

THE IN A HURRY SERIES

Pain

in a Hurry

What it is, and why it lies

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The Whole Thing in One Page

Pain feels like a diagnosis delivered from inside the body. Put your hand on a hot pan and the apparent conclusion is immediate: damage happened there, pain came from there, and its intensity reports how serious the damage is. That picture is dependable enough for daily life and too simple for almost every difficult case.

Tissue does not send pain to the brain. Specialised sensory neurons turn potentially damaging heat, pressure and chemicals into electrical activity. This detection and encoding of noxious events, which damage or threaten normal tissue, is called nociception. Pain is the unpleasant sensory and emotional experience of the person. The two often travel together, but neither guarantees the other. A badly injured person may feel little at first. A light brush can become agonising after nerve damage. An amputated hand can hurt years after the hand has gone. Capsaicin can make an intact mouth burn because it activates some of the same molecular machinery as noxious heat.

Pain usually has an adaptive role. It interrupts attention, reorganises movement, promotes care and teaches avoidance. It is therefore closer to an urgent demand than to a photograph. Yet an evolved capacity can be useful even when a particular episode is inaccurate, unnecessary or harmful now. Pain itself can become pathological, and continuing disease can remain active when altered processing has joined it.

The experience is assembled through several levels. Nociceptors respond selectively at the body's edges. Spinal circuits amplify, inhibit and route their activity. Descending pathways alter that processing according to behavioural state, expectation and context. Distributed brain systems contribute location, unpleasantness, bodily meaning and action. There is no single pain centre and no established clinical painometer that can replace a person's report.

This is why pain lies, with one strict qualification. The person is not lying, and the suffering is not an error. The mismatch lies in what the experience

appears to establish about its own cause. Pain may exaggerate present danger, locate a problem away from its source, persist after an initiating injury has healed or remain quiet during catastrophe. It can be conclusive evidence that someone hurts while remaining uncertain evidence about tissue state, mechanism and prognosis.

Different mechanisms can produce the same word. Nociceptive pain is driven chiefly by activation of nociceptors during potentially damaging events in non-neural tissue. Neuropathic pain requires a lesion or disease of the somatosensory system. Nociplastic pain describes altered nociception not adequately explained by either route. These mechanisms overlap. Peripheral and central sensitisation, inflammation, nerve injury, learning, sleep, mood, work and social conditions can change the system without becoming interchangeable explanations.

Assessment therefore follows pattern, function and mechanism rather than intensity alone. Treat a removable cause where one exists. Match medicines, procedures, rehabilitation and psychological treatment to defensible targets and harms. New, severe or changing pain still needs proportionate assessment. Chronic pain deserves the same credibility even when no scan can display it.

The permanent correction is this: pain is certain evidence of an experience and a powerful prompt to investigate. It is not tissue's verdict on what is damaged, where the cause sits or what must happen next.

That is the book.

Why You Should Care

In northern Pakistan, researchers heard about a boy who entertained crowds by pushing knives through his arms and walking on hot coals. He seemed immune to pain. Before they could examine him, he died after jumping from a house roof on his fourteenth birthday. Six people from three other families were later found to have inherited failures affecting the same sodium channel needed by many pain-sensing neurons. They accumulated injuries without the usual warning. Some had badly damaged their tongues and lips by biting them as young children. A life without pain had not made their tissues any less vulnerable.

Pain is one of the body's most forceful ways of changing behaviour. It can keep a hand away from heat after a withdrawal has begun, make an ankle unload while tissue repairs and teach a child that a blade deserves distance. It does so by moving the body to the front of attention. Hunger can be deferred. A phone can be ignored. Pain recruits movement, memory, emotion and help before a detached analysis would finish.

That authority creates the problem this book must solve. Pain operates before certainty is available. Peripheral receptors sample selected conditions, not the tissue's complete state. Spinal and brain systems combine those inputs with recent activity, context and competing demands. The resulting experience may arrive before damage, exceed what later tests reveal, point away from its source or continue after the initiating event has changed. During an emergency it may do the reverse and appear late.

