOCIETY
EXPERTISE WHERE IT MATTERS

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SAGE

Marcia Schofield *Specialist in Pain Medicine*

You may have noticed more and more patients acting a bit shifty in Pain Clinic. They shuffle in, stare at the floor a bit and finally look at you and ask The Question:

So what do you think about this cannabis oil, Doc?

Cannabidiol is a phenomenon. Research carried out by the Brightfield Group estimated the market was worth about $170 million in 2017. This year, the market is estimated to be worth $512 million, with $2.1 billion predicted by 2020. Compared with the market for other supplements used in pain, there is a 'green rush' to invest in cannabidiol; and in allied industries, such as growing equipment, packaging and filling equipment.

St John's wort was the last herb to be the subject of a boom-and-bust craze – with worldwide consumption reaching 1,000 tonnes at its height on slim evidence of effect on depression in small trials. However, fashions come and go: last year, the worldwide consumption was only 300 tonnes, with many manufacturers holding large quantities of 'old stock' that they were left with, and the market collapsing.

Compared with glucosamine, another supplement shown to be moderately effective in some musculoskeletal pain, the worldwide market is growing by only 11%. Although the 2025 market for glucosamine is predicted to reach a worth of $1.2 billion, this pales into insignificance beside the current craze for cannabidiol.

So what is cannabidiol and what is all the fuss about?

The short answer is that it is a constituent molecule produced by the cannabis plant (as well as the hemp plant). In contrast to delta-tetrahydrocannabinol (THC) which is known to be an agonist at CB1 and CB2 receptors (as well as possessing psychoactive properties), cannabidiol does not seem to be a direct agonist. The best guess is that cannabidiol is an allosteric modulator at both CB1 and CB2, with effects on at least 20 other G-protein receptor systems throughout all mammalian species.

Patients have used cannabidiol for a range of conditions for which western medicine has disappointing efficacy. The

Credit: Yavdat.

research on cannabidiol suggests that it is effective in reducing seizure frequency in some difficult childhood epileptic conditions in humans; has some neuroprotective effect in experimentally induced stroke in animals; may be helpful for spasticity and pain in both animals and humans; and may help with other neurological and psychiatric conditions. However, evidence is sparse and comes from either animal studies, small phase-2 trials, or anecdotal or registry information.

The difficulties are that international regulation aimed at stopping the trafficking of cannabis for recreational use has stymied further scientific exploration of the cannabinoids. What little research has been done has been subjected to heavy-handed regulation in all major research centres (the European Union, North America and the Far East) with the result that very little research has been done on humans, and virtually none on the various medical conditions that are claimed to be ameliorated or cured by patient anecdote, including autism, attention-deficit disorder (ADD), schizophrenia and cancer metastases.

Until recently, cannabidiol had the status of a food supplement. As the regulatory framework for foods is much

less strict than that for medicines, companies supplying cannabidiol products were not compelled to publish analytical data, percentages or sources of their cannabidiol. In many cases, they are also not required to list excipients, preservatives, colourings or flavourings. Products claiming to be cannabidiol were legal, as long as they contained less than 0.2% of THC and were not sold with claims of medical efficacy.

As cannabidiol is available in various oral, sublingual and topical preparations, it is impossible to tell what dose of cannabidiol is being consumed. It would also be impossible for patients to tell how much or whether their cannabidiol product even contained the same concentration as the previous batch.

Vaping is also widespread. A recent Food and Drug Administration (FDA) analysis of 87 different vaping products claiming to be cannabidiol found that only 30% of the products bore any resemblance to the claims made on the packaging or website, with some containing entirely synthetic cannabidiol and one containing industrial grade propylene glycol (which becomes formaldehyde at vaping temperatures and is unsafe for human consumption).

Research on cannabinoids for medical use in humans has been dominated by one pharmaceutical company (GW Pharmaceuticals) which has produced two FDA-approved (and Medicines and Healthcare products Regulatory Agency (MHRA)-approved) whole-plant extracts for medical use. Basic science exploration has also been limited: mostly to academic Israeli laboratories, which have been subjected to a much less restrictive regime. Currently, no other country has managed to duplicate the trials due to prohibitions on the supply of cannabinoids. A few other companies (Canopy, Zynerba and Insys) are currently in phase-2 and phase-3 trials for a variety of conditions. Other basic research has been limited by the availability of raw materials, consistency of supply and the physicochemical properties of cannabidiol (the oral bioavailability is less that 10% in some studies).

The toxicity information obtained from the few human trials suggests that although efficacy may not be supported, there is very little evidence of harm. Doses up to 6g daily have been reported in healthy volunteers without serious adverse effects. By contrast, most of the commercially available products contain less that 10mg per oral dose.

Sales of cannabidiol may become more difficult over the next 12 months if the United Kingdom decides to adopt the EU's designation of cannabidiol as a 'novel food'. Sales would have to be stopped or restricted until research could prove that cannabidiol has no unpredictable or dangerous effects when used as a food supplement. However, the large players such as Philip Morris, Coca Cola, and large food and beverage companies are investing heavily in cannabidiol-containing products, cashing in on the desire for the 'naturopathic food-as-medicine' reputation of cannabidiol.

So where does this leave the patient who is taking CBD oil which they have bought on the local high street or from the many Internet suppliers? Some patients are adamant that their oil is the only thing that has helped them or their family member and are loath to discontinue what they see as an effective treatment. These patients can and do get admitted to hospital, and the clinician may have to face angry patients or relatives who are determined to continue their cannabidiol preparations.

In our hospital, we have adopted a common-sense approach. We have a written policy concerning the self-administration of over-the-counter medicines and supplements. While keeping a close eye on the legal position of cannabidiol, we have not banned patients using the supplements. We have put together as much information for patients as we can summon and have urged caution as no one is aware what the potential interactions with common medicines may be (as they have not been sufficiently studied). We will not supply cannabidiol preparations as they do not have the status of medicines (and are therefore not MHRA-approved and not prescribable). We have, and do, pursue individual funding requests for Sativex (and will certainly do so for Epidiolex post-authorisation) for conditions for which it has good evidence. We comply with stipulations about setting and satisfying outcomes and tell patients that continued prescribing or Sativex will require hard evidence of effectiveness. We have and do discontinue these medicines if they are not tolerated or effective.

As clinicians, we are reluctant to prescribe or recommend medicines which we are unfamiliar with. Cannabis and cannabinoids appear to occupy a very distinctive place in the hearts and minds of patients – as traditional folk herbal remedies – and hope stems from decades of activism to deregulate, legalise and legitimise them as medicines. However, where legislation has been slow to react, the enormous financial opportunities to be derived from the exploitation of cannabinoids may finally see reputable science. Indeed, trials registered on ClinicalTrials.Gov have grown from less than 20 in 2005 to more than 160 this year. Hopefully, we will soon have the science to support patients' decision-making.

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Professional Perspective

To EBM or not to EBM (or how to avoid making a hash of it?)

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EXPERTISE WHERE IT MATTERS

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SAGE

Jenny Jessop *Retired Consultant in Pain Medicine*

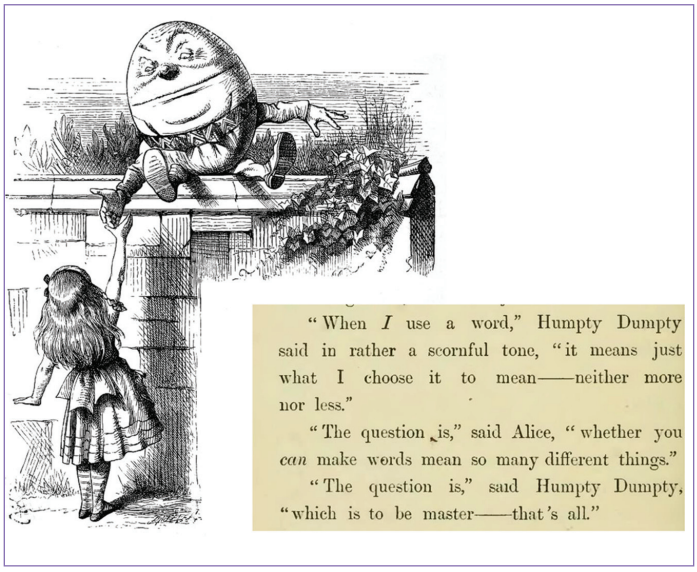

Image by John Tenniel.[1,2]

> 'When I use a word', Humpty Dumpty said in rather a scornful tone, 'it means just what I choose it to mean -neither more nor less'.
>
> Alice Through The Looking Glass; Lewis Carroll.

For as long as I was involved in chronic pain, there was a proud tradition of consultants being prepared to try almost anything that might just help their patients, and for almost as long as that there was, in parallel, a push to make the specialty a lot more evidence based. Pain is a relatively recent specialty, so perhaps a little less hidebound than the ones that perceive themselves as 'senior', and it can credit itself with being much more focused on the evidence for what they offer patients than much of the rest of medical practice. However, patients with chronic pain are very complex compared, for example, to a patient with acute appendicitis, and patients with very similar types of pain may have greatly differing blends of the elements of the biopsychosocial soup, which means they may respond very differently to the same treatment.

This has meant that chronic pain specialists have, perforce, retained a degree of pragmatism when treating their patients. This Gordian Knot has been tightened somewhat by the recent changes in legislation which were designed to make it easier for doctors to prescribe medical cannabis for their patients. It is interesting to explore the tension between evidence-based medicine (EBM) and the desire to try anything reasonable to help a patient in that very contentious context.

Which treatment a patient ends up receiving involves three groups of stakeholders. The first is the government and its agents, the commissioners of service. The second is the doctor and his peers, and the third is the public, of whom the index patient is usually, but not always, the most important person. Each of these three groups of stakeholders may have more than one agenda, and these heavily influence what happens. The commissioners usually present themselves as bearing down rigorously on clinical and cost effectiveness, but because their masters, the government, need to get re-elected, they may also be under pressure to deliver treatment in a way that wins votes. Doctors generally want to be practising in a way that is considered to be excellent by their peers, because nobody wants to end up being struck off or sued. However, they are also fiercely protective of their clinical freedom and their ability to exercise it when treating patients, even if that means deviating from practice that is strictly evidence based. However, they are often pretty defensive with patients who have read enough to consider themselves experts courtesy of Dr Google and Facebook. Patients are generally looking for someone to fix them, are unhappy when that isn't possible and outraged when they have read an article saying there is a good treatment that doctors wilfully refuse to prescribe or the government will not let them prescribe.

Many of these agendas could, in management terms, be considered to be holes in the Swiss cheese, and we are taught that disasters generally occur when the holes happen to line up. And when the medicinal cannabis needed to treat a child with intractable epilepsy was seized by the Home Office, the holes lined up in earnest. No political party can afford to be denying treatment to a sick child, especially when there is evidence that it works. It is a bad look in terms of getting re-elected, so they fell into the trap of enacting legislation on the hoof. Enabling doctors to prescribe medicinal cannabis conjured up visions of happy epileptic children and their parents and, of course, of plenty of votes at the next election. What they failed to think about properly was the prospect of a sizable proportion of the

Alexander cuts the Gordian Knot by Jean-Simon Berthélemy (1743–1812).[3]

population who experience chronic pain seeing this new legislation as the answer to all their prayers.

The reason they failed to think about this was that they spoke to the Royal College of Physicians, but forgot to speak to the appropriate experts in the chronic pain field. Had they done so, not only would they have been warned about the tsunami of demand that was inevitable but they would also have been advised not only that the evidence for the efficacy of cannabis in treating chronic pain was extremely sparse but also that the known risks were significant. The specialty had to resort to a letter in The Times, signed by several hundred consultants, to get the point across. By this time, the expectation of patients had been raised to undeliverable levels. Consultants described doing pain clinics where every single patient had asked about being prescribed medical cannabis. Patient groups were vociferous in condemning doctors for denying them the answer to their prayers and, in places, this turned ugly. A politician in the Channel Islands went so far as to suggest that doctors who refused to prescribe cannabis for pain should lose their jobs, a direct challenge to the local consultant who dared to say that he was not prepared to prescribe a drug in the absence of evidence of its therapeutic value and safety. To the credit of the specialty, they have stuck to their guns and the government has had to listen. This has incensed patient pressure groups and left politicians nervous about the reaction in their constituencies. Hopefully, one good outcome is that the specialty has now made people realise that what they say is worth hearing and that it is less trouble for politicians in the long run to engage with them than to ignore them!

There is, of course, a bit of irony to all this. The specialty has come out in public insisting that the government should not make them prescribe outside the evidence base. Back in the clinic, however, there is a persistent belief that individual consultants should be free to prescribe a treatment on the basis that it provided 5 months of relief for a particular patient of theirs back in 2003. However, I suspect most would only deviate from the evidence base on the basis of their own experience, and not that of their patients. Hence, there was little concerted resistance from consultants when a large number of patients who used facet joint injections or acupuncture successfully as part of their chronic pain management strategy had that treatment withdrawn by commissioners, even if there was nothing else that worked for them. In contrast to cannabis, the risks from acupuncture done in a National Health Service (NHS) setting with short, fine, single-use needles is almost entirely confined to bruises and faints, but this has not stopped some commissioners from refusing to fund it. In this case, the individual experience of patients seemed to count for virtually nothing, either to commissioners or to many clinicians.

This leaves me on the horns of a dilemma. It is great that consultants care enough to strive to provide something that, from their experience, might just help individual patients to manage their chronic pain, even though the evidence base is unconvincing or absent. It is also good to have witnessed the growth of chronic pain as an evidence-based specialty and heartening to see that when consultants argue in unison from the evidence base, government can be forced to listen, as they were over medical cannabis. However, the next time the specialty tries the same thing, particularly if it causes the government embarrassment, then expect one of the stakeholders (or their journalistic friends) to take a close look at how closely consultants stick to the evidence base in their everyday practice. Finding that many may be prepared to provide a treatment on the basis of anecdotal evidence only could make for an interesting conversation. What's sauce for the goose is, as they say, sauce for the gander, and if patients can be recommended obscure injections or drugs, with limited evidence of efficacy for their various pains, then why cannot they have cannabis instead? Or, even more so, a relatively harmless treatment such as acupuncture, that was previously provided on the NHS and that worked for them? Answers on a postcard, please …

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An integrated medical, psychiatric and behavioural perspective on CRPS: reflections on publication of 'Complex Regional Pain Syndrome in Adults in the UK: guidelines for diagnosis, referral and management in primary and secondary care'

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Introduction

Publication of the second edition of 'Complex Regional Pain Syndrome in Adults: UK Guidelines for diagnosis, referral and management in primary and secondary care' (*'the guidelines'*)[1] offers an opportunity to reflect on the Complex Regional Pain Syndrome (CRPS). In this article, authors consider the contribution of medical, psychiatric and behavioural factors in relation to diagnosis, psycho-neuro-biology, prevention and management.

