Anaesthesia and Pain Management
RESEARCH REVIEW™

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Issue 1 – 2019

In this issue:

Abbreviations used in this issue
CRPS = complex regional pain syndrome
ECR = emergency clot retrieval
QST = quantitative sensory testing
RCT = randomised clinical trial
TIVA = total intravenous anaesthesia
TKA = total knee arthroplasty
TOFR = train-of-four ratio

Welcome to this issue of Anaesthesia and Pain Management Research Review.

This issue begins with an interesting study investigating the perineural administration of dexamethasone combined with ropivacaine for interscalene brachial plexus block, demonstrating a dose-response relationship. We also review a study investigating the effect of anaesthetic agents on outcomes in patients undergoing hepatectomy for hepatocellular carcinoma and receiving desflurane or propofol.

We welcome a guest reviewer for this issue, Dr David Rice. Among his selections is a paper presenting data on the impact that an opioid safety intervention for US veterans has had on pain and opioid prescriptions following TKA (total knee arthroplasty). This issue concludes with research reporting that exposure to chronic insufficient sleep may increase chronic pain via alterations in pain habituation and sensitisation processes.

We hope you find this selection of research enlightening, and we welcome your feedback and suggestions.

Kind regards,

Dr John Barnard
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Dose-response relationship of perineural dexamethasone for interscalene brachial plexus block

Authors: Albrecht E et al.

Summary: This randomised, placebo-controlled trial tested whether perineural dexamethasone 1–4mg could dose-dependently prolong analgesia duration in 80 patients with an ASA physical status of 1–2 undergoing shoulder arthroscopy under general anaesthesia with ultrasound-guided interscalene brachial plexus block. The median duration of analgesia (time between block and first analgesic request) was dose-dependently prolonged (835, 904, 965 and 2013 min for dexamethasone 1mg, 2mg, 3mg and 4mg, respectively, vs. 685 min for placebo [p=0.03]).

Comment (JB): How dexamethasone prolongs the duration of local anaesthetic-induced nerve 'block' remains uncertain. While working better when given with the local anaesthetic, it is surprisingly effective at prolonging block, even when given intravenously. Perhaps it has more than one mode of action in this regional analgesia context, a peripheral effect requiring high concentrations, and a central effect requiring much lower tissue concentrations. The relative exposure of the target nerves to dexamethasone (in this case with an interscalene brachial plexus block) must be orders of magnitude different because similar milligram doses of the steroid are given regardless of the route of administration, intravenous or perineural. These authors filled in a gap in the knowledge base demonstrating a clear perineural dose-effect relationship across the range 1 to 4mg. Previous work had indicated there is probably not much to be gained in block duration by administering doses higher than 4mg. My impression is that more targeted regional analgesia techniques have supplanted interscalene block as the preferred option for most shoulder surgery, avoiding the numb arm, the impaired hemidiaphragm, and the occasional Horner's syndrome. Also, I start getting anxious if a single-shot brachial plexus block lasts longer than 24 hours. Which dexamethasone preparation do you use? The 8mg/2mL or the 4mg/1mL ampoule? The latter formulation is cheaper per ampoule (and only 15% more expensive per milligram). Also, according to the Medsafe datasheet, it contains no sulphites, so if you are keen to avoid sulphites as much as possible due to potential neurotoxicity and allergy concerns, then the 4mg/1mL would be a better choice. Our department uses the 8mg/2mL but maybe we should swap. It is no great drama to draw up 2 or 3 ampoules for the occasional times an 8mg or 12mg dose is needed. As another brain teaser, if a 'block' solution was accidently injected intraneurally, would having a preservative- and antioxidant-free steroid in the solution increase or decrease the potential for lasting adverse sequelae?

Reference: Anaesthesia 2019;74:1001–8

Abstract

Independent commentary by Dr John Barnard

Dr John Barnard works as an anaesthetist at Waikato Hospital with a part time academic component. In addition to his role in the operating theatres, four years ago he became the Clinical Director of the Hospital Pharmacy and Chairman of the hospital's Medicines and Therapeutics Committee.

Reversal of partial neuromuscular block and the ventilatory response to hypoxia

Authors: Broens SJL et al.