Everyone eventually meets that mismatch. It appears in the headache with no visible lesion, the alarming knee scan in a person who feels fine, the shingles rash whose nerve pain outlives the skin and the missing limb that still burns. It also appears when a familiar chronic pain changes because of a new fracture, infection or disease. The lesson cannot be that tissue never matters. It is that pain and pathology must be related by evidence rather than assumed to be identical.

Medicine has no laboratory instrument that directly measures another

person's pain. Heart rate, facial expression, scans and neural recordings can provide clues, but none proves how much someone hurts. Report therefore remains central. That makes pain both intimate and socially exposed. People whose symptoms lack a neat image may be dismissed. Others receive procedures aimed at abnormalities that were never the main cause. Sex, race, age, language, disability and a clinician's expectations can affect whose account is believed and what care follows.

The stakes rise when pain persists. Chronic pain can narrow sleep, work, movement, relationships and identity while repeated tests fail to produce one complete explanation. Desperation makes single answers attractive. Inflammation, nerve injury and altered processing are real mechanisms, but none explains everyone. A phrase such as trapped emotion may sound equally complete without identifying a demonstrated mechanism at all. Chronic primary pain and pain secondary to disease can also coexist. The absence of one tidy answer does not make every proposed answer equally good.

Understanding pain will not remove the need for diagnosis, treatment or caution. It changes what you ask of them. Intensity stops being a damage score. A scan becomes one piece of a causal case. A placebo response no longer exposes fraud. Rehabilitation no longer implies that the symptom began in thought. Persistent pain can be recognised as a health condition without granting any one mechanism permanent ownership of it.

Pain is often the experience interpreted with the greatest speed and certainty. Learning where that certainty is earned, and where it outruns the evidence, gives you a better chance of taking suffering seriously without mistaking the sensation for its own diagnosis.

The Core Ideas

Pain Is an Experience, Not a Damage Reading

Warm water can feel pleasant on one shoulder and sting on the other if that shoulder is sunburnt. The temperature is the same; the skin and nervous system meeting it are not. Pain belongs to that encounter, not to a fixed quantity of hurt carried by the water. The International Association for the Study of Pain treats it as a sensory and emotional experience linked to actual or potential damage, or resembling such an experience. Injury is not a requirement, and unpleasantness is part of the pain rather than a judgement pasted on afterwards.

The neural encoding of noxious events is nociception. Nociception can occur without pain. A withdrawal response can begin through spinal circuits before a conscious experience is fully formed, and anaesthesia can separate nociceptive activity from awareness. Pain can also occur in a body part that no longer exists, as phantom-limb pain shows, while peripheral, spinal and brain mechanisms may all contribute. What travels along a peripheral nerve is patterned electrical activity. The hurt belongs to the person in whom the wider process occurs.

Produced by a nervous system does not mean invented in the everyday sense. A cough and a seizure are produced by nervous systems too. Pain is personal because no observer can occupy another person's experience directly. Someone can report pain without an explanatory scan, and someone unable to speak can still suffer. Behaviour, physiology and context can assist assessment, but they are indirect. They do not turn pain into an external quantity waiting to be read.

These dimensions can vary without marching in step. Imagine an injection you have chosen for a clear medical benefit. The needle still hurts, but knowing why it is happening and when it will end may make it easier to bear. The same intensity, unexplained and inescapable, can have a different hold on attention. This is an illustration, not a promise that understanding will relieve every pain. Unpleasantness belongs to the experience; fear of what

the experience means can add to it.

Pain usually serves an adaptive role, but usually is the important word. It can interrupt activity, promote care of a body part, recruit assistance and teach avoidance. Congenital inability to feel pain shows the value of those effects. People born with severe loss of pain sensation can accumulate burns, fractures, joint damage and injuries to the mouth. Touch and temperature perception may remain, yet the missing unpleasant experience weakens warning and learning. The body has not become tougher. A major behavioural safeguard has gone.

An adaptive role across evolution does not guarantee present benefit in every episode. Severe postoperative pain can obstruct breathing and movement. Neuropathic pain can be generated by a diseased sensory system. Chronic primary pain can constitute a health condition in its own right. Even pain that began with useful guarding may later impose disability without protecting enough to justify its cost. Calling pain adaptive is a population-level account of why the capacity exists, not a moral defence of untreated suffering.