Issues considered here include the following:

- The role of the core diagnostic criterion of disproportionality of pain in CRPS;
- The place of CRPS within the broad group of chronic pain and related conditions;
- The possible role of adverse childhood experiences (ACEs) and current psychosocial stressors as predisposing or aggravating factors;
- The crucial role of patient and clinician behaviour in preventing, understanding and treating CRPS.

The authors emphasise issues that may aid understanding of the dynamic development of symptoms and clinical presentation of CRPS; also, the importance of patient-clinician communication and their behaviour in understanding, preventing and managing the condition.

Diagnosing CRPS

CRPS is defined by 'disproportionate' pain and (1) sensory, (2) vasomotor, (3) sudomotor/oedema and (4) motor/trophic changes. Sensory symptoms refer to allodynia, hyperalgesia and hyperaesthesia; vasomotor symptoms to temperature asymmetry and/or skin colour changes and/or skin colour asymmetry; sudo-motor/oedema to oedema and/or sweating changes and/or sweating asymmetry; and motor/trophic to decreased range of motion and/or motor dysfunction (weakness, tremor, dystonia) and/or trophic changes (hair/nails/skin). The 'Budapest Diagnostic Criteria for CRPS' ('Budapest Criteria'), preferred by the International Association for the Study of Pain (IASP), are commonly used in practice (Box 1).

As *the guidelines* make clear, some symptoms and signs of CRPS are very common after any injury (see Figures 1–6 for an illustration of such a case) and commonly found after limb surgery. They constitute part of the normal bodily response to injury which in the vast majority improve with time and would not be considered to be CRPS according to the current criteria.

Most patients who go on to develop CRPS according to the *Budapest Criteria*, however, can also be reassured that they will recover within the first year. In the smaller number of cases where CRPS will evolve into a chronic debilitating condition, research has documented both peripheral limb and central nervous system (CNS) biological changes.[3] These may be

Box 1 (reproduced from Goebel et al.[1]).

Table 1 Diagnostic criteria for CRPS ('Budapest criteria')21 (A–D must apply)†

A) The patient has continuing pain which is disproportionate to any inciting event	☐
B) The patient has at least one sign in two or more of the categories	☐
C) The patient reports at least one symptom in three or more of the categories	☐
D) No other diagnosis can better explain the signs and symptoms	☐

Category		Sign (you can see or feel a problem)	Symptom (the patient reports a problem)
1. 'Sensory'	*Allodynia* (to light touch and/or temperature sensation and/or deep somatic pressure and/or *hyperalgesia* (to pinprick)	☐	Hyperesthesia does also qualify as a symptom ☐
2. 'Vasomotor'	Temperature asymmetry and/or skin colour changes and/or skin colour asymmetry	If you notice temperature asymmetry: must be >1°C ☐	☐
3. 'Sudomotor/ oedema'	Oedema and/or sweating changes and/or sweating asymmetry	☐	☐
4. 'Motor/trophic'	Decreased range of motion and/or motor dysfunction (weakness, tremor, dystonia) and/or trophic changes (hair/nail/skin)	☐	☐

considered as a maladaptive escalation of the original acute combined 'mind-body' response. Such biological changes have also been reported with no injury or surgery at all, though this is a far less frequent occurrence.[4,5]

The *sine qua non* for diagnosis of CRPS is the presence of pain which is disproportionate to any inciting event and not better explained by any other diagnosis. Neither the *Budapest Criteria* nor *the guidelines* advise how this cardinal criterion may be judged. We would like to highlight the following troubling issues in relation to this:

1. There is a risk that this criterion may open the door to poorly evidenced and understood clinical value judgements about the objective basis of patients' pain levels. We know, irrespective of CRPS, that tissue damage is not related in a linear way to pain experience.
2. Even if one was to accept such a linear relationship, there is evidence that increased initial intensity of pain experience and fear of pain may be risk factors for the development of chronic CRPS.[6–9]
3. In assessing this criterion, clinicians should establish whether there is or has been prior use (or misuse) of significant doses of opiates or other illicit drugs that could lead to a tolerance effect on current analgesics, or to opiate-induced hyperalgesia.[10]
4. There is also the question of whether the pain may be disproportionate to the original injury or other stimulus but not disproportionate to subsequent, possibly ongoing, adverse experiences in clinical care (e.g. recurrent surgery or injury to the same site or swelling because of persistently maintained overtight cast).
5. Even if disproportionate to the injury itself, the criteria and guidelines do not comment on whether the pain may be proportionate to any pre-existing and ongoing physical or psychiatric disorder and/or emotional trauma associated with the inciting event.

Little is known about the incidence or relevance of other pre-existing chronic widespread pain disorders to a subsequent diagnosis of CRPS. It has been suggested that fibromyalgia and CRPS may share a common pathway such as neurogenic neuroinflammation[11] and that fibromyalgia, repetitive strain injury and CRPS share more common denominators than just pain.[12] Fibromyalgia may be an association with and predictor of CRPS after distal radius fracture.[13,14] A retrospective analysis of 190 patients diagnosed with CRPS at a tertiary pain medicine referral centre reported a >10% incidence of widespread pain, which all patients considered as an important factor affecting their quality of life.[15] Furthermore, the longer since the inciting

Figure 1. 62-year-old female patient with chronic CRPS affecting the right lower leg. Eight years previously, she underwent surgery to straighten the second toe. This was complicated by infection, osteomyelitis and subsequent amputation of the digit. She states that she has never looked at it since. She developed symptoms and signs of CRPS shortly afterwards. Over time, she has had increasingly severe body dysmorphia with inability to look at, touch, wash or interact with the limb. She has neglect-like symptoms which are described in CRPS, and took to hiding the limb under two layers of knee length stocking socks which are worn constantly. The limb is never exposed to others including health professionals. Her husband has seen it on a few occasions and she hides it from him. There is a history of severe sustained childhood abuse. Figures 2–6 are also from the same individual (permission granted by patient for all figures).

Figure 2. Patient's drawing of her feelings towards the CRPS affected limb.

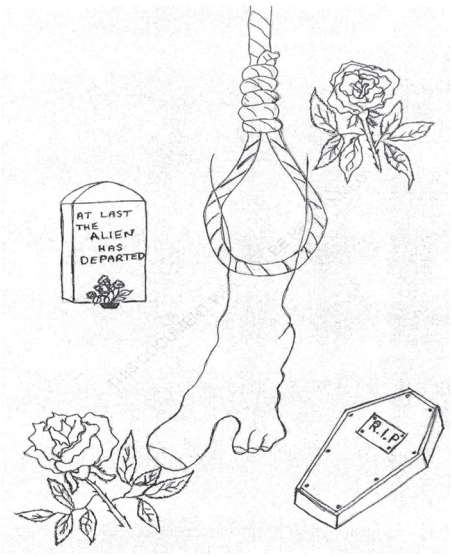
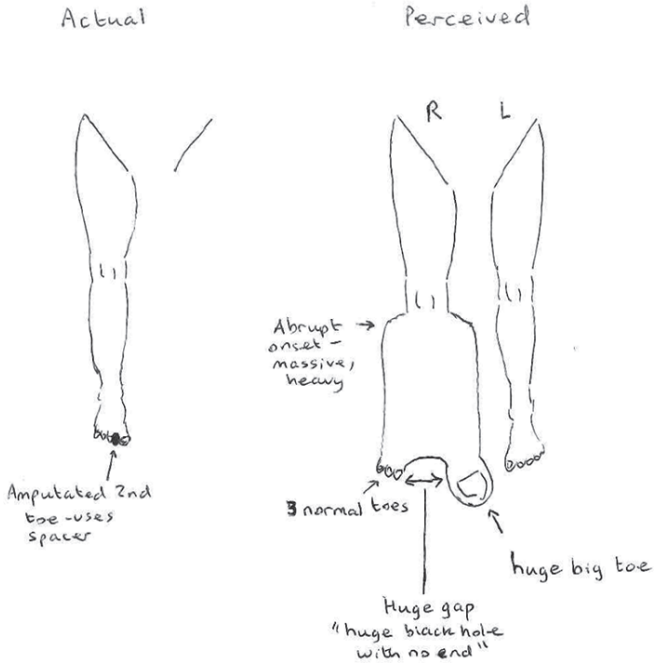


Figure 3. The Bath Body Perception Scale for CRPS[2] includes a section where the patient is asked to describe the perception of their limb with their eyes closed. This is a diagrammatic representation of her description (HC).



event, the more likely CRPS patients are to report symptoms suggestive of centralised pain (assessed by utilising the 2011 Fibromyalgia Survey Criteria).[16]

Both history of psychiatric disorder and childhood adversity, even in the absence of psychiatric disorder at the time of injury, may predispose to increased perception and fear of pain; also, to increased difficulties in achieving patient-clinician concordance in care, particularly in the case of personality disorders.[17–19] There is sparse literature in CRPS, suggesting an association with early traumatic experiences and somatoform dissociation;[20] stressful life events in children[21] and adults;[22] and increased post-traumatic stress disorder (PTSD) prevalence.[23] Gupta and Gupta[24] found that stressful life events

Figure 4. The pseudo neglect and constant covering of the skin has caused poor skin condition and susceptibility to recurrent acute severe episodes of cellulitis/erysipelas. This is a picture during one such episode. Her body dysmorphia is such that even during severe episodes of infection, she will not attend Accident and Emergency or her GP for fear of having to expose the limb. She has been able to develop enough trust over time to show the leg to two members of staff (A.L. and H.C.) only.

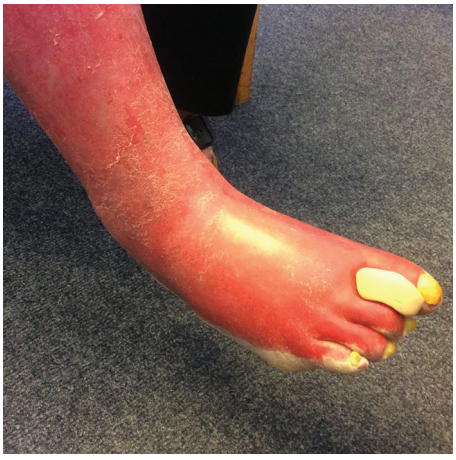


Figure 5. The episodes of severe cellulitis/erysipelas are followed by marked skin desquamation.

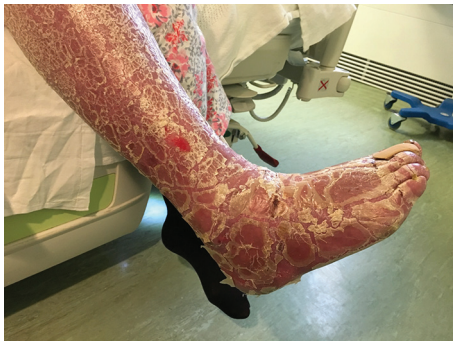
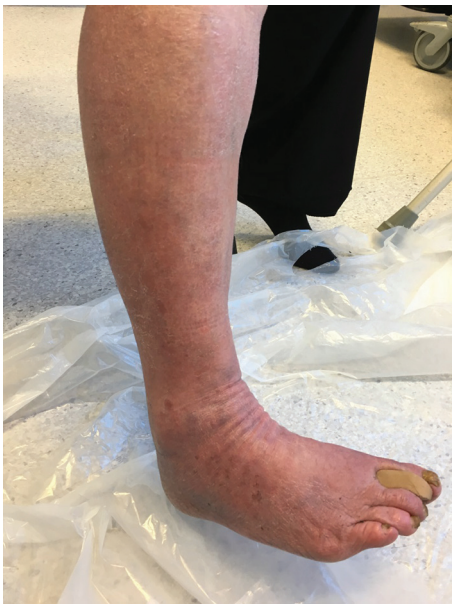


Figure 6. There have been brief occasions between infections when the limb is in reasonable condition, as below, post prolonged IV antibiotic treatment. Unfortunately, the coverage of the limb, poor skin condition and thus vulnerability to infection continues.



in a community-based non-clinical (therefore non-CRPS too) population sample correlated with a higher frequency and severity of cutaneous sensory symptoms and remained significant after the possible confounding effect of psychological factors on cutaneous symptoms was factored out statistically. The total number of major life events experienced over the previous 6 months correlated with the severity of the individual cutaneous symptoms ($0.22 < \text{or} = \text{Pearson } r < \text{or} = 0.41$, $p < 0.001$) and with the total cutaneous symptom severity score (sum of all cutaneous severity ratings) (Pearson $r = 0.40$, $p < 0.001$). This correlation remained significant after the possible confounding effect of psychological factors on cutaneous symptoms was partialed out statistically (partial $r = 0.19$, $p = 0.001$).

There is very limited research as to whether pre-existing or somatic symptom disorder, or somatoform disorder[20] and personality disorder[25] predispose to CRPS; there is none that we are aware of on whether the recently proposed bodily distress syndrome[26] predisposes to CRPS, though there are well-known associations between these mental disorders, adult pain and ACEs.[17] *The guidelines* working group chair (Goebel A, personal communication) and the present authors agree the importance of eliciting a history of distressing events or life situations (singular or cumulative) in patients with chronic CRPS, despite the lack of current evidence specifically or exclusively linking such events to the CRPS onset.

CRPS: the brain, mental disorder and ACE

The guidelines do not discuss in detail the nature and significance of the CNS changes found in CRPS. This is appropriate, as they

aim at clinical identification and management of patients rather than a neurobiological or psychological review. Lack of detailed discussion is also justified by the fact that research conducted in this area is of variable quality and the findings inconsistent. In this uncertain[27,28] and constantly evolving field, the present authors share the view that any changes that may be reliably demonstrated in the future may transpire to be adaptive CNS responses;[29] also, may be consistent with what we know about adaptive CNS responses to ACEs (emotional/physical/sexual abuse or neglect) or adult trauma or stress.[30]

Teicher et al.[31] have reviewed what is known about brain imaging in young and adult people who have suffered 'childhood maltreatment'. His formulation is that such maltreatment is associated with neurobiological changes and that these are specific to the characteristics of the victim as well as timing, duration and nature of maltreatment.