Summary: Thirty-four healthy male volunteers were randomised to reversal of a partial neuromuscular block with neostigmine 1mg/atropine 0.5mg (n=11), sugammadex 2 mg/kg (n=11) or placebo (n=12). Ventilatory responses to 5 minutes of hypoxia and ventilation at hyperoxic isohypercapnia were assessed at baseline, during rocuronium-induced partial neuromuscular block (TOFR [train-of-four ratio] 0.7 measured at the adductor pollicis muscle) and following reversal until the TOFR reached unity. Low-dose rocuronium significantly reduced ventilatory responses to hypoxia. Following full reversal (measured at the thumb), significant persistent residual blunting of the hypoxic ventilatory response was evident. The effect of treatment was not significant (p=0.299) with chemoreflex impairment rates of 45% and 64% following sugammadex and neostigmine reversal, respectively, and 83% after spontaneous reversal to a TOFR of 1.

Comment (JB): The take-home messages are: modest degrees of partial curarisation (TOFR 0.7) with rocuronium significantly impair the ventilatory response to hypoxia; this effect is much more marked than the effect on the ventilatory response to hypercapnia; the impaired response to hypoxia likely represents the neuromuscular blocker inhibiting afferent nerves as they synapse at the carotid body; and even after recovery to a TOFR ≥0.9, the subjects response to hypoxia did not return to baseline values. All the findings except the failure to return to baseline were consistent with previous studies using atracurium or vecuronium. Generating this kind of data requires an impressive respiratory lab setup and a bunch of volunteers willing to be partially paralysed, while remaining completely awake, for an hour or so. There are some fascinating extra bits of detail relating to the need to modify the experimental protocol after the study was underway. For example, the original intention was to expose each subject to two levels of partial curarisation, a TOFR of 0.6 and a TOFR of 0.8. It rapidly became apparent that at a level of 0.6 too many subjects developed upper airway obstruction. Their tidal volumes and the measured end-tidal values of CO_2 values became much too variable to drive the logic of the mass flow controllers. These flow controllers manipulated the inspired gas mixture with each breath to maintain the desired end-tidal CO_2 and O_2 values. Another point of interest was the failure to return to baseline values of hypoxic ventilatory responses, even when the TOFR was 1, and the reversal was achieved by a generous dose of sugammadex. With a TOFR of 0.7, 80% of the subjects noted diplopia, 40% difficulty swallowing, and 10% ptosis.

Reference: Anesthesiology 2019;131:467–76
Abstract

Outcomes of general anesthesia versus conscious sedation for stroke undergoing endovascular treatment

Authors: Wan T-F et al.

Summary: This was a meta-analysis of 23 studies (n=6703) reporting on general anaesthesia versus conscious sedation in patients with acute ischaemic stroke undergoing endovascular treatment. Compared with conscious sedation, general anaesthesia was associated with a lower odds of a favourable functional outcome (odds ratio 0.62 [95% CI 0.49, 0.77]) and higher risks of mortality (1.68, [1.49, 1.90]), pneumonia (1.78 [1.40, 2.26]) and symptomatic intracranial haemorrhage (1.64 [1.13, 2.37]); however, there was no significant difference for recanalisation, vessel dissection/perforation or asymptomatic intracranial haemorrhage. An RCT subgroup analysis revealed general anaesthesia was not associated with a lower favourable functional outcome compared with conscious sedation (odds ratio 1.84 [95% CI 1.17, 2.89]), and there was no significant difference for mortality.

Comment (JB): Anaesthesia support for acute stroke ECR (emergency clot retrieval) services is an important contemporary issue. This is a service where minutes may count. 'Time is brain' is the classic quote (noting that non-functioning brain tissue is surprisingly good at hanging on for a few hours if there is just enough oxygenation and blood supply to maintain viability). The endovascular techniques and the associated management of coagulation are maturing, and the results measured by speed and quality of functional recovery are increasingly compelling. One of the areas of uncertainty is whether conscious sedation is associated with better or worse outcomes than general anaesthesia. The former option allows continuous assessment of neurological function and may allow the procedure to start with less delay, especially if the attending anaesthetist has to come in from home or is already busy in theatre with another urgent case. On the other hand, anaesthesia guarantees less movement and frees the proceduralist up from the need to administer local anaesthetic to the vessel access site(s). If you look beyond the poor quality of the English language in this paper, you will find a useful synthesis of the current literature and an insightful discussion. It is particularly interesting to see that the data from retrospective case series indicate a clear outcome benefit associated with sedation compared to general anaesthesia, whereas the combined data from the three recent RCTs demonstrates the opposite association. For some patients, the zone between a medication regimen intended to achieve conscious sedation and one intended to achieve anaesthesia may be very slim and alarmingly fluid, liable to suddenly lurch towards one state or the other with little change in medication administration. Being in charge of 'the top end' is not for the fainthearted. As of October 2018, only Auckland, Wellington and Christchurch were offering a dedicated ECR service for acute stroke, and I suspect these units were initially set up to facilitate participation in multicentre trials. ECR for acute stroke is rapidly moving from a research endeavour to a standard of care, and this is exposing major questions around equity of access. A national ECR Service Improvement project is underway. No doubt it will be coming to a DHB near you soon.