The system also works with incomplete evidence. Nociceptors sample particular mechanical, thermal and chemical events. Central circuits combine that activity with bodily state, recent history and present demands. A bias towards caution can be advantageous when delay carries high cost, but the smoke-alarm analogy reaches only this far. Pain is a conscious embodied experience, not a detector with one threshold or one purpose.

This is the first meaning of the subtitle. Pain speaks with the authority of direct experience while offering no direct access to its own cause. The suffering is established. The apparent diagnosis within it, that this tissue is damaged to this degree and this action is unsafe, remains an inference to test.

Nociceptors Encode Noxious Events, Not Pain

A chilli can make your mouth burn without being hot enough to burn it. Capsaicin, the chemical responsible, activates TRPV1, a molecular channel also responsive to noxious heat and acidity. The burning is real. A thermal burn is not required. This is a useful introduction to nociceptors: specialised sensory receptors that respond to events capable of damaging or threatening normal tissue. They occur in skin, joints, muscle, viscera and many other structures. Calling them pain receptors is convenient and misleading. They provide input to pain, but they neither feel nor contain it.

The first operation is transduction: turning one kind of event into another. Mechanical force, temperature or a chemical changes ion channels in a nerve ending. These channels allow charged particles to cross its membrane, changing its electrical state. If the change reaches threshold, voltage-gated channels regenerate an impulse along the axon towards the spinal cord. Nothing resembling a tiny burn travels up the nerve. The tissue event has become a pattern of electrical activity, carried by a cell whose responsiveness can itself change.

Signals travel along fibres with different properties. Thin, myelinated A-delta fibres conduct faster and often contribute to the sharp, relatively well-localised first pain after sudden stimulation. Unmyelinated C fibres conduct more slowly and often contribute to later burning or aching. The division is a useful teaching pattern rather than a complete wiring rule. Fibre populations vary, qualities overlap and the final experience depends on processing far beyond conduction speed.

Nociceptors need not wait for cells to die. Their high thresholds place many of them near the boundary at which normal tissue is at risk. A hot surface can provoke withdrawal before a deep burn develops. Strong pressure can hurt before a finger or joint is structurally damaged. This anticipatory margin helps explain why pain intensity cannot be read as a tissue ledger. Discharge reflects the event, the receptor and its state. Injury also depends on duration, location, tissue vulnerability and what happens next.

The sample is selective. Skin often permits precise localisation of a splinter. Visceral input is commonly more diffuse. Some structures can change substantially before producing much pain, and nociceptors do not monitor every form of biological failure. Silence may accompany safety, slow change, sparse innervation, impaired sensory function or a process outside the variables being sampled. Lack of pain is therefore no certificate that tissue is well.

After injury, local chemistry changes the instrument. Damaged cells, immune cells and blood vessels release mediators that lower thresholds and increase responsiveness. Warm water on sunburnt skin now hurts. A sprained ankle becomes tender to pressure that was ordinary yesterday. This peripheral sensitisation can reduce use while tissue is vulnerable, though the same process also contributes to suffering and can become excessive.

The change shows why a stimulus has no fixed pain value. The same load through a healthy tendon, an inflamed tendon and a healing surgical wound is not biologically equivalent. Receptors alter their responsiveness, mediators interact and some previously quiet fibres become active. The variability begins before the signal reaches the spinal cord.

The same bathwater can therefore meet a different threshold tomorrow. More responsive nerve endings need less provocation to produce activity. Variation can begin at the periphery; it need not originate in expectation or attention. Nociceptors tell the wider system something about conditions at the periphery. They cannot, on their own, tell the person how much damage exists or what it means.

The Spinal Cord Is a Control Point

A finger lands on a drawing pin. Before the person can describe the pain, muscles are already pulling the hand away. Sensory fibres enter through the dorsal roots and connect with interneurons and projection neurons in the dorsal horn of the spinal cord. Some circuits recruit motor output quickly enough to begin withdrawal before a conscious judgement is available. The body can start moving while the experience is still forming.

A simple diagram makes this look like a relay: peripheral fibre in, spinal tract up, pain out. The dorsal horn does more. Excitatory and inhibitory neurons combine activity from nociceptors, touch fibres, local circuits and pathways descending from the brain. Receptors change their responsiveness. Repeated input can build rather than remain constant. Activity leaving a spinal segment is therefore a transformed selection, not a copy of what entered it.