For example, boys suffering persistent verbal abuse at a certain age will manifest different changes compared to girls suffering sexual abuse at another age and for a longer or shorter time. Subject to such differentials, Teicher argues that maltreated children demonstrated the same brain changes whether they developed childhood mental disorder or not. This has led him to suggest that the changes are adaptive rather than pathological. Although adaptive at the time of maltreatment, he also suggests that they expose maltreatment survivors to later vulnerability, for example, increased rates of mental disorder in adult life when subjected to stress. Interestingly, similar brain imaging changes were found in studies of a variety of adult mental disorders but only in those cases where there was a history of childhood maltreatment. It is also well known that childhood maltreatment exposes survivors to the risk of a variety of other medical disorders.[17,32]

It is not suggested that ACEs and mental disorder are relevant to all cases of CRPS. Survivors of ACEs/childhood maltreatment, people with mental ill health and patients with CRPS are viewed here as overlapping but not identical populations.

In clinical practice, patients with CRPS are encountered where no history of mental disorder or childhood maltreatment is established after careful inquiry. However, given current research evidence and clinical observations, an area meriting further careful investigation may be whether some patients who develop chronic CRPS in the absence of psychiatric disorder at the time of the injury may be survivors of childhood maltreatment. Thus, having survived adaptively for years, but remained vulnerable, they may have decompensated when they suffered serious physical injury or trauma because of long-term underlying vulnerability. In the cases where no physical or surgical injury precedes the onset of CRPS symptoms, the precipitant may be life events and/or emotional distress.[21–24] Comparative functional imaging of (1) adult survivors of childhood maltreatment, (2) adults with common mental disorders (anxiety and depression) and/or somatoform or personality disorders, (3) adults with CRPS and (4) adults with combinations of 1/2/3 may help address relevant questions. It is hypothesised that such research may identify different CNS changes in different CRPS subpopulations.

CRPS and behaviour

Discussion of epidemiological, psychiatric and biological factors should not obscure the importance of behavioural factors in CRPS. Indeed, behaviour may be the way through which these factors exercise their influence.

Patient behaviour

The guidelines recognise that simple immobilisation of the extremity even in the absence of any formal psychiatric disorder may of itself produce some symptoms and signs of CRPS, though some authorities doubt whether it is enough to produce the full syndrome as defined above. Be that as it may, fear of movement of the affected limb (referred to as kinesiophobia or fear of movement) is a common feature of the full syndrome. We recognise that there are well-established significant psychological and psychiatric predictors of kinesiophobia following injury and development of pain syndromes generally. These observations also suggest it is valuable to consider such pre-existing psychological factors as risk for developing CRPS. *In any case, the guidelines* sensibly suggest, an essential part of treatment is to encourage the patient to move and use the affected limb.

It has also been suggested that patients with CRPS may have made up, induced or exaggerated their symptoms. It is crucial to distinguish between psychological and behavioural factors as aetiological factors or reactive factors, on one hand, and deception and deliberate exaggeration, on the other hand. Although there have been well-documented cases of factitious disorder reported in the literature,[33–37] it is the present authors' assessment that the majority of CRPS patients in the clinical setting are genuine, though potentially less so in the medicolegal context where covert surveillance sometimes uncovers malingering. Mailis-Gagnon et al.[37] concluded that 5 out of 15 women referred for neuropathic pain over 2 years, and fulfilling 'modified IASP CRPS criteria', demonstrated convincing 'active self-induced signs and symptoms'. As to exaggeration, it is our experience that most patients' reports are generally not disproportionate when both the extent of objective physical symptomatology and comorbid psychiatric disorder are considered,[38] at least not obviously more than in other medical or surgical conditions. Once more, experience varies between the clinical and medicolegal contexts.

Clinician behaviour

What may not have attracted enough attention in the literature on CRPS is clinicians' behaviour and its importance in the

understanding and prevention of CRPS. Two aspects of clinician behaviour will be discussed here:

1. Enquiry about psychological factors during assessment, including ACEs and past or present adversity or life events where this may be relevant;
2. Clinical care and associated pathways.

With respect to eliciting a detailed psychosocial history, the authors' observation is that there is divergence in practice between different clinicians specialising in the assessment and treatment of patients with CRPS. This is not unusual in medicine.[39] Reluctance by some to enquire in detail likely reflects in part the clinical reality of stigma against mental disorder and patients affected by it. This arises out of misunderstanding, leading some patients and others to think that survivors of childhood maltreatment are to blame for or exaggerate their experiences; indeed, that mental disorder is deliberate or under voluntary control or a sign of moral inferiority.[40] In the circumstances, clinicians may avoid 'going there' for fear of 'upsetting the patient' and being perceived as suggesting it is 'all in the mind'. There may also be fear of 'opening a can of worms' and thus 'making matters worse' for the patient.

The above considerations may loom large, especially in a busy outpatient clinic. However, Felitti[41] reports that in a general medical population (i.e. not specifically CRPS)

> *Analysis of a 125,000-patient cohort where comprehensive biopsychosocial screening was routinely used showed a 35% reduction in doctor office visits (DOVs) during the following year. By contrast the previous purely biomedical screening approach in the same Department of Preventive Medicine produced an 11% net reduction in DOVs*.

There is preliminary evidence therefore that asking about ACEs may increase, rather than decrease the efficiency of clinics. Perhaps stigma-related issues, difficulties in establishing relations with survivors who are continuing to grapple with the legacy of ACEs or current stress and lack of training and support of clinicians may be more important than efficiency in practice.

Some may argue that communication about issues relating to mental illness and history of maltreatment in CRPS is best left to mental health professionals, particularly psychologists. The employment of psychologists in chronic pain services is increasingly the case. Indeed, one of the surprising features of *the guidelines* is that there is no chapter on psychology, though there are distinct chapters for primary care and a variety of medical specialties and therapies. This may reflect lack of evidence for efficacy of psychological treatments, though there is hardly more evidence for some other therapeutic modalities commonly used and endorsed in *the guidelines* either. However, it should not deter the employment of psychologists. They may help explore the sense of identity and relationship with the limb, that is, the issue of alienation and disengaging from the limb; also, employ effective interventions for the comorbid mental health problems frequently found in this chronic pain population.

The role of psychologists is complementary and not exclusive to that of other clinicians in formulating fully the aetiology and treatment of patients with CRPS. We should not allow the perpetuation of the now discredited mind-body dualism in clinical practice. A specific risk, that might arise if psychological enquiry is left entirely to psychologists, is that their assessment may misconceive of psychological morbidity found in such patients as simply a reaction to CRPS, rather than something that may be of greater aetiological significance. Such misconception is not necessarily the rule, but the risk increases unless all members of the clinical team have some appropriate skills to enquire about psychological morbidity and history of childhood maltreatment.

There is another more straightforward way in which clinician behaviour may be important in the understanding and treatment of CRPS. The reason for stating this is that the 'Liverpool pathway' devised by physiotherapists has led to a dramatic fall in incidence of CRPS following radial/ulnar injury from 25% to less than 1%.[42] The pathway requires alertness to the symptoms and chronicity, consistently respectful support to the patient, practical advice about movement and posture and quick review and appropriate adjustment of clinical management when relevant problems emerge or persist. Interestingly, having achieved a dramatic reduction, they observed resurgence of case incidence at some point after they first introduced their pathway. On investigating this further, they found that this resurgence coincided with a change in junior doctors in the service. Consistency therefore is essential for prevention. With attention to this, further reduction in new incident cases has been achieved.

It is noteworthy that the authors of this report on the Liverpool pathway agree that, as well as aiming at change in the behaviour of clinicians, their intervention may be conceptualised as a form of behaviour therapy of the patient, with elements of emotional support, education about their condition, challenging negative assumptions and changing behaviour through facing down unfounded negative predictions (Cowell F and Gillespie S, personal communication).

Conclusion

CRPS is certainly not 'all in the mind'. For those patients who find themselves chronically affected, it is a true nightmare. In

that sense, although it may not be all in the mind, it is very much in the mind as well as the body.

CRPS is a real medical condition where behaviours play a significant role in its aetiology and persistence. The relevant behaviour in CRPS includes, but may not be limited to, immobilisation of the affected extremity. However, the 'Liverpool Pathway' evidence suggests that clinician and therapist behaviour may also be crucial.

Clinicians may contribute to better understanding and reduce chronicity with two distinct modes of behaviour. First, they may be able to elicit full appropriate psychosocial history, including but not limited to how this may predispose to kinesiophobia. Second, they may respond in a timely and sensitive way when the patient presents with complications. Clinical experience and common sense suggest that this requires good communication skills by clinicians, which engender trust and appropriate disclosure by patients. The development of such skills should be part of core training. The remarkable success of the Liverpool pathway suggests that effective communication and responsive practice effect demonstrably good clinical outcomes.

We will conclude by turning our attention briefly to psychiatry. As with psychology, there is no section dedicated to the role of psychiatry in the current guidelines.[43] G.I. was invited to represent the Royal College of Psychiatrists Liaison Psychiatry Faculty on *the guidelines* working group. Regrettably, he was not able to recommend endorsement of the proposed psychiatry and CRPS section to the Faculty.[44,45] Nor were the leaders of the working group able to accept *the guidelines* currently in use at the Royal National Orthopaedic Hospital. These latter guidelines, in our view, offer a balanced view of what psychiatrists have to offer to CRPS patients and the clinicians looking after them.[46] It is to be hoped that future revisions of guidelines may evolve to include mutually acceptable dedicated psychology and psychiatry sections, especially as evidence suggests that the integration of both psychology and psychiatry can facilitate both research and practice in the clinical care of patients with long-term disabilities.[47]

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SAGE

Analgesic effect of 2-Hz electric percutaneous neuromodulation in participants suffering from neuropathic pain

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Introduction

This is a short-term observational study in participants suffering from neuropathic pain and treated regularly with percutaneous electric neuromodulation. Percutaneous electric neuromodulation is a non-pharmacological, minimally invasive method of treatment for neuropathic pain.

The first modern recorded use of electric neuromodulation was described by Wall and Sweet[1] following the introduction of the portable transistorised nerve locator by Greenblatt and Denson.[2] The Gate Control Theory by Melzack and Wall[3] provided a rationale for direct stimulation of peripheral nerves as a means to provide pain relief. Other mechanisms may also be involved, including long-term depression (LTD) which is observed at the synapses between nociceptive afferents and superficial dorsal horn neurons, affecting the conditioned pathway which was produced by 1-Hz electric stimulation in a rat model.[4] LTD-like reduction of human pain perception has also been observed by many authors.[5–12] Some of these LTD-like experimental studies use point electrodes in a circle.[5] This is perhaps reminiscent of a technique known as 'circling the Dragon' (known also as fencing in the dragon or surrounding the dragon) which is widely used in Chinese acupuncture. In these techniques, the affected area is surrounded with a ring of needles about 2.5 cm apart, as the axon reflex covers a 25-mm radius.[13–17] There is now increasing evidence that different modalities of electric neuromodulation can improve pain management in a variety of chronic pain conditions.[18–30]

The purpose of this observational study was to perform a short-term evaluation of percutaneous electric neuromodulation in participants with chronic neuropathic pain, studying both its analgesic effect, as well as the participants' perceived effects on quality of life and any adverse effects.

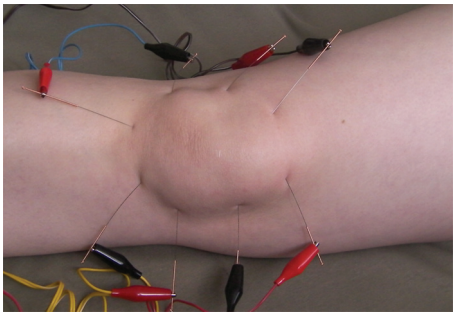
Material and methods

The proposal for this observational study and data collection tool was reviewed and approved by the Clinical Audit Review Panel – Lewisham and Greenwich National Health Service (NHS) Trust (audit no. 4045). Patients who were suffering from neuropathic pain and booked for routine electric neuromodulation treatment were included in this study. Patients who were unable to read, write, communicate any form of English, use e-mail or use a mobile phone were excluded from this study. Patients who were unable to take part in this study for any other reason were also excluded.

Before treatment, participants were asked to rate their pain level (with a numeric pain rating scale going from 0 to 10, with 0 being no pain and 10 being their worst pain imaginable) and how much their pain had affected their daily activities, for example, walking, sleeping and well-being, over the week before treatment (with a numeric rating scale for the pain affection, going from 0 to 10, with 0 being no effect and 10 being a complete impairment).

After receiving written informed consent, percutaneous electric neuromodulation was applied aseptically for 10 minutes

Figure 1. The frontal aspect of the knee joint surrounded with a ring of needles (needle diameter 0.25 mm, length 45 mm) and connected to 2-Hz electric stimulator



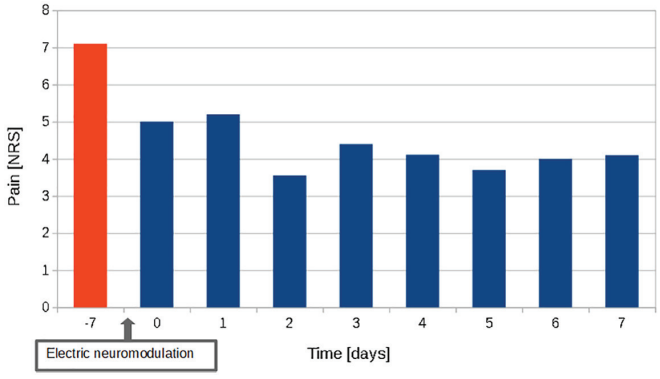
using a ring of acupuncture needles (2–8 needles/patient) surrounding the affected area (Figure 1) and a Pointer F-3 electroacupuncture stimulator (with a frequency of 2 Hz, pulse width of 350 µs, a biphasic rectangular pulse shape, an intensity of 0–18 mA and a duration of 10 minutes). The intensity of the stimulation was titrated up to the maximum intensity which did not cause any pain.

Immediately after stimulation, participants were asked to complete a short questionnaire to assess any adverse effects of treatment. Every day, for 7 days following treatment, participants were asked to report pain intensity and how much the pain affected their daily activities, walking, sleeping and well-being once per day using e-mail or SMS. If the patient failed to report their pain or level of activity for a day, they were contacted by a member of the audit team as a reminder, since it is very important to examine the trends for the entire period. The primary endpoint was a reduction of pain intensity, measured by the Numeric Rate Scale. Secondary endpoints included improvement in daily activities, walking, sleeping and well-being.

Results

A total of 14 participants were screened, and 11 participants were recruited. The average age range was 44–70 years (median 58); eight were female and three were male. The neuropathic character of pain in the recruited participants was confirmed using the DN4 questionnaire (range DN4 = 4–10, except for one patient who scored DN4 = 1 but, in the investigator's opinion, suffered from neuropathic pain). Five participants were retired and five were still working. The neuropathic pain was localised at a knee for five participants, at the hands for two participants, at the face for one participant, at an upper limb for one participant, in the subcostal area for one participant and at multiple locations for one participant.