Reference: BMC Anesthesiol 2019;19:69
Abstract

Propofol-based total intravenous anaesthesia is associated with better survival than desflurane anaesthesia in hepatectomy for hepatocellular carcinoma

Authors: Lai H-C et al.

Summary: This study examined the effect of anaesthetic agents on outcomes in a retrospective cohort of patients receiving desflurane (n=492) or propofol (n=452) during hepatectomy for hepatocellular carcinoma. In total there were 369 deaths (75.0%) with desflurane versus 139 deaths (30.8%) with propofol anaesthesia. Propensity matching of 335 patients in each group indicated that propofol had a better survival with a hazard ratio of 0.47 (95% CI 0.38, 0.59). Subgroup analyses also suggested greater survival in the absence of distant metastasis (hazard ratio 0.47 [95% CI 0.37, 0.60]) or local recurrence (0.22 [0.14, 0.34]).

Comment (JB): Previously, these authors interrogated a large clinical database to examine the effect of mode of anaesthesia, propofol-based TIVA versus desflurane, on outcomes after bowel cancer surgery. This time they used the same study design to look at the effect of mode of anaesthesia on outcome after hepatocellular liver tumour resection. In keeping with their previous bowel cancer work, the patients receiving desflurane did worse, markedly worse, but they were also clearly sicker and had more advanced disease at the time of their surgery than the TIVA patients. The editor included four key points with this study, the first of which was *"Considerable evidence suggests that anaesthetic techniques can influence cancer metastasis and outcomes"*. Really this paper just poses questions rather than providing answers, and as the editor suggests in his fourth key point, good quality RCTs are needed. No amount of statistical manipulation, and these authors did seem to take a 'more is better' approach to generating numbers, will sway the discussion if a large good-quality RCT shows no benefit of TIVA over volatile agents for liver resection in the same clinical context. The discussion and reference sections provide a useful resource of current research and opinion about why propofol might be the better option. Unfortunately, the authors did not really discuss why the attending anaesthetists in their liver surgery centre would choose desflurane over propofol for the sicker patients. Could propofol be therapeutic rather than simply less depressive on the immune system? Would using the combination of propofol and sevoflurane (which seems to be quite popular at Waikato Hospital currently) negate the apparent protective effect of propofol? One interesting factoid from the study's introductory section was that hepatocellular carcinoma is the fifth most common cancer-related cause of death in Taiwan, so better outcomes should translate into lives saved.

Reference: Br J Anaesth 2019;123:151–60
Abstract

Intravenous dexmedetomidine for the treatment of shivering during cesarean delivery under neuraxial anesthesia

Authors: Lamontagne C et al.

Summary: This prospective, randomised, placebo-controlled trial in 80 parturients undergoing caesarean delivery and experiencing shivering under neuraxial anaesthesia tested whether the α2-adrenergic agonist dexmedetomidine would reduce the duration of shivering. Dexmedetomidine reduced mean shivering duration from 17.9 minutes with placebo to 2.6 minutes (difference –15.3 [95% CI –11.2, –19.4). The effect persisted for 15 minutes after dexmedetomidine was administered, with shivering completely stopped in 90% of patients versus 22.5% of placebo recipients (relative risk 4.0 [95% CI 2.2, 7.2]). No adverse effects, including bradycardia, were observed.