Melzack and Wall's gate-control theory made this regulation impossible to ignore. Their 1965 proposal was not a modern circuit map, but it broke the picture of a dedicated pain wire. Rubbing a bumped elbow can reduce the hurt for a time because touch-related input recruits inhibitory processes within the spinal cord. The rubbing does not reverse the injury. It changes the balance of activity being passed onwards.

The gate metaphor can become too neat. No single little door opens and shuts. Different spinal laminae, cell types, receptors and pathways contribute, and their behaviour changes with inflammation, nerve injury and repeated stimulation. The enduring point is narrower: nociceptive transmission is regulated at its first central junction.

The spinal cord also helps explain why pain can be felt away from its source. Visceral and somatic inputs may converge on overlapping neurons. The nervous system has a more detailed history of mapping skin and muscle than internal organs, so activity from an organ may be experienced at a familiar body surface. Cardiac ischaemia can be felt in the chest, arm, jaw or elsewhere. The clinical pattern matters, but the heart is not sending

separate pain messages to every site that hurts.

Timing matters here. A single mild input may fade, while repeated C-fibre activity can make dorsal-horn neurons respond more strongly to later pulses. This short-term increase, often called wind-up in experimental work, shows that identical events need not produce identical central output. The system retains a recent history even across seconds.

Control also travels downwards. Brainstem pathways can inhibit or facilitate spinal nociceptive processing through several chemical systems, including endogenous opioids. Attention, expectation, stress, goals and behavioural state can alter that control. During escape, inhibition may permit movement despite injury. Under persistent threat or after neural change, facilitation may contribute to continuing sensitivity. Neither direction is fully voluntary or fixed.

This two-way traffic dissolves the false divide between body and mind. A thought does not float down and cancel a real signal. Neural systems involved in attention, fear and learning change the excitability of other neural systems. Tissue input, prior experience and present demands meet within one biology.

Pain Has No Single Centre

An amputated hand can still feel clenched. There may be burning in fingers that are no longer there, or cramp in a limb that cannot be stretched.

Phantom-limb pain makes the puzzle unavoidable: how can an experience have such a definite address when the body part at that address has gone?

The nervous system retains representations of the body. After amputation, activity from the residual limb, spinal changes, brain representations and prior experience may all contribute to pain. The case is often recruited to prove one favourite theory, especially cortical remapping. Several mechanisms are implicated; no single explanation fits every phantom. The missing hand does not make the remaining nerves irrelevant.

What phantom pain establishes is narrower and more useful. The

experienced location of pain is an output, not a pin dropped by tissue on a map. Incoming signals, body representation and vision usually agree well enough for a splinter to feel as though it is exactly where it is. Remove a limb and its representation does not vanish on command. A precise bodily experience can survive a radical change in the body.

There is no single destination in the brain at which all this becomes pain. Nociceptive activity reaches the thalamus and engages distributed systems. Somatosensory regions contribute to location and physical qualities. The insula helps represent bodily state, while parts of the anterior cingulate cortex contribute to unpleasantness, attention and action. Other cortical and brainstem systems bring memory, arousal and control into the result. These are overlapping contributions, not separate offices with exclusive responsibilities.

Nor is a collection of coloured patches on a scan automatically a picture of pain. Many regions once grouped as a pain matrix also respond to salient sights, sounds and touches that demand attention without hurting. Seeing activity in a region involved in pain does not establish that pain caused the activity. A fire station can be busy without your house being on fire.

More sophisticated methods use patterns rather than single regions. A study published in 2026 followed two people with chronic pain through repeated scanning over more than half a year. Personalised models tracked fluctuations in their pain, but neither model transferred successfully to the other person. That is progress towards measurement, not a general clinical test that can overrule a patient's account.

Distributed organisation also explains why pain has separable dimensions. A sensation can be intense yet feel less threatening when its cause is understood and controlled. Another can be modest in physical intensity but intolerable because it is unpredictable, inescapable or associated with a feared disease. Brain injury and experimental manipulation can affect location, unpleasantness, attention or bodily response differently. The experience feels unified even though its components need not change together.