Figure 2. Primary endpoint: combined data for pain intensity for all participants (average)



–7 denotes the pain intensity in the week prior to treatment (in red); 0 denotes pain intensity at 7PM on the treatment day, after treatment (in blue); 1 to 7 denotes pain intensity at 7PM on days 1–7 after treatment (in blue).

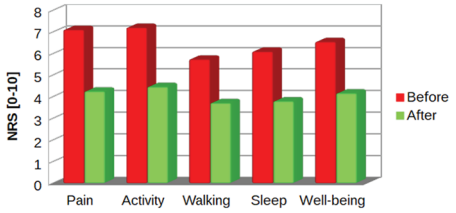
Prior to this observational study, these participants had received between 1 and 19 (median 7) percutaneous electric neuromodulation treatment sessions identical to that described in this article; previous sessions were usually 1–3 months apart. Combined data for pain intensity for all participants are presented in Figure 2. Combined data for all endpoints (pain, activity, walking, sleeping and well-being) are presented in Figure 3. On average, pain intensity was reduced by 40.1%, and the effect of pain on activity was reduced by 37.9%, on walking by 35.2%, on sleeping by 37.7% and on well-being by 36.5%. Participants did not report any adverse reactions to 2-Hz electric neuromodulation treatment except for some pain due to needle insertion.

Discussion

This study showed the efficacy of 2-Hz percutaneous electric neuromodulation in a small case series of participants suffering from chronic neuropathic pain. The intensity of pain (primary endpoint) as well as daily activities, walking, sleeping and well-being (secondary endpoints) was improved by at least 35%. Treatment was well tolerated by all participants. The study population was not naive to the percutaneous electric neuromodulation treatment, which may suggest that the placebo component of pain relief was at least partially suppressed. The use of e-mail or SMS was a simple and effective method for robust data collection in the patient population.

The technique of percutaneous electric neuromodulation presented in this article may look similar to transcutaneous electric nerve stimulation (TENS). Both percutaneous electric

Figure 3. Primary and secondary endpoints: combined data for pain, activity, walking, sleeping and well-being



'Before' denotes value in Numeric Rate Scale (0–10) for week before treatment; 'After' denotes average value for all participants for days 0–7; 'Pain' denotes pain intensity; 'Activity, walking, sleeping, well-being' denotes how much pain affected them.

neuromodulation and TENS use similar parameters of stimulation (voltage range, current range) but the size of electrodes and their geometry are significantly different, resulting in the following:

1. The estimated vector of the electrical field intensity is perpendicular to the skin surface for TENS and parallel to the skin surface, with much more complicated geometry, in percutaneous electric neuromodulation.
2. In both techniques, the intensity of the electrical field is the highest near to the electrode surface. In TENS, the gradient of electric potential is created due to a difference between the high resistance of the corneal layer and the low resistance of deeper layers of the skin. In percutaneous electric neuromodulation, the high gradient of the electric field, near the electrode surface, is a result of the small diameter of the electrode.
3. In TENS, due to the high resistance of the corneal layer, stimulation is most likely delivered only to the most superficial nerve endings in the skin. In contrast, during electric neuromodulation, the needle is inserted into tissue and electric stimulation is directly delivered to the full thickness of skin, subcutaneous tissue, muscles, nerves, blood vessels, and so on, without any high-resistance layer.
4. There is a significant difference in the current density at the electrode surface. Electric current with an intensity of 20 mA delivered to TENS electrode 5 cm × 5 cm (area 25 cm^2) creates a current density at the TENS electrode surface of 8 A/m^2. Surface current density of the acupuncture needle (length 2 cm, diameter 0.25 mm) connected to 20-mA stimulator exceeds 6,000 A/m^2.

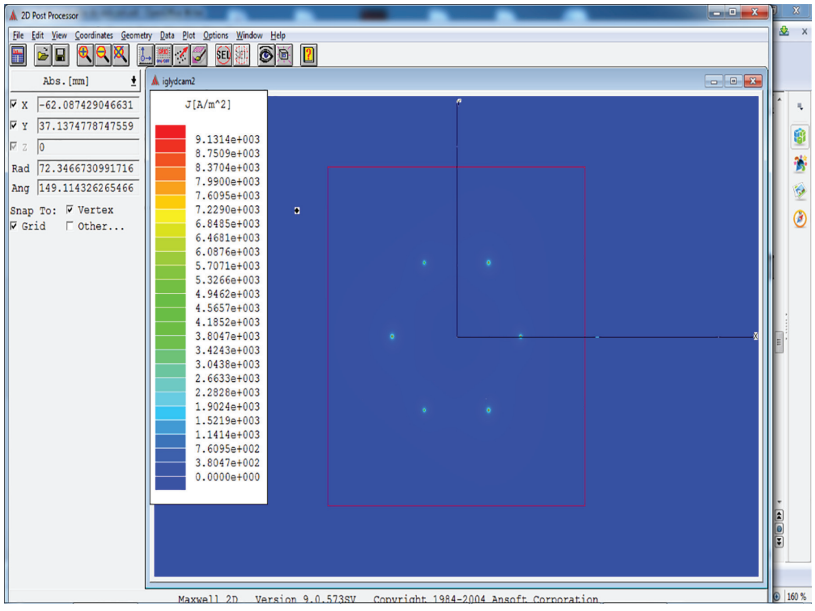
The geometry and magnitude of the electric field, lack of isolation layer, the direct transfer of electric current to deep layers of tissue and very high electric current density at electrode surface may be responsible for the analgesic effect of percutaneous electric neuromodulation. In addition, flare (redness) often present during percutaneous electric neuromodulation may suggest activation of high-threshold C fibres in a mechanism known as the Lewis reflex.[31] Similar geometry, a grid with lines 1.5 cm apart, was used in studies of direct analgesic effect of botulinum toxin type A in localised chronic neuropathic pain.[32]

A finite element model simulator (Maxwell 2D version 9.0.573SV by the Ansoft Corporation, Pittsburgh, PA) was used to better understand the mechanism of percutaneous stimulation. The geometry of the simulation was defined as six needles with diameter 0.2 mm in a regular hexagonal pattern with the distance between needles equal to 2.5 cm. To mimic biological conditions, the medium was defined as 0.9% NaCl solution (permittivity = 80 and bulk conductivity = 1 S/m). The needles were alternately connected to positive and negative potential. The potential difference was equal to 5 V. The simulated area was divided into 40,000 triangles creating a set of 40,000 equations. To visualise current density, this set of equations was solved for the minimum energy of electric field. The results of the simulation are presented in Figure 4. Before the simulation, we expected that when the affected area is surrounded by a ring of the needles (Figure 1), the maximum density of the electric field will be present in the middle of the affected area. This scenario will strongly support the peripheral mechanism of percutaneous stimulation. The results of the finite element model produce the opposite effect. An electric field density strong enough to stimulate nerve fibres is present only in the immediate proximity of the needles (which is in agreement with a subjective sensation during simulation). Even more surprisingly, in the middle of the stimulated area, the electric field density is equal to exactly zero. These results suggest that during a percutaneous electric stimulation with needles inserted around the affected area (Figure 4), there is no direct stimulation of the centre affected area, and the therapeutic effect originates from the stimulation of the surrounding healthy (non-painful) tissue.

The multifaceted response to the electric stimulation of the nociceptive peripheral nerve also includes the combination of axon reflex, also called the flare response, and dorsal root reflexes (DRR) together with the complex interaction of the locally released substances such as substance P and calcitonin gene-related peptide (CGRP)[33–40] which may mediate part of the analgesic response.

It has been suggested that the differential contribution of low-frequency (20 V, 5 Hz, 0.5 ms) electrical stimulation of the central stump of the cut dorsal root, which produces bilateral vasodilatation of the innervated area, but not plasma extravasation, was observed in an experimental animal study.[41] We postulate that low-frequency peripheral electric stimulation

Figure 4. 2D results of finite element simulation of current density (A/m^2)



can produce a selective neuro-inflammatory response: vasodilatation without plasma extravasation.

In our observational study, we did not directly measure vasodilation or plasma extravasation. However, we observed significant redness of the affected area without any noticeable swelling, which supports the possibility of vasodilatation without plasma extravasation.

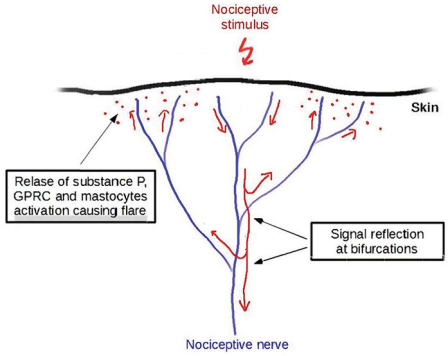
The vasodilatation without plasma extravasation does result in increased blood flow to the area affected by neuropathic pain and, subsequently, increased oxygenation of the affected tissues, which may have a direct effect on tissue regeneration and wound healing. Increased blood flow has a possible beneficial effect on the reduction of locally secreted chemical mediators of inflammatory process. The interaction between the endogenous electrical field and the electrical stimulation also plays an important part in the wound-healing process.[42]

In the light of current knowledge, which is unfortunately incomplete, based on basic science investigation and clinical observation studies on the effects of the various frequencies applied in peripheral neurostimulation in neuropathic and nociceptive pain syndrome, the optimal frequency has not been established. However, multiple animal studies and clinical reports suggest that low-frequency stimulation (LFS) offers significant advantages when compared to high-frequency stimulation (HFS).[18,19,21,23,27,29,30,5,41–56]

The axon reflex, also called the flare response, is the response to stimulation of the nociceptive peripheral nerve. When one of the branches of the nociceptive nerve is stimulated, the signal thus produced travels orthodromically towards the neuron cell (dorsal root ganglion). However, at the bifurcation, the signal reflects and travels antidromically (backward) towards the skin where substance P is produced causing vasodilatation (Figure 5). The axon reflex is limited to the area enervated by a single multi-branched axon, without any integration centre or chemical synapses. Flare response can precisely be measured by laser Doppler flowmetry.[57] It has been suggested that DRRs are involved in vasodilatation, but not in plasma extravasation in neurogenic inflammation.[41]

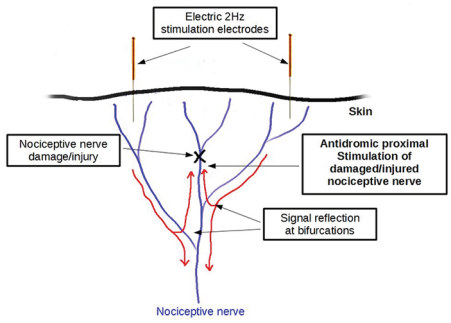
When neuropathic pain is present at a limited skin area (e.g. around a scar), we may presume that the damaged/injured branches of nociceptive nerves terminate in the centre of the affected area. When needles for electrostimulation are inserted around the affected area, they will stimulate nociceptive nerve endings, of which some are healthy branches of affected nerves. The produced signal will travel orthodromically towards the neuron body. However, due to the axonal reflex, it will reflect at bifurcation, travelling antidromically (backward) towards the skin. At damaged/injured nerve branches, this will produce antidromic proximal stimulation (Figure 6). Regeneration of peripheral nerves

Figure 5. Axonal reflex



Nociceptive stimulation of one of the branches of the nociceptive nerve produces a signal which travels orthodromically towards the neuron cell; however, at the bifurcation, the signal reflects and travels antidromically (backward) towards the skin where substance P is excreted causing vasodilatation.

Figure 6. Antidromic proximal stimulation of damaged/injured nociceptive nerve



Low-frequency electric stimulation round the affected area causes activation of nociceptive nerve endings, of which some are healthy branches of affected nerves; the produced signal will travel orthodromically towards the neuron body; however, due to axonal reflex, it will reflect at bifurcation, travelling antidromically (backward) towards the skin; at the damage/injured nerve branches, this will produce antidromic proximal stimulation.

due to electric stimulation proximal to injury was previously reported.[58–61]

Based on the above results, we would like to postulate an axon reflex mechanism of percutaneous electric stimulation. Stimulation of healthy branches of affected nerves produces an antidromic proximal stimulation of a damaged/injured branch which may promote its regeneration in addition to selective vasodilatation without plasma extravasation.

This was a pilot study to evaluate participants' benefit when receiving electrical neuromodulation for their chronic neuropathic pain. A period of 7 days was selected because it allowed for the evaluating team to have continuous contact with, and feedback from, the participants on a daily basis. Participants were also more clearly able to recall the changes in their pain and activity levels within the last 24 hours. Prior to this study, patients had reported a decrease in pain levels and an increase in activity levels, but were unable to provide any descriptive details of their progress by the next treatment session, which often occurred 1–3 months later. Moreover, it can be seen that participants benefitted in the short term from a decrease in their chronic neuropathic pain during the course of the 7-day observation.

An observational study is easy to complete and less expensive than clinical trials. The limitations of the study include a lack of randomisation and placebo control, which may lead to an overestimation of the results. However, comparison of the results from well-designed observational studies and clinical trials of the same topic found that the average results of observational studies were similar to the results of clinical trials.[62] For a future study on the long-term benefits of electric neuromodulation, a larger population may be recruited to see if the same analgesic effect and reduced impact of pain on daily activities, walking, sleeping and well-being occurs using similar inclusion and exclusion criteria.

The results of this study, together with previously published data on 2-Hz percutaneous electric stimulation, may suggest that 2-Hz percutaneous electric neuromodulation is a simple, effective and well-tolerated treatment option for selected participants suffering from neuropathic pain. Percutaneous electric neuromodulation may be a valuable treatment option for those participants who may not respond well to pharmacological therapy, or for those who wish to avoid it.

Acknowledgements

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Treating pain and drug dependence: getting it 'least wrong'

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SAGE

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Introduction

This is the second of two articles outlining one psychiatrist's pragmatic views on the management of pain in patients with co-morbid opioid dependence. The first article outlined the drugs used to treat dependence, elements in the assessment of dependent patients and special considerations in this group. This article looks at difficulties in managing patients with acute and chronic pain. For brevity, I have assumed that acute pain is managed in hospital, chronic pain in outpatients.

Patients who are dependent on opiates often present to services, due to consequences of drug use (accidents, complications of injecting, blood-borne viruses). The ageing cohort of addicts in opiate substitution treatment have poor physical and mental health as well as the poverty and psychosocial problems associated with drug misuse. As in every patient, the intensity of the pain is determined by the degree of tissue injury alongside cognitive and affective influences, including current mood, past experience of pain and concerns about the cause. The backgrounds, habits and coping styles of opioid users will have militating effects in all these spheres.