Comment (JB): In this study, 50% of the patients had significant shivering, and the 30µg dose of dexmedetomidine was clearly effective at reducing this unpleasant side effect. These were patients that were shivering despite already receiving ondansetron 4mg (given to prevent nausea and vomiting, but the 5HT-3 antagonists also decrease shivering). There was no increase in side effects compared with the control group receiving saline, noting that the single timepoint assessment of rouseability 15 minutes after drug administration does not adequately exclude unwanted sedation. At just over $70 per 200µg ampoule, I presume dexmedetomidine remains too expensive to routinely keep in anaesthetists' drug drawers (maybe we should do a survey to answer that question). It is a useful adjuvant in an expanding range of anaesthesia scenarios. Concurrently there is an anti-opioid wave sweeping the profession, encouraging us to consider opioid-'lite' or opioid-free solutions. Increased usage of this medication both in ICU and anaesthesia seems inevitable, and at some point, unless the purchase price drops, the dollar value of the dexmedetomidine contract must attract Pharmac's attention. Imagine you were given the task of developing a list of restrictions (remembering that the primary purpose of the restrictions is to constrain costs). I doubt that treating neuraxial anaesthesia-associated shivering would make it on to this list of restrictions, despite its obvious effectiveness. That is a pity really. For anaesthetists dexmedetomidine is often a better medication to use than clonidine – faster onset, much more specific for α-2A adrenoceptors, less imidazoline activity, and resulting in less hypotension and bradycardia for a given amount of sedation and analgesia.

Reference: Can J Anesth 2019;66:762–71
Abstract

Patterns of opioid administration among opioid-naive inpatients and associations with postdischarge opioid use

Authors: Donohue JM et al.

Summary: This retrospective cohort study investigated the timing, duration and setting of opioid administration in 148,068 opioid-naive hospitalised patients (191,249 admissions), and explored associations with postdischarge use. Nearly half the admissions (48%) included opioid administrations, which were given for a mean of 67.9% of the patients' hospitalisations. There was considerable variability in the location of administration of first opioid on admission, the timing of last opioid before discharge and receipt of nonopioid analgesics. Outpatient opioid use at 90 days was greater for inpatients who received opioids than for those who didn't, and in those who received opioids <12 hours before discharge compared with those who were opioid-free for ≥24 hours before discharge. The difference in opioid use rates at 90 days for patients with opioid use for ≥75% versus ≤25% was modest. Similar associations were seen for opioid use 365 days postdischarge.

Comment (DR): The opioid epidemic continues to be a crisis for the healthcare system and people of the US, with other countries, including NZ (Pain Ther 2017;6:203–15), also experiencing notable increases in opioid prescriptions and opioid-related deaths over the last two decades. One problem that is increasingly recognised is that the use of opioids to treat acute pain, while highly effective, may also increase the risk of persistent opioid use. This study focused on describing and characterising risk factors for persistent opioid use in nearly 150,000 opioid-naïve patients (defined as no opioid prescriptions in the 12 months prior to admission) admitted to 12 different hospitals over a 5-year period. This included both medical and surgical admissions. While retrospective in nature and relying on electronic health record data (which aren't always entirely accurate!), their very large sample size and detailed health records allowed them to make some interesting observations. The punchline was that, even after controlling for a number of important confounders, opioid-naïve patients who were administered opioids during their stay (48% of all patients) were significantly more likely to become persistent opioid users 90 days after admission (5.9% vs. 3.0%; mean difference 3.0% [95% CI 2.8, 3.2%]). While 3% doesn't sound like a large difference, when applied to the study cohort, this equates to nearly 4500 additional persistent opioid users at 90 days. Most surprising to me was that, across all admissions, nonopioid analgesics were very rarely used before opioids were administered (7.9–22.2% of cases, depending on admission type). I'd like to think this doesn't reflect practice in NZ. Either way, these findings are important and should give all prescribers pause when managing acute pain in the hospital setting.

Reference: Ann Intern Med 2019;171:81–90
Abstract

Impact of an opioid safety initiative on patients undergoing total knee arthroplasty

Authors: Chen Q et al.

Summary: This time-series analysis examined the impact of the Opioid Safety Initiative, which was designed to decrease high-dose prescriptions among US veterans, on pain scores and opioid prescriptions for TKA, using group-level data for 700–850 patients per month over 72 consecutive months covering periods before and after the initiative was implemented. After the initiative was introduced, the patients were slightly older and sicker, but had lower mortality rates. Postoperative pain scores were slightly higher and 871 fewer patients received chronic postoperative opioid prescriptions. Time-series analyses revealed that the mean postoperative minus preoperative pain score increased from 0.65 to 0.81. The respective proportions of patients with chronic postoperative and chronic preoperative opioid prescriptions declined by 20% and 13%, and nonopioid analgesia prescriptions increased. These findings were confirmed in sensitivity analyses.