The reverse mismatch occurs too. A serious injury may initially produce little pain when attention and survival are directed elsewhere. Later, as the situation changes, the hurt appears. This does not require a heroic person overriding a switch. Competition among bodily priorities changes awareness and action.

None of this turns pain into a free-floating story. Particular lesions create characteristic patterns. Nerve roots, peripheral nerves and organs produce clues worth following. But those clues enter a system that also remembers a body, attends to a threat and prepares a response. The unity of the result conceals the different operations producing it.

A phantom hand is the extreme case, not a different species of experience. Ordinary pain also arrives already located and already important. Its certainty about where it hurts can make its implied explanation seem equally certain. The two do not have the same authority.

Context Changes the Biology

The same stimulus can hurt differently without the person choosing the difference. A vaccination needle watched closely may feel sharper than one received during absorbing conversation. An expected movement can provoke pain before its mechanical load becomes high. A controllable stimulus is often tolerated better than an identical one imposed without warning. Attention, predictability, meaning and control alter how the nervous system handles evidence.

These effects are sometimes described as psychological, as though they occur outside the body. They are implemented through it. Attention changes sensory competition. Expectation recruits cortical and brainstem systems that influence descending modulation. Fear changes muscle activity, autonomic state and learning. Poor sleep can increase pain sensitivity and reduce coping. Social safety or threat changes what an ambiguous sensation predicts. The categories are useful for organising causes, not for dividing real from unreal.

Placebo analgesia is the most abused example. A placebo is not proof that

nothing was wrong. Treatment rituals, words, prior learning and expectations can alter symptoms through measurable physiological processes. Endogenous opioid systems contribute to some forms of placebo analgesia, though not every placebo effect uses the same mechanism. Conditioning can matter even when conscious belief is incomplete. Such effects reflect context, not exposure of a liar.

An experiment with the short-acting opioid remifentanyl made the interaction unusually clear. Healthy volunteers received painful heat while the infusion continued. When they expected strong relief, the drug's analgesic benefit roughly doubled compared with the neutral condition. When they were told the infusion had stopped and pain might worsen, the observed benefit disappeared. This was a controlled experimental setting, not a universal law or a treatment plan. It shows that an active drug and expectation can combine inside one nervous system rather than occupying rival explanations.

Nocebo effects run in the other direction. Warnings, alarming interpretations and previous bad experiences can increase symptoms or side effects. Honest consent still requires disclosure of material risks. The problem is treating communication as inert. A clinician who lists every frightening possibility without proportion or says a spine is crumbling may change the threat attached to ordinary sensation. Reassurance can also fail when it dismisses experience instead of reducing uncertainty.

Meaning can change within minutes. The ache during a final kilometre may be accepted as evidence of effort when the runner chose the pace and knows the finish is near. A similar sensation during an unexplained night-time episode can be frightening. Context has not altered the past. It has changed the future the sensation seems to predict.

Learning gives context duration. If bending repeatedly predicts a severe flare, the movement can acquire threat before tissue load reaches the old level. Avoidance then prevents the person from discovering that some forms or amounts of bending may now be safe. This cycle is common enough to matter and variable enough not to diagnose anyone by itself. Ongoing

disease, nerve damage and mechanical sensitivity may still be present.

Context also includes whose body is being judged. A person's language, sex, ethnicity, age, communication ability and relationship with the clinician can shape whether a report is believed. Bias can lead to undertreatment, misattribution or reliance on tests that appear objective. The absence of a painometer leaves room for judgement, and judgement carries social history.

The practical consequence is easy to underestimate. The explanation accompanying treatment is itself part of the encounter, not packaging to be discarded once the medicine arrives. It can make uncertainty more manageable or give it a frightening shape. Neither effect removes the need to treat disease. It means that good care must attend to what the patient has been led to expect as well as what has been prescribed.

One Word Covers Different Mechanisms

A broken wrist, diabetic nerve damage and widespread pain without one adequate lesion can all hurt. Treating them as the same biological event because all three hurt is like treating every fever as one disease. Pain is the common experience. The mechanisms beneath it differ, overlap and change over time.