There is no high-grade evidence from randomised controlled, nor good observational, studies, on which to recommend pain management in recovering or current opioid addicts.

Acute pain

Setting goals when managing acute pain is usually straightforward: make the pain go away or at least keep the patient as pain-free and free from distress as possible until an intervention resolves the cause of the pain. The additional goal for opioid-dependent patients is to stabilise their opioid dose as quickly and safely as possible so that they can be treated for their other medical conditions.

Credit: Wildpixel.

Assess as described in the first article: establish the dependence, establish whether the patient is receiving opioid substitution treatment, confirm the prescribed and dispensed doses and whether the consumption is supervised, and obtain corroborative information including urine drug screens.

Management of patients who are dependent on opioids varies according to where in the addiction journey they find themselves.

Abstinent addicts

Opioids are not contraindicated in abstinent, former addicts with acute pain. There is a poorly defined, probably small risk that exposure to opioids will trigger relapse to addiction, but it is a great concern to recovered opioid users, especially those who maintain their abstinence with the support of Narcotics Anonymous or similar organisations. You will need to discuss the pros and cons of analgesia to facilitate an informed choice regarding whether opioids should be prescribed. If they have already been administered as an emergency, discussion about

the best way of ceasing opioid use will need to be undertaken. Usually a short reducing regime is enough, but some may need referral into addiction support services to manage their concerns and fears regarding relapse.

Stable in treatment

Those who are stable on opioid substitution treatment are probably the easiest group to manage. Verify the timing and dose of the most recent opioid substitution and check for other medications, including alcohol and benzodiazepines. Ensure the usual opioid substitution is continued and review if they run into difficulties when additional opioids are used for pain relief. Be alert for drug interactions which may reduce opioid levels and precipitate withdrawal.

Unstable in treatment

Those who are receiving opioid substitution treatment but have not successfully managed to refrain from illicit drug use are the most time-consuming. Verifying doses and dispensing details are essential in this group. If methadone or buprenorphine is not taken under supervision, there is no guarantee that the dose prescribed is actually being consumed and inadvertent overdosing can ensue. The community pharmacist who dispenses the medication is an invaluable source of help. They will know when the last dose was dispensed and how many days' supply were issued, thus whether the patient may have additional medication in their possession. They can indicate whether the patient is someone who regularly misses out on the collection of their medication, implying that they will have been taking doses erratically, may have lost tolerance to medication or may be diverting their supply.

Opiate substitution should be undertaken using small frequent doses of medication. Cautious administration of lower than prescribed doses of substitute medication should be used, trying to find the right balance between withdrawal symptoms and oversedation while maintaining adequate analgesia. Methadone may be preferable given the potential of buprenorphine to interfere with pain management, but if a patient is already prescribed, or if respiratory depression is a concern, buprenorphine may be preferred. For both opioid substitutes, the principle is frequent small doses, with extra doses if withdrawal is a problem and omission if oversedation prevails. I suggest 10 mg methadone as 1 mg/mL mixture or 2 mg buprenorphine every 6 hours, with increases titrated against withdrawal symptoms; you may elect to use a validated assessment scale for opioid withdrawal, for example, the Short Opiate Withdrawal Scale.[1] Methadone has a long and variable half-life (13–47 hours): if the same dose is given repeatedly, the drug can accumulate and doses which might have been safe on day 1 can be excessive by day 3. Close monitoring for signs of toxicity is important.

Newly diagnosed, not in treatment

The patient may be someone who has not acknowledged their addiction or who has not engaged with opioid substitution treatment for a variety of reasons. Inpatient management depends on whether to utilise the admission to detoxify the individual or to initiate opioid substitution to manage withdrawal. While taking the opportunity of a hospital admission to detoxify may occasionally be useful, if unplanned and solely in response to the admission, the patient is very likely to relapse on leaving hospital, exposing them to a substantially increased risk of overdose. This must be explained if they are able to give informed consent to detoxify. It may be more useful to initiate opioid substitution, ideally in conjunction with the local drug treatment services. Methadone is preferable given the potential of buprenorphine to complicate analgesia. Start with low and frequent doses as described above. If opioid substitution is to be continued following discharge from hospital, this must be in liaison with community drug treatment services.

Dependence on prescription drugs

The overuse of prescribed painkillers and over-the-counter analgesics containing opiates is increasingly recognised, but in practice, problems may only become apparent when withdrawal symptoms develop. Individuals often do not recognise their dependence, possibly because they have never been in a situation where their prescription medication is not available (see vignette). Indicators include co-morbid psychiatric illness, use of painkillers to attenuate unpleasant thoughts and experiences, high doses and the prescription of other psychotropic medication such as benzodiazepines. Patients can react with horror if it is implied that they are drug-dependent and the mention of methadone prescribing causes dismay.

The dilemma in such cases is whether to initiate opioid substitution treatment as an inpatient. Given the resistance to the idea of addiction, the high levels of emotion encountered and the likely brief duration of admission, my pragmatic approach is to continue the medication prescribed in primary care with analgesia in addition (as I would for someone on methadone) and to discuss referral to drug treatment services with the patient. The GP must be alerted to the recognition of dependence on prescribed medication so that it can be addressed at a more opportune moment.

Split dose of substitution

Splitting the dose of opioid substitution treatment into two or three daily doses has advantages. There may be some analgesic effect if the dose is divided and given at intervals. The patient may be less anxious as they know that they will be

Case vignette: unrecognised prescription drug dependence in an emergency admission

A 40-year-old woman was admitted with acute on chronic abdominal pain and underwent a cholecystectomy. Her postoperative pain was satisfactorily managed with patient-controlled parenteral anaesthesia, but when she was transferred to oral medication, her complaints of severe pain led to conflict with nursing staff.

Review of her previous medication revealed that she had been taking eight tablets a day of prescribed co-codamol 50/300 for the past 3 years. The admitting doctor had noted the co-codamol prescription but had not specified formulation or amounts.

Her pain was a combination of anxiety and drug withdrawal as the conversion from parenteral to oral analgesia had not accounted for her chronic opiate use.

receiving regular doses of their medication. If there is any doubt about the dose of substitute medication usually consumed, splitting the dose reduces the risk of overdosing and allows detection of any toxicity before large doses have been administered.

Use short-acting opiates

Analgesia in opioid-dependent patients should be the same as in any other patient in acute pain. Frequent doses of short-acting opiates would usually be prescribed: it should be no different in this group. Patient-controlled analgesia is effective and appropriate. Pain perception may be greater, tolerance to opiates may have developed and there may be hyperalgesia exacerbated by anxiety: higher than usual doses may be required in this population.

Patients on buprenorphine undergoing elective surgery should continue their usual dose and have additional opioid analgesia titrated against response.[2] Occasionally, patients on buprenorphine may experience little relief from opioids, and alternate approaches such as regional anaesthesia and non-opioid analgesics may be required. Increasing buprenorphine doses is unlikely to work due to the 'ceiling effect'. As μ-receptor occupancy can persist for 72 hours, discontinuing buprenorphine is rarely a sufficiently timely option: alternative approaches will be needed. This also applies to patients who have been taking naltrexone.

Reduce dose as pain resolves

As acute pain is likely to be short-lived, there should be a clear plan for reducing the added medications as the acute pain subsides. This plan should be clearly communicated to the patient and any carers (confidentiality permitting). If reduction is to be undertaken outside hospital, the GP and drug treatment service must be informed of the plan and it should be clear who is taking responsibility for issuing and monitoring prescriptions.

Discharge

Ideally, discharge should be planned with at least 24 hours' notice to the GP and drug services. Take-home medication should be of short duration, with a plan for review and clear communication as to who is prescribing subsequently. Prescriptions of short-acting opiates for more than a week's supply should be avoided. The GP or drug service should be contacted on the day of discharge and informed of what medication has been administered at the hospital and the dose, the number of days' supply the patient is taking home and any other medication prescribed. The patient should have an appointment for review by their usual prescriber made and given to them before they leave hospital.

Patients who have been newly initiated on opiate substitution should be discussed with the drug services: this is an indication for rapid access to prescribing. Regardless of whether the patient wishes to engage with treatment services, long-duration prescriptions of opioids should not be given. For patients who do not wish to address their drug use, the focus should be on risk reduction. A short admission (1 or 2 days) is unlikely to have affected tolerance to their opiate of choice, but patients who have been admitted for longer need to be aware that the effects of street drugs or non-prescribed medication may be different following discharge, with the risk of inadvertent overdose if old levels of use are resumed.

Formerly abstinent addicts who have been treated with opiates for acute pain should be withdrawn from medication while in hospital. They will still need to be able to access support on discharge. Refer to the drug treatment services (for counselling, rather than prescribing) or encourage to attend their usual support services (such as Narcotics Anonymous).

Chronic pain

Patients who have a history of heroin dependence have many reasons for the development of chronic pain. Physical injuries due to trauma or drug-related illness are common. Blood-borne infections and addicted lifestyle can contribute to chronic, painful conditions, such as infected leg ulcers secondary to injecting. Drug dependence sits alongside poverty and social deprivation with a high prevalence of chronic physical illnesses, and the lifestyle of many addicts contributes to demoralisation, a sense of distress and hopelessness with loss of motivation. As the population of patients on opioid substitution ages, the

usual diseases of ageing develop. Patients may develop emergent pain as opioid substitution treatment is reduced. In any patient, when pain becomes chronic, emotional factors predominate in determining intensity of pain with distress and anxiety often the biggest contributors. Addiction is additionally associated with emotional dysregulation, with low mood and focus on physical symptoms. All these factors contribute to experience of bodily pain.

Consider alternatives

In recent years, considerable doubt has been cast on the effectiveness of opioids in the relief of persistent non-cancer pain.[3] Chronic pain is difficult to treat, with both pharmacological and non-pharmacological interventions helping no more than 20% to 30% of patients. For those patients who do respond, reductions in pain intensity are modest and complete pain relief is not a realistic goal. These limitations need to be made explicit to patients. The opioid-dependent population may be resistant to this: they are used to perceiving a refusal to prescribe as synonymous with not being believed and may interpret the absence of a prescription due to stigma. They may also be less accepting of non-pharmacological treatments than their non-dependent counterparts as the experience of years of addiction provides a powerful memory that drugs will relieve the pain, at least in the short term. The clinical challenge is to engage the patient, as any attempt to change management without the patient's willingness to do so is likely to result in the new treatment 'not working'. The aspiration is to retain a stance of empathy and positive regard for patients while not accepting the patient's attribution of their problem to a simple physical cause, and challenging their belief that medication is the solution.

Beware gabapentinoids

Pregabalin and gabapentin are licensed for the treatment of neuropathic pain and in the United States for fibromyalgia. Evidence for misuse has been accumulating, particularly in people who misuse other drugs and in specific settings such as prisons. The doses misused are often many multiples of therapeutic ranges. The drugs are used to produce euphoria and a sense of calm. Some users have reported a stimulant effect. They are purported to enhance psychoactive effects of other drugs. Pregabalin appears to be more sought after for misuse than gabapentin, which may relate to differing pharmacokinetics, with pregabalin able to achieve higher doses in the body. Dependence on both has been reported although the mechanism is not well understood. Reported symptoms and signs of withdrawal from gabapentinoids include insomnia, headache, nausea, anxiety, diarrhoea, flu-like symptoms, nervousness, depression, pain, fits, hyperhidrosis and dizziness.

Set goals and review progress

National US Guidelines published in 2009 stated that the factor that appears to be most strongly predictive of drug abuse, misuse or other aberrant drug-related behaviours after initiation of chronic opioid therapy is a current or past history of addiction or a family history of alcohol dependence.[4] This is unhelpful to clinicians or addict patients confronted with the problem of managing pain. Such caution can increase the stigma associated with addiction and becomes a further barrier to management of pain in this population.

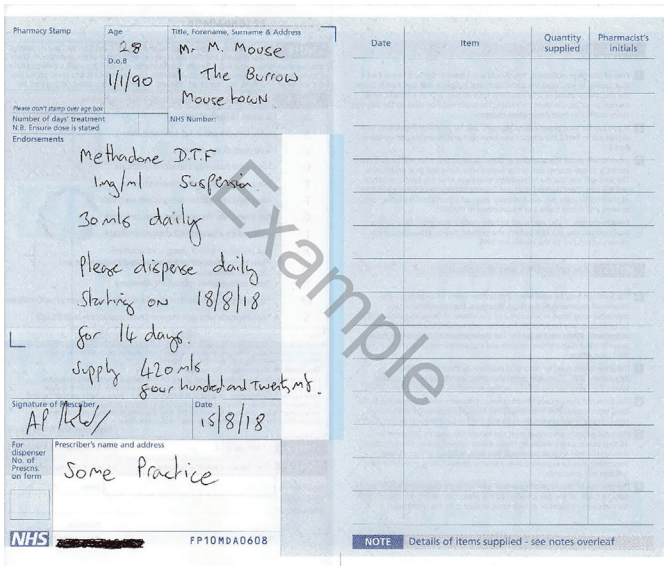
If alternatives to opioid prescribing have been exhausted and comorbidities have been identified and treated, it may be appropriate to consider a trial of opioids, but there are caveats. Decide whether the benefits for pain and function outweigh the harms. Have clear expectations that short-term improvement in pain should be exploited to maximise the effects of non-pharmacological measures. Assess where the patient is on their addiction journey: adequate pain control is unlikely in an unstable drug user or one who is using illicit drugs in addition to opiate substitution treatment. Such individuals will need careful liaison with drug treatment services and a clear plan of who is prescribing, the frequency of dispensing and whether any consumption is supervised. If the patient is stable on opioid substitution therapy, continue that therapy: any prescribed opioid should be in addition to the background opioid treatment.

You need to agree clear goals with the patient outlining the objectives of treatment: what would success look like? Without mutually acceptable objectives, there is a risk of unrealistic expectations. The goals should be SMART (specific, measurable, achievable, relevant and timely) and should include meaningful functional outcomes in addition to subjective measures like improved sleep or reduced pain. Specify the duration of the treatment trial: the Royal College of Anaesthetists' 'Opioids Aware' website[5] recommends up to 2 weeks if pain is constant, or sufficiently long to have experienced up to three cycles of pain if the pain is intermittent. Explicitly discuss how therapy will be discontinued if goals are not achieved and repeatedly reassess harms and benefits. Remember that short-term success is not the same as long-term efficacy; be clear that opioid use is not going to be indefinite even if objectives are achieved.