Comment (DR): This study focused on persistent opioid use before and after TKA, a population in which persistent opioid use has been identified as a problem and an area that we are actively researching in the NZ setting. The researchers examined preoperative and postoperative opioid use and pain scores in patients undergoing TKA before and after a system-wide Opioid Safety Initiative that involved dissemination of new treatment guidelines for chronic pain, face-to-face tutorials with prescribers and (perhaps crucially) a computerised dashboard tool that tracked and visually represented all opioid prescriptions at national, regional, facility and provider levels, so that leadership at each facility could audit and provide feedback on these data. Interestingly, no opioid prescription targets were identified or provided; this was left to the discretion of the individual facilities and providers. While limited by its ecological 'before versus after' design, which may introduce several confounding factors, some impressive reductions in persistent opioid use were observed both before and after TKA following the rollout of this initiative, with a small decrease in mortality rates at 30 days, 90 days and 1 year and a negligible impact on postoperative pain intensity in the first 6 months after surgery. Unsurprisingly, the use of nonopioid analgesics increased. It would be interesting to see if there were any differences in long-term outcomes of pain, function and/or patient satisfaction, but I suspect this may be a follow-up publication. These observations are highly promising and in line with previous findings of a large system-wide reduction in high-dose opioid prescribing after the rollout of the Opioid Safety Initiative. It seems education and transparency might actually work!

Reference: Anesthesiology 2019;131:369–80
Abstract

Ketamine infusions for chronic pain

Authors: Orhurhu V et al.

Summary: This systematic review and meta-analysis included seven RCTs, of which six were at high risk of bias, comparing intravenous ketamine with placebo for neuropathic, mixed and non-neuropathic (nociplastic or nociceptive) pain. Three studies reported that ketamine provided a significant but small analgesic effect assessed using a 10-point numerical rating scale for up to 2 weeks postinfusion (mean difference –1.83 points [p<0.0001]), and three studies reported higher responder rates (proportion with a positive outcome) among ketamine recipients compared with placebo recipients (51.3% vs. 19.4% [p=0.029]). There were no differences based on pain classification or condition. Compared with low-dose ketamine studies and investigations that evaluated non-CRPS (complex regional pain syndrome) conditions, a small, nonsignificantly greater reduction in pain scores was seen with high-dose ketamine (p=0.213) and in participants with CRPS (p=0.079).

Comment (DR): Intravenous ketamine infusions are sometimes used to treat chronic pain that is refractory to other treatments, often with the purported target of reducing central sensitisation through ketamine's effect as an NMDA (*N*-methyl-D-aspartate) receptor antagonist – although other mechanisms and receptor systems have also been implicated. This systematic review and meta-analysis aimed to synthesise the existing evidence for the use of intravenous ketamine in chronic pain from available RCTs. Unfortunately, only seven RCTs that met the inclusion criteria were identified, with follow-up times ranging from 48 hours to 12 weeks. Ultimately, the authors concluded that there is low-quality evidence (i.e. limited confidence; the true effect may be significantly different from the study estimates) that intravenous ketamine infusion provides short-term pain relief in the treatment of chronic pain. They mention several times in the paper that higher doses appear more effective, yet strangely, the results of their own subgroup analysis suggest no difference in outcome in high- versus low-dose regimens (p=0.213). Compared with placebo, the ketamine group also had a significantly higher risk of nausea (relative risk 3.52 [95% CI 1.74, 7.14]) and psychotomimetic effects (5.92 [2.95, 11.89]). Surprisingly, the primary outcome measure that the authors chose to focus on was the single lowest recorded pain score any time ≥48 hours after cessation of treatment. This doesn't seem particularly valid in capturing the overall burden of the pain experience for the patient. Overall, the quality of the available evidence was poor, with all but one study considered to have a high risk of bias. Incredibly, none of the included studies clearly reported blinding their primary outcome assessor(s) to which treatment the patients received and long-term follow-up was all but nonexistent. In summary, more work is required here!

Reference: Anesth Analg 2019;129:241–54
Abstract

Chronic exposure to insufficient sleep alters processes of pain habituation and sensitization

Authors: Simpson NS et al.

Summary: Seventeen healthy adults participated in 3 weeks of in-laboratory restricted sleep with limited recovery versus control sleep conditions; 14 participants completed both 3-week protocols. Mild but statistically significant increases in spontaneous pain were seen with the sleep-restricted protocol when compared against the control protocol. Following the first week of sleep restriction, significant decreases in heat-pain threshold were recorded, but these normalised with further exposure to sleep restriction. In contrast, chronic exposure to restricted sleep led to significantly decreased habituation and increased temporal summation in response to cold pain, although only during the prior 2 weeks of exposure to this protocol. These alterations in pain-modulatory processes did not completely resolve after limited recovery sleep.

Comment (DR): The bidirectional relationship between sleep and pain is well established. Pain not only disturbs our sleep but disrupted sleep can reduce the function of central pain inhibitory pathways, alter immune system activity and increase clinical pain intensity. In fact, a 2013 review of the evidence concluded that sleep impairments more reliably predict pain than pain predicts sleep impairments. The novelty of this study is in their sleep restriction paradigm, which was designed to mimic sleep restriction during the work week (5 days of 4 hours per night), followed by 2 days of 'catch-up' sleep (8 hours per night) over the weekend. As in previous studies, sleep restriction led to increased spontaneous pain reports and increased sensitivity to experimentally induced pain across a range of tests, including measures of central nociceptive processing (temporal summation and habituation). Interestingly, changes in central nociceptive processing only occurred in weeks 2 and 3 of sleep restriction and failed to normalise after 2 days of 'catch-up' sleep, suggesting that repeated sleep restriction is required for these changes to manifest but, once present, an extended period of normal sleep may be required to fully recover nociceptive function. The findings of this paper should again remind us that sleep is an essential and multidimensional factor that deserves to be formally assessed in chronic pain patients and that, wherever possible, treatment of sleep disorders should form part of an overall patient management plan. Importantly, improving sleep is likely to reduce pain as well as enhance an individual's quality of life.

Reference: Pain 2018;159:33–40
Abstract

Quantitative sensory testing and predicting outcomes for musculoskeletal pain, disability, and negative affect

Authors: Georgopoulos V et al.

Summary: This was a systematic review and meta-analysis of 32 prospective cohort studies and five RCTs (overall n=3860) reporting on QST (quantitative sensory testing) for predicting pain, disability and negative affect; pain was an outcome in 30 studies, disability in 11 and negative affect in three. Baseline QST was found to be a significant predictor of musculoskeletal pain and disability. Temporal summation and conditioned pain modulation were significantly associated with follow-up pain, whereas baseline mechanical threshold modalities predicted follow-up disability.

Comment (DR): This study aimed to systematically review the evidence for the ability of QST measures to prospectively predict pain, disability and negative affect in people with a range of chronic musculoskeletal conditions, including knee osteoarthritis, several different types of surgery, whiplash-associated disorders, lower back pain, shoulder pain and fibromyalgia. Heightened pain sensitivity at baseline on a variety of QST measures, likely to at least partly reflect increased central sensitisation, was consistently associated with worse pain and disability at follow-up. While the results of this meta-analysis are in many ways unsurprising, it is important to note that, even taking into account the upper bound values of the confidence intervals, the strength of the associations between baseline QST measures and pain and disability at follow-up were consistently weak-to-moderate. This may partly reflect the different mechanisms that each QST measure is purported to measure. However, to me it highlights the multidimensional, biopsychosocial nature of pain and the involvement of a range of other important factors (e.g. illness and treatment beliefs, expectation, depression, anxiety, social support, etc) that may strongly contribute to a person's levels of pain and disability.

Reference: Pain 2019;160:1920–32
Abstract

Independent commentary by Dr David Rice

Dr David Rice is a Senior Lecturer in the School of Clinical Sciences and a Senior Research Officer in the Health and Rehabilitation Research Institute at AUT University. He also has a position as a Scientific Officer in the Department of Anaesthesiology and Perioperative Medicine at Waitemata DHB. David has a PhD in neurophysiology. His research focuses on the neuromuscular consequences and management of joint injury and arthritis, predictors of post-surgical pain, the mechanisms and management of chronic pain conditions and the effects of pain on motor performance.

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