Reduce risks: daily prescriptions, monitor and urine test

There are risks attached to the prescription of opioids, in any population. These include erratic consumption, hoarding of medication and diversion of supply. If you are prescribing for high-risk individuals, consider using a prescription that allows dispensing of daily doses of

Figure 1. A sample multi-dispensing prescription for a controlled drug.



medication: in England, an FP10MDA (see Figure 1). These can be used for all controlled drugs, not just opioid substitution treatment.

Regular urine testing can confirm the consumption of prescribed medication and alert you to the presence of non-prescribed drugs.

Summary

This is a difficult area to get right. Research is poor and opinions are plentiful. The best we can hope for is to get it 'least wrong'. You need to suspect drug dependence; assess for level of use, withdrawal and harm; corroborate self-report with GPs, pharmacists and urine tests; maintain opioid substitution treatment where safe to do so; treat pain in addition to the long-term opioids; set SMART goals; review often; and liaise with the GP and drug services.

Further reading

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Peripheral neuromodulation: part 2: somatosensory, head and facial pain

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SAGE

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A doctor demonstrating electrotherapy on a young woman (from the Wellcome Collection L0012521).

This is part 2 of a 3-part article on the history, current practice and future directions of peripheral neuromodulation.

In part 1, we previously covered the theory of peripheral nerve stimulation.

In part 2, we present the role of peripheral modulation for specific clinical indications.

Stimulation of nerve plexuses

Brachial plexus stimulation

Neuropathic pain in the upper limb is often difficult to treat effectively.[1] The upper limb is a very good target for neuromodulation. The peripheral stimulation of the brachial plexus is not only simpler but also seems to be more effective compared to SCS, dorsal root ganglion (DRG) stimulation and deep brain stimulation (DBS).

The first patient who underwent peripheral stimulation of the brachial plexus had extremely severe neuropathic pain (10/10) caused by brachial plexus injury; the pain co-occurred with arm paralysis on the affected side. A preliminary, direct stimulation of the brachial plexus with low-frequency electric current (2 Hz) for 5 minutes relieved the pain by 95% for 7 hours.[2] Subsequently, the stimulation electrode was inserted percutaneously into the brachial plexus from posterior access. Using the electrode in that site, stimulation with electric current at a frequency from 2 to 10 Hz reduced the pain by 95%, which was similar to the effect of the preliminary stimulation. Notably, after the treatment with the percutaneous electrode, allodynia resolved within several hours after the procedure, and a normal sense of touch returned within several weeks. The arm function continued to improve slowly over the next 3 months.[2] Currently, insertion of stimulation electrodes via the medial supraclavicular access under the guidance of stimulation, ultrasonography or fluoroscopy is the method of choice.[3] This method is effective and much easier than insertion from the posterior approach. An interesting variant of the medial approach, which involves ultrasound guidance, was proposed by Bouche from Nantes, who successfully gives this treatment to patients who have not responded to SCS.[4] To date, this brachial plexus stimulation (BPS) has been described in about 50 patients. Preliminary or trial stimulation, for up to 2–3 weeks, can be performed in most settings. This stimulation can be achieved with simple, inexpensive catheters that are typically used for continuous peripheral anaesthesia. Using brachial plexus neuromodulation, together with a continuous block to treat patients with upper limb ischaemia, may be a viable therapeutic option.[5] BPS may be considered as an attractive alternative method of nerve stimulation in patients with pain of the upper limb. However, trials to compare the effects of BPS with those of standard treatment are required.

The following images are of BPS and majority of implants are for severe neuropathic pain and/or CRPS (Figures 1–3).

Stimulation of the lumbar plexus/paravertebral stimulation

Stimulation of the lumbar plexus can be beneficial in patients with intractable pain in the hip and knee joints.

It is relatively simple to insert an electrode percutaneously into the lumbar plexus from paravertebral access at the L4 level

Figure 1. Brachial plexus neurostimulation trial, mono-lead. Majority of patients have implants for severe neuropathic pain and/or CRPS. (TG copyright, with permission).

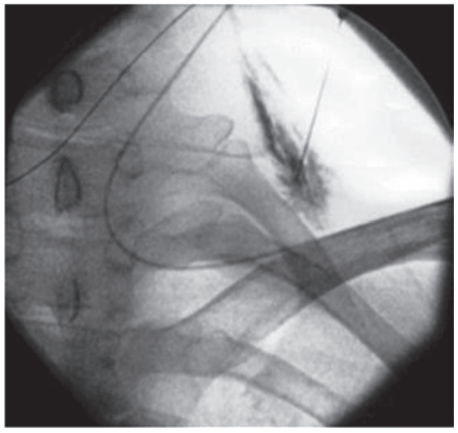
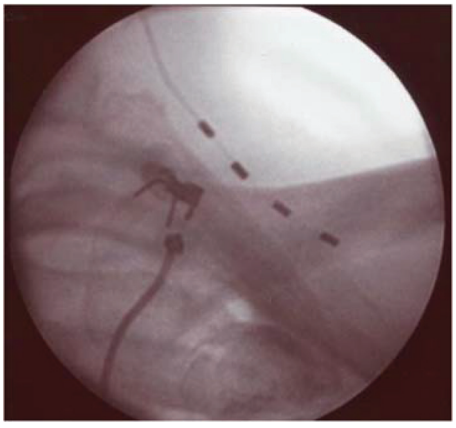


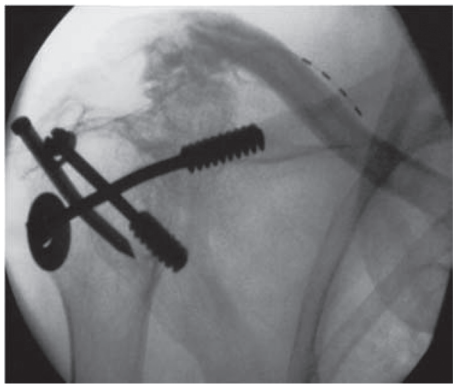
Figure 2. Brachial plexus stimulation (TG copyright, with permission).



with continuous diagnostic stimulation (2 Hz) and direct fluoroscopy or ultrasound guidance. In a small study among patients with knee pain, stimulation of the lumbar plexus relieved pain in three quarters of patients, and, in two patients, pain relief was achieved despite unsuccessful spinal cord stimulation.[6]

Paravertebral stimulation at the level of the chest can be a promising alternative to SCS or DRG stimulation for patients with unilateral chest pain. Paravertebral stimulation offers good electrode stability and substantial pain relief. The paravertebral

Figure 3. Brachial plexus stimulation (TG copyright, with permission).



stimulation is based on the same principle as the standard techniques of paravertebral anaesthesia.[7]

Nerve stimulation for headache and facial pain

Of the 15 occipital neuralgia patients of Weiner and Reed,[8] eight in fact actually suffered from chronic migraine for which it also proved successful. It is suggested that occipital nerve stimulation (ONS) is effective in chronic migraine because the signals from the trigeminal nerve, dura mater and cervical spinal nerves converge in the brainstem.[9]

The activation of the afferent fibres from the caudal portion of the trigeminal nucleus, at the C2 level, can cause pain in the trigeminal and cervical distributions. Thus, it is hypothesised that electrical stimulation modulating the function of occipital nerves can affect the mechanisms of pain in the areas innervated by the cervical nerves and the trigeminal nerve.[9]

The great occipital nerve is a branch of the C2 spinal nerve, and it is an easy target for stimulation-based treatments. In patients with chronic migraine who underwent ONS, position emission tomography (PET) showed increased blood flow in the areas assumed to mediate pain relief, that is, the posterior pons, anterior cingulate cortex and cuneus.[10]

Further series of case reports on the promising effects of ONS in patients with chronic headaches and migraine prompted large controlled trials assessing the effectiveness of this treatment.[10–13]

A total of 66 patients with drug-resistant migraine were enrolled in the ONSTIM study assessing the effects of bilateral ONS. The patients were randomly allocated to receive one of the three following treatments: variable ONS, fixed ONS and medical treatment.[11] Among the patients who received ONS, 39% responded to the variable ONS, and 6% of patients, to

fixed ONS. Patients who received medications did not improve.

There may be a large placebo response as indicated in the PRISM study, when 132 patients were randomly allocated to undergo either nerve stimulation or sham stimulation.[12] The stimulation was given to patients for 12 weeks. The mean reduction in the number of days with migraine was 27% in the patients who received active stimulation, compared to 20% in those who underwent the sham stimulation, which was not significant.

In another study, 157 patients with refractory migraine were randomly allocated to receive either active stimulation or sham stimulation.[13] The results showed there was a significant difference between the groups that received either active or sham stimulation in achieving at least a 30% (but not 50%) reduction of headaches. This difference translated into a reduction in the number of days with headache by 3 days during a month and a decrease in the Migraine Disability Assessment Scale (MIDAS) scores by 44 points.

Cluster headache – ONS

Because the hypothalamus is known to be active during cluster headaches, it was the target of the first neuromodulation attempts to treat patients with cluster headaches who did not respond to medications.[14] However, the hypothalamus stimulation led to complications, and new targets for nerve stimulation in patients with cluster headaches were tested.[15,16] In patients with cluster headaches, PET showed an increased metabolism in the hypothalamus, pons and midbrain. This increased metabolism could be reversed by the stimulation of the occipital nerve.[17–21] A randomised, controlled trial comparing low-frequency and high-frequency paraesthesia is ongoing.[22]

Cluster headache – stimulation of the sphenopalatine ganglion

Stimulation of the sphenopalatine ganglion (SPG) is another neuromodulation target for cluster headache. The SPG lies in the pterygopalatine fossa, and the post-ganglionic parasympathetic and sensory fibres originating from the ganglion run along the blood vessels supplying the face, dura mater and brain. Initially, blockade and radiofrequency ablation of the SPG was used to treat patients with cluster headache who did not improve with standard treatment.[23,24] The SPG was chosen as the target for neuromodulation because, in animal studies, the electrical stimulation of this ganglion reversed hypoxia and increased blood flow in the relevant area.[25] A study among 28 patients with chronic cluster headache resistant to standard treatment tested the effects of a neurostimulator that was implanted through the mouth, with the tip of the electrode placed in the pterygopalatine fossa.[26] This device was controlled externally via a radiofrequency transmitter. The treatment with the SPG stimulator alleviated cluster headaches in 67.1% of patients, and it reduced the frequency of cluster headaches in 36% of all patients.[26] This improvement in cluster headache symptoms suggests that stimulation of the SPG is effective during acute episodes of cluster headache and can be also used in the prophylaxis of these headaches. Temporary sensory disturbances in the face were the most common adverse effects of the SPG stimulation.

Facial pain

Nerve stimulation, in the treatment of patients with facial pain, involves the stimulation of nerves located in the pain centres and pathways transmitting pain signals such as the trigeminal ganglion and its branches.[27–29] Clinical indications include trigeminal neuralgia, post-stroke pain, peripheral nerve injury, and post-herpetic neuralgia. Interestingly, in a case series, Taub et al.[30] observed that stimulation of the trigeminal ganglion successfully relieved pain in five of seven patients after stroke, but it did not improve post-herpetic neuralgia in any patient. Transcutaneous supraorbital nerve stimulation (tSNS) is promising, and it may prove to be an effective treatment for patients with migraine.[12,31,32]

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Informing practice

A cautionary tale on standards of medical recordkeeping: current medical practice versus the law – the law wins!

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THE BRITISH PAIN SOCIETY
EXPERTISE WHERE IT MATTERS

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SAGE

The problem created for a clinician by the clinician's inadequate note and record making, and indeed, inadequate letters and websites, is illustrated in a recently reported case. If a dispute arises, as in a claim for damages or in fitness to practise proceedings, as to what a clinician said to a patient, or what the clinician actually did on a particular occasion, for the clinician to have to rely on her recollection as to what occurred, or to rely on her having carried out her 'normal practice', or even 'invariable practice', is extremely unwise and highly unreliable.

To a lawyer that is obvious, particularly when operating in a field where proof is 'on the balance of probabilities', but it is apparent that it is not so obvious for many clinicians. Repeated examples of inadequate notes and records arise, and of inconsistency in giving evidence, with all the unfortunate consequences that follow. And yet, proper, complete, comprehensible and very brief notes will overcome those problems and probably ensure a better quality of practice.

Hassell v Hillingdon Hospitals NHS Foundation Trust[i] concerned a spinal orthopaedic surgeon who performed a C5/6 decompression and disc replacement operation on his patient. Unfortunately, she suffered a spinal cord injury during the operation which caused tetraparesis and rendered her permanently disabled. A dispute arose as to whether the surgeon had warned the patient that the operation might leave her paralysed and whether he had discussed with her other conservative treatments before the decision to have the operation was made, all of which he was required to do when consenting a patient following the decision in *Montgomery.*[ii] Although the surgeon asserted that he had warned the patient about the risks of paralysis and also discussed other conservative treatment options, the patient's claim succeeded on the basis of a failure to obtain informed consent and she recovered substantial damages.

Credit: Epitavi.

At the trial, the judge had to decide what passed between the surgeon and the patient. In his judgement, he gave seven reasons why he concluded that, on the balance of probabilities, the patient's recollection of events was to be preferred. In considering these reasons, one can gain an insight into the way a judge's mind (indeed any lawyer's mind) works in establishing probable (and therefore, for court purposes, proved) facts.

First, the surgeon claimed in evidence to have discussed conservative treatment options, including physiotherapy, with the patient and that he understood that she had already had physiotherapy for her neck. This was a misunderstanding, as her previous physiotherapy had not been for her neck and upper arm issues, and the judge concluded that had there been a proper discussion with her about other treatment options, the true situation would have become clear.

Second, in his witness statement, which was provided previously for the purposes of the litigation, the surgeon said he would have mentioned the possible complications of deep vein thrombosis (DVT) and pulmonary embolism (PE), it being his 'invariable practice' to mention them in the context of a cervical discectomy. In contrast, in his oral evidence in court, he did not refer to possible DVT or PE when addressing what occurred. This inconsistency led the judge to conclude that although the surgeon believed that it was his invariable practice to do so, in fact, what he said did sometimes differ from his 'invariable' practice. Although the occurrence of a DVT or PE was not at the heart of the case, that inconsistency was relevant when considering the surgeon's evidence as a whole.

Third, the judge found that the patient's recollection was clear and carried weight. As a working mother of three children, she could have been expected to recall the serious risk of paralysis if mentioned and her account was consistent with the situation described by her in her letter of complaint.

Fourth, the surgeon wrote in a letter following the surgery that the operation could result in paralysis and that the risks were similar to those explained to the patient for previous spinal surgery. In fact, the earlier letter in relation to the previous surgery made no reference to the risk of paralysis.

Fifth, although the surgeon mentioned the possibility of further injections as an alternative treatment in his oral evidence in court, he had not mentioned this in his earlier witness statement. The judge found that these inconsistencies made the surgeon's evidence unreliable.

Sixth, the surgeon's evidence that he referred patients to his website to understand better the risks and benefits of the surgery was not helped by the fact that the website in fact omitted any reference to the risk of paralysis.

Finally, in a letter dictated by the surgeon, in front of the patient, prior to the surgery, there was no reference to the risk of paralysis, again raising doubt as to whether that risk was in fact mentioned to the patient.

To a lawyer, accuracy and consistency are crucial in providing credible evidence and therefore such inconsistencies as the judge found would inevitably be crucial in preferring the recollection of events as put forward by the patient.

Lessons to be learnt

It is therefore essential that clinicians record the essentials of any communication with a patient in a clear and reliable form at the time it is communicated, so as to be able to assert confidently at any later inquiry what that communication involved. This can readily be facilitated by preparing in advance a checklist of matters to be raised with the patient, whether a full list or an acronym, and whether on paper or electronically, and using some simple and effective system, such as ticks or crosses, to show that they were in fact raised. It is also crucial that the clinician gives a consistent account of what took place at every occasion that the matter is addressed, in correspondence, on websites, in witness statements and in oral evidence.

Although this example relates to the process of consenting, the same advice applies to all other areas in which a clinician works. For example, if a clinician, in a subsequent inquiry, is asked to justify a decision made in the treatment of a patient, then a contemporaneous brief note of the material facts relied on, the decision made and the reasons for the decision, will stand the clinician in good stead and frequently provide convincing evidence as to why a complaint or a claim against the clinician will not succeed.

Notes

i. [2018] EWHC 164, http://www.bailii.org/ew/cases/EWHC/QB/2018/164.html
ii. Montgomery v Lanarkshire Health Board [2015] SC 11.

Informing practice

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SAGE

Psychological predictors of successful outcomes after interventional pain procedures for chronic lower back and radicular pain

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Introduction

Lower back pain (LBP) and lumbo-sacral radicular pain represent a common and significant problem in the western world with huge economic and social costs.[1,2] Therapeutic options for the management of LBP and/or sciatica are the following: surgical, pharmacological, physiotherapy, psychological therapies and targeted injection treatments.[3]

Various interventional treatments exist for LBP and lumbar radicular pain. In the lumbar region, multiple structures can be a source of pain. Pain originating in the facet joints or related structures can be diagnosed by diagnostic block of the medial branch nerves of the dorsal rami. Injection therapy for facet joint pain includes facet joint injection of steroid with or without local anaesthetic, and radiofrequency ablation of the medial branch of the dorsal rami. Needle-based interventions for lumbo-sacral radicular pain include nerve root injection of steroid, transforaminal steroid injection and interlaminar/caudal epidural injection.[4] The range of procedures performed at our institution and included in this study will be examined later in this article.

The above procedures are practised in large numbers in many countries, but their overall effectiveness continues to be challenged.

LBP is complex and multiple structures and multiple pain-generating mechanisms may be involved. For some of these, no well-evidenced needle-based therapies exist. It is not often possible to accurately establish the pathoanatomic cause of a patient's lower back or lumbo-sacral radicular pain, such cases often being termed 'non-specific'. History and physical examination offer poor accuracy for establishing the cause of patients' symptoms in lower back and radicular pain.[5,6]

The effectiveness of injection therapy for LBP and radicular symptoms is debated, with some studies and meta-analysis finding no strong evidence of effectiveness.[7] There is a large degree of heterogeneity in the populations studied and a lack of reliable predictors of response to injection therapy.[8]

The biological aspects of chronic pain do not exist in isolation, and the biopsychosocial approach to pain takes into account the patient's cognitive, emotional, and behavioural context.[9]

Given the widely accepted contribution that psychosocial factors play in the development and maintenance of chronic pain states, we hypothesised that these factors may also contribute to the response to needle-based interventions. We aimed to elicit any association between psychological factors commonly measured in the pain clinic and response to therapeutic injection treatments for chronic lower back and radicular pain.

Methods

Consecutive patients with chronic lower back and/or lumbar radicular pain who underwent spinal interventional procedures performed by the same pain physician between April 2013 and March 2015 were identified. Exclusion criteria included interventions for cancer pain, incomplete pre-procedure screening data or technical failure of the procedure. Data were collected retrospectively from questionnaire-based psychosocial evaluation forms, which are routinely completed in all new patient assessments at our institution.

Pre-intervention pain and psychological evaluation included the following:

- Brief Pain Inventory short form (BPI) for multidimensional pain assessment;[1]
- Hospital Anxiety and Depression Score (HADS) to screen for depression and anxiety;
- Pain Self-Efficacy Questionnaire (PSEQ) to assess the patient's confidence in performing tasks despite pain;[10]
- EuroQol-visual analogue scale (EQ-VAS) to assess self-reported quality of life.

Table 1. Number of procedures included for analysis.

Type of procedure	Number of patients (total 176 patients)
Epidural type injections	93
Radiofrequency ablation	37
Intra-articular injections	46

Patient selection

Patients were selected for interventional procedures by consultant pain physicians experienced in the assessment and treatment of chronic pain in a large tertiary chronic pain clinic in the United Kingdom. The physicians had access to the pre-procedure screening information and performed their assessment independent of this study. Patients were selected for epidural type steroid injections by clinical judgement including the following criteria: history of radicular/neuropathic signs, symptoms supporting nerve root irritations such as positive straight raised leg test or magnetic resonance imaging (MRI) finding supporting nerve root compression. Radiofrequency ablation was performed after one positive (50% pain reduction within first 6 hours) diagnostic medial branch block. Intra-articular facet joint injections were performed if patient presented history of LBP and symptoms such as paraspinal tenderness associated with muscle spasm. Sacroiliac joint (SIJ) injections were performed on the basis of LBP localised below the L5 level with at least one positive SIJ stress test.

At 3 months post-procedure, a pain clinic specialist nurse not previously involved in the patient's care performed telephone follow-up to establish the outcome of the procedure.

Primary outcome

The primary outcome was success of the interventional procedure as defined by >50% reduction in the targeted pain at 3 months post-procedure. This outcome allowed standardisation between different pain levels pre-procedure. This was assessed by direct verbal questioning – 'has your pain reduced by more than 50%?'

Statistics

Data from the primary outcome measure will allow comparison of pre-implantation differences between two subgroups: group A, which achieved the primary outcome of >50% pain reduction at 3 months, and Group B, which did not.

Analysis of the pre-implantation factors between group A and B was conducted as follows. Continuous variables with normal distribution are presented as mean ± standard deviation (SD) and the comparison of means for such data (age of patients) was performed by t-test (normal distribution confirmed with Kolmogorov–Smirnov test). Categorical data are described as percentages and compared with chi-square test. Ordinal variables (BPI, HAD, PSEQ and EQ-VAS) are presented as median (lower quartiles, upper quartiles) and analysed with Mann–Whitney test.

Subsequently, ROC (receiver-operating characteristic) curve analysis for parameters that achieved statistical significance across two subgroups was produced for identifying optimal cut-off values. The population was subdivided into new subgroups above and below the ROC-derived threshold and univariable and multivariable logistical regression was performed to find dependent and independent pre-implantation predictors. A p value <0.05 was considered statistically significant. All analyses were performed using MedCalc 14.8 statistical software (MedCalc Software, Ostend, Belgium).

Results

Retrospective analysis of 229 consecutive patients who fulfilled the selection criteria was performed. After reviewing patients' notes, 53 patients were excluded due to incomplete screening data available, leaving 176 patients included for further analysis.

The following interventional procedures were included in the study: 93 epidural type injections (interlaminar, transforaminal and caudal), 46 intra-articular injections (facet joint injections, SIJ injections) and 37 radiofrequency ablation to lumbar medial branches (Table 1).

Out of 176 patients, 58 achieved defined primary outcome (Group A) – 50% pain reduction at 3 months post-procedure, Group B – 118 patients with pain score reduced less than 50%. There were no significant age and gender distribution differences between groups. Age of group A: 58.4 years, SD = 16.5 versus group B: 57.7 years, SD = 16.9, p = 0.796; Females in group A: 30/58 versus group B: 74/118, p = 0.218.

Scores from BPI–severity and HAD-A showed no statistically significant difference between groups A and B (median BPI–severity Group A = 6.5 vs Group B = 7, p = 0.058 and HAD-A Group A = 8.5 vs Group B = 11, p = 0.072; Table 2).

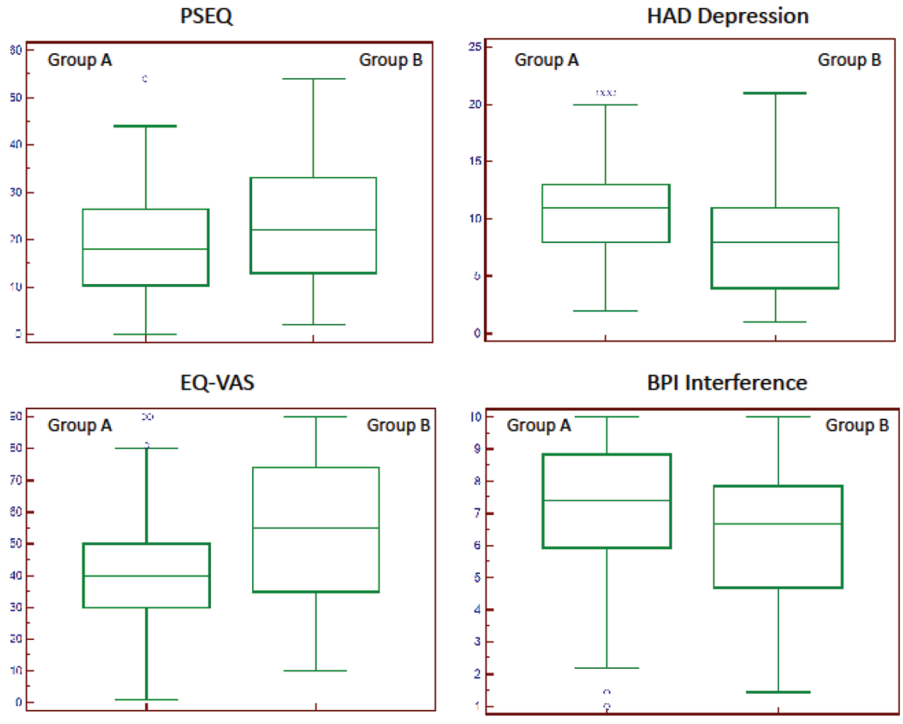
Scores for PSEQ, HAD-D, EQ-VAS and BPI–interference differed significantly between groups A and B (PSEQ p = 0.047, HAD-D p = 0.0086, EQ-VAS p = 0.0009 and BPI-interference p = 0.0082, respectively) and were included for further analysis (Graph 1).

Table 2. Statistical analysis of psychological factors.

Screening tool	Group A >50% pain relief	Group B <50% pain relief	p value
BPI–severity	Median = 6.5	Median = 7	0.058
BPI–interference	Median = 6.7	Median = 7.49	0.0082
HAD-A	Median = 8.5	Median = 11	0.072
HAD-D	Median = 8	Median = 10	0.0086
PSEQ	Median = 21.5	Median = 18	0.047
EQ-VAS	Median = 55	Median = 40	0.0009

BPI: Brief Pain Inventory; HAD-A: Hospital Anxiety and Depression–Anxiety; HAD-D: Hospital Anxiety and Depression–Depression; PSEQ: Pain Self-Efficacy Questionnaire; EQ-VAS: EuroQol-visual analogue scale.

Graph 1. Box and Whisker plots present differences in medial values of PSEQ, HAD depression, EQ-VAS and BPI (interference subsection) between the two groups (p = 0.047, p = 0.0086, p = 0.0009 and p = 0.0082, respectively).



Those variables that demonstrated significant difference between groups A and B (PSEQ, HAD-D, EQ-VAS and BPI-interference) were analysed using an ROC curve (Table 3). ROC curve analysis identified optimal cut-off values to subdivide patients to below and above threshold subgroups (Graph 2).

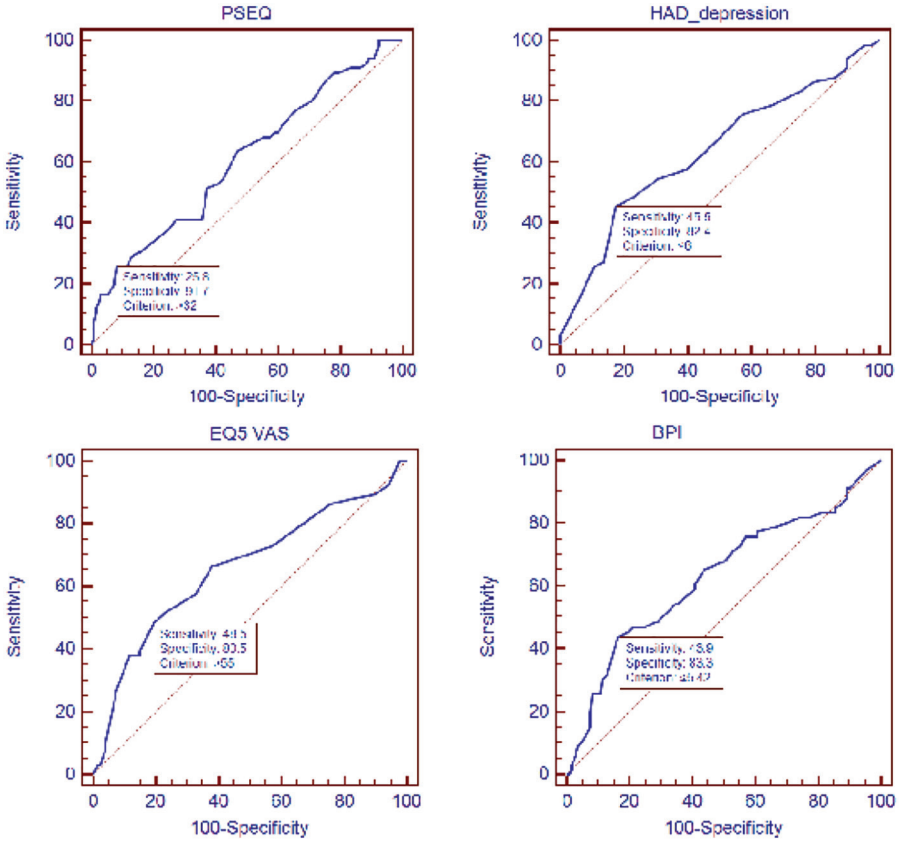
The population was subdivided into new subgroups above and below the ROC-derived threshold for multiple logistic regression analysis. EQ-VAS > 50 was the variable most predictive of success of achieving the primary outcome and therefore EQ-VAS < 50 is an independent risk factor for failure

Table 3. ROC curve analysis.

Screening tool	Cut-off value	Sensitivity (%)	Specificity (%)
BPI–interference	⩽5.42	43.1	83.9
HAD-D	⩽6	44.8	81.9
PSEQ	>18	62.1	53.4
EQ-VAS	>50	51.7	78.9

BPI: Brief Pain Inventory; HAD-D: Hospital Anxiety and Depression–Depression; PSEQ: Pain Self-Efficacy Questionnaire; EQ-VAS: EuroQol-visual analogue scale.

Graph 2. ROC curve analysis of PSEQ, HAD-D, EQ-VAS and BPI–interference to identify optimal cut-off values to subdivide patients to below and above threshold subgroups.



of interventional procedure (p=0.0268, odds ratio (OR)=2.61, 95% confidence interval (CI), (1.1 to 6.1)).

Discussion

In our study, neither BPI–severity nor HAD-A were found to be statically different between those who achieved the primary outcome of >50% pain reduction and those who did not. This implies that the severity of a patient's pain as self-reported pre-procedure was not predictive of response to treatment. Pre-operative pain severity and psychological factors have previously been found to correlate with chronic pain following total knee arthroplasty (TKA)[11] and knee arthroscopy,[12] but our study did not show this to be the case with needle-based

interventions. Anxiety has been identified as a risk factor for the development of chronic pain after surgery in some studies, both for patients with pre-operative pain[13] and those without pre-operative pain.[14] Despite anxiety being a risk factor for post-operative pain, our study did not find a significant difference between the anxiety scores of responders and non-responders.

Similar to our findings, a 2017 review of post-operative risk factors for chronic pain after total knee replacement (TKR) did not find sufficient evidence for any psychological risk factor for the development of chronic post-surgical pain.[15] The effect of psychological variables on outcomes following spinal surgery or spinal cord stimulation was the subject of a 2009 systemic review.[16] This review found that symptoms of prior somatisation, depression, anxiety and poor coping were most predictive of poor response to surgery or spinal cord stimulation; however, activity interference and pre-treatment pain intensity were minimally predictive.

Our study found EQ-VAS >50 to be the most predictive of successful outcome following interventional procedures for chronic back and radicular pain. While this metric is not classically a psychological or pain screening tool, it is widely used in multidimensional assessment of patients with chronic pain and other patient-reported outcome measures (PROMS). The assessment tool is used as a quality of life assessment and its advantages are ease of use, simplicity and generally high validity.[17] The relationship between the EQ-VAS score and other psychological metrics is not well understood and the EQ-VAS may be influenced by the psychological profile of the patient. Co-existent psychological characteristics relating to the patients' overall health-related outlook may impact on their self-reported global assessment of health.

Our study found the sensitivity of EQ-VAS as a predictor to be 51.7%, which falls well below generally acceptable limits for a screening tool. Almost 50% of predicted successful outcomes will be missed using EQ-VAS >50 as a cut-off value. Specificity was more encouraging at 78.9%, meaning that an EQ-VAS >50 results in a low level of false-positive predictions of success. Based on these numbers, if an EQ-VAS >50 was used as a strict selector of whether to proceed with interventional procedures, we would expect the rate of successful procedures to increase significantly. However, many people (approximately 50%) who would benefit from intervention would be denied the treatment.

Limitations of study

There are several limitations to this study. The pre-procedure variables analysed were available to the clinician selecting patients and therefore may have influenced the selection of patients. The methods used by the clinician to select patients for procedures were based on clinical assessment and judgement as well as patient preference, which may introduce bias. There are inherent limitations to a retrospective study of this nature. The collection of outcome data was, however, blinded to other elements of the study.

As the data were retrospectively analysed only from complete data sets, 53 out of 229 patients were not included due to incomplete data available. This represents 23% of patients, and as the screening metrics of this group are unknown, they may possess a different profile to the remaining population included in the study.

Conclusion

The pre-procedure variables analysed in this study and the underlying characteristics that they measure are known to impact on chronic pain states. We are not aware of another study that has analysed the impact of such variables on outcomes following interventional procedures for chronic pain and as such it represents a significant contribution to the literature.

Interventions for chronic back pain and radicular pain have good efficacy in certain patients; however, robust methods of selecting these patients are not clear. It would be immensely useful to identify such factors in order to better target interventions to patients most likely to respond.

This study has identified an EQ-VAS score of >50 out of 100 as an independent predictor of successful intervention pain treatment. This could be of use when selecting patients for interventional pain procedures for lower back and radicular pain and therefore we support its inclusion in the multidimensional pain assessment.

There was no strong evidence for any of the other screening variables examined as predictive factors in chronic pain interventions in this study.

The suboptimal sensitivity (51.7%) of EQ-VAS >50 demonstrated in this study may limit its clinical utility as a predictor of outcome and this single measure should be interpreted within the context of a multidimensional pain assessment and clinical judgement when selecting patients for interventional procedures.

Further analysis, larger sample size, and prospective sampling and evaluation are needed to further establish the significance and validity of these findings.

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The British Pain Society is nothing without you, its members, and we appreciate your continuing involvement and support. We recognise that, for many members, in recent years, the decision to pay the membership fee for a non-compulsory professional society has been more challenging so we will continue to look closely at our fees and we will take care to limit any increases. We hope that you will continue to encourage your colleagues to joins us.

May we also remind you that The British Pain Society is a registered charity and we welcome funds received from legacies and through sponsorship. As we know from the numbers who have joined fun runs at previous ASMs, many of our members are actively engaged in sporting activities. So, if you are signing up for any marathons, half-marathons, triathlons, swims or tiddlywinks contests, please consider nominating The Society as your chosen charity.

Thank you for supporting the BPS!

Book review

THE BRITISH PAIN SOCIETY
EXPERTISE WHERE IT MATTERS

Pain News
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SAGE

***Perioperative Pain Management for Orthopedic and Spine Surgery*, edited by John S Reach, James J Yue, Deepak Narayan, Alan D Kaye, Nalini Vadivelu; Oxford University Press, ISBN: 9780190626761.**

Reviewed by Manohar Sharma, Consultant in Pain Medicine

Perioperative Pain Management for Orthopedic and Spine Surgery is a textbook outlining and detailing perioperative pain management for orthopaedic and spine surgery. It is targeted at trainees in pain medicine, anaesthetics, orthopaedic and spinal surgery.

The authors have set themselves a wide remit of not only covering perioperative pain management for orthopaedic and spine surgery but also addressing chronic pain management.

The first five chapters of this book are specifically dedicated to Pathophysiology of Pain and Pain Pathways, Preventive Analgesia for the Management of General Surgical Pain, Perioperative Nonopioid Analgesics of Use in Pain Management for Spine Surgery, Perioperative Opioid Analgesics of Use in Pain Management for Spinal Pain Surgery and Local Anaesthetics.
Chapter 6 refers to Susceptibility of Peripheral Nerves in Diabetes to Compression and Implications in Pain Treatment. These initial six chapters provide excellent reading to bring together these various aspects to improve perioperative pain management in orthopaedics and spine surgery.

The next 13 chapters of this book cover various aspects of chronic pain assessment and management options. These include assessment and management of patients with Cervical Pain, Thoracic Spinal Pain, Lumbosacral Pain, Lumbar Neurogenic Claudication, Needle Placement Techniques for Chronic Pain Interventions as well as Disc Treatment Techniques, Sympathetic Pain Syndrome, Endoscopic Medial Branch Rhizotomy and Spinal Cord Stimulation and Intrathecal Drug Delivery, in particular, the delivery techniques.

The authors are to be commended by providing information on chronic pain assessment and chronic pain intervention techniques. The authors have covered the implications for surgeons when they are operating on the spine on someone implanted with a spinal cord stimulator or intrathecal pump and what precautions to take. This makes the book relevant to clinical practice as we will now all see more patients being offered neuromodulation techniques for chronic pain management who then need spine or orthopaedic surgery.

This book also provides extensive updates in the field of chronic pain medicine such that it is also relevance for practitioners interested in chronic pain management.

Chapter 19 on 'Long Overdue Paradigm Disruption with the Interventional Pain Management Ladder' is thought provoking. They suggest that pain management techniques should be brought forward in the patient pathway for chronic spinal pain in context of spinal surgery. The reason given is that pain interventions techniques are less invasive and less disruptive to the human anatomy with lower risk of further aggravation of chronic pain. The treatments are reversible, and the patients still can be offered further spine surgical options if required in future. This is further helped by easing of restriction on MRI scans for patients implanted with these devices.

Overall, the authors have done well on bringing together Perioperative Pain Management for Orthopaedic and Spine Surgery and integrating with chronic pain management options for this group of patients.

Unfortunately, the title of the book does not reflect the excellent content of the book. This text book recognises the need for clinicians to be well rounded in their knowledge in spinal and orthopaedic pain management not only in the perioperative period but also in the longer term for chronic pain management. This book is recommended.

How not to be a doctor

John Launer

THE BRITISH PAIN SOCIETY
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'*How can I help you?' I asked. It isn*'t the way I always open consultations, but I was making a teaching video, so I thought I would be conventional for a change. As it turned out, it was a fortunate move. '*I'm not sure if you really can help me*', the patient answered. '*I've seen lots of specialists, and none of them have managed to help me so far. You see, I keep having these funny turns …*'. Two weeks later, when showing the video to a group of senior house officers, I stopped the recording at this point and asked them to write down the woman's presenting complaint. All 10 of them wrote down 'funny turns'. They were wrong, of course. The woman's presenting problem was that she wasn't sure if I could really help her. The funny turns were at this point a lesser problem.

There were more shocks in store for the group. I spent almost the entire consultation asking the woman about her experience of other doctors, and what they had got wrong. I listened as dispassionately as I could, without dismissing her catalogue of disappointment or offering any hint that I might do any better myself. In the end, I asked her what she thought the doctors ought to have done instead. She told me, '*a referral for homoeopathy or acupuncture*'. I asked her which of these she would prefer. She chose the homoeopathy referral, and I said I would arrange this. As she left, I thought she was going to cry with relief.

After I had finished showing the video, one junior doctor erupted. How could I have been so incompetent – not to take a full history, or indeed any history at all? How could I be so irresponsible, by assuming that the other doctors had done their job properly? How could I be certain that her funny turns did not presage some terrible, terminal disease? If I thought the problem was psychosomatic, why didn't I take a decent psychiatric history instead? And how could I possibly direct her, without a clear diagnosis, towards a form of treatment that I probably didn't believe in, and which lacked a thorough evidence base?

A number of other doctors in the group came to my defence. Some had realised that I might have looked at the notes in advance, and that I might be willing to trust local colleagues not to make gross errors of judgement. Others had heard the patient mention that she had gone through the mill of extensive and futile investigations several times over. One or two had noticed how the patient gave indications of an aversion to anything remotely suggesting psychological inquiry. A particularly thoughtful doctor pointed out that no intervention was without its risks; at this stage, it would probably cause the patient more risk if I started all over again, instead of just doing what she wanted. Yet, their sceptical colleague remained unconvinced. How could I have behaved so … so … well, so unlike a doctor? I took the question as a compliment.

Of all professions, doctors are almost invariably the most proficient at not listening. Indeed, a friend of mine sometimes describes my educational work in consultation skills as '*remedial therapy for selective brain damage*'. It is a cruel characterisation, but I do not entirely object to it. I am struck again and again by how much medical listening – even the kind that sometimes passes for being 'patient-centred' – falls desperately short of anything that one might expect from an attentive, untrained friend. Many doctors seem to tune out totally from any words or phrases that do not fit the medical construction of the world. In addition, most appear to be extraordinarily timid about going where the patient wants to lead, for fear that this will break some rule, or upset any other doctor who might hear about it.

When it comes to unexplained symptoms, I often observe doctors fall back on an impoverished list of questions such as '*are you under any stress?*' rather than displaying any true curiosity about the story itself. There are two other common consultation ploys that bring me out in an allergic reaction. One is the question '*How did you feel about that?*'. It is generally asked as the doctor asking leans forward in a theatrical pose of solicitousness, but with eyes glazed over in weary automatism. The question seems to go with a belief that it will elicit some definitional nugget of truth, accompanied by a sublime catharsis on the part of the patient. It arises, I guess, from some ghastly misreading of Freud's more minor followers, but 99 times out of a 100, it is emotionally bogus. The other manoeuvre that I find equally offensive is the phrase '*It sounds*

as if …' (as in '*it sounds as if you're very upset* …'). Believe me, if it's so bloody obvious that even a doctor has noticed, it usually isn't worth saying.

Lois Shawver, a Californian therapist and teacher whom I much respect, has come up with a wonderful distinction between 'listening in order to speak' and 'speaking in order to listen'. In the former, you merely scan the words that patients are saying, looking for opportunities to dive in and tell them what is 'really' going on. In the latter, you do the opposite: speaking only in order to give them more opportunities to explain their own view of the world. In a post-modern age where the authority of professional knowledge is gradually waning away, Shawver argues that we will have to learn how to speak less and listen more.

In the same vein, the late Harry Goolishian, one of the founders of narrative approaches to psychiatry, offered the advice: '*Don't listen to what patients mean, listen to what they say!*' Quite simple really, except that we probably still fail to do this, most of the time.

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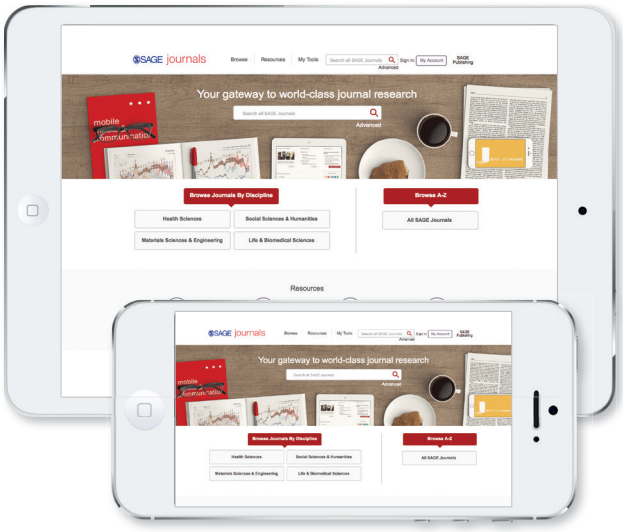

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