Nociceptive pain arises from actual or threatened damage to non-neural tissue and activation of nociceptors. A burn, arthritic joint, kidney stone or surgical wound can fit, though each has its own biology. Inflammation may sensitise the area, and movement or pressure may increase input. The category does not make pain a precise measure of damage. It identifies normally functioning nociceptive pathways as an important route into the experience.

Neuropathic pain is caused by a lesion or disease of the somatosensory nervous system. A traumatic nerve injury, diabetic polyneuropathy, post-herpetic neuralgia and some pain after stroke are examples. Burning, electric shocks, numbness and pain in a plausible nerve distribution can support the suspicion, but descriptive words alone are insufficient. The

diagnosis requires a relevant lesion or disease, a neuroanatomically credible pattern and the best available supporting evidence.

Nociplastic pain describes pain arising from altered nociception despite no clear evidence that actual or threatened non-neural tissue damage, or a somatosensory lesion or disease, adequately explains it. The term fills a real gap between established nociceptive and neuropathic accounts. It is not a synonym for imaginary, psychological, medically unexplained or centrally generated pain. It also names a descriptor, not one settled lesion or pathway.

Clinical criteria have been proposed for chronic nociplastic pain affecting musculoskeletal structures. They use features such as regional or widespread pain and evoked hypersensitivity, with further features increasing confidence. These criteria are useful within their intended setting and do not create a universal test for every organ system or condition. A person may also have nociceptive, neuropathic and nociplastic contributions at the same time.

These names sit at different levels of explanation. Nociplastic describes a pain presentation and its proposed mechanistic character. Central sensitisation describes increased responsiveness in central nociceptive neurons. Chronic primary pain is a clinical category, not a synonym for either. One label does not prove the others. Confusing them turns a useful distinction into a circular diagnosis: the person hurts because the system is sensitised, and the system must be sensitised because the person hurts.

The three mechanistic labels answer a different question from acute and chronic. Acute and chronic concern duration and clinical organisation. Chronic pain is commonly defined as lasting or recurring for more than three months. The threshold aids recognition, classification and access to care. It is not a biological midnight at which an injury turns into a different substance.

International classification also distinguishes chronic primary from chronic secondary pain. In chronic primary pain, pain and related distress or

disability constitute a health condition in their own right when another diagnosis does not adequately account for the presentation. Chronic secondary pain remains associated with an underlying condition such as cancer, surgery, nerve disease, secondary headache, visceral disease or musculoskeletal disease. The categories can coexist. They recognise different clinical relationships without settling every causal question.

Mechanisms cross all these borders. Osteoarthritis may involve nociceptive input, inflammation, sensitisation, sleep disruption and fear of movement. A nerve injury can generate ectopic firing and central change. Cancer pain may reflect tumour pressure, inflammation, treatment injury and neuropathy. Persistent low-back pain may contain changing contributions from tissue, nerves, altered processing, movement and wider health. One word on a chart cannot carry that mixture.

Classification is therefore a working model at a stated level of confidence. It should connect observed pattern to a rational next step and remain revisable. An anti-inflammatory drug, nerve-targeted medicine, local procedure, rehabilitation and psychological therapy act through different proposed routes and carry different risks. The useful question is not which label wins. It is which processes are sufficiently supported to target now, what remains uncertain and what evidence would require the model to change.

Pain Can Outlast Its First Cause

Pain systems change with use and injury. That plasticity helps a sunburn become tender, an injured ankle receive less load and a child learn from a hot pan. It also means the relationship between an initiating event and later pain can change. The first cause may resolve, persist, expose a nerve lesion or leave altered processing behind.

Healing and pain do not share one clock. Skin, muscle, bone, tendon, nerve and confidence recover on different timetables, while disease can fluctuate or progress. Sensitivity may remain after basic tissue repair. In other cases continuing pain is evidence that the original diagnosis, the estimated

recovery or the assumption of healing was wrong. Persistence is a reason to update the explanation, not proof that one particular explanation has won.

Peripheral sensitisation begins near affected tissue. Inflammatory mediators can lower nociceptor thresholds and increase responsiveness. Central sensitisation refers to increased responsiveness of nociceptive neurons in the central nervous system to normal or subthreshold input. Repeated or intense activity can strengthen excitation, reduce inhibition and broaden responses. A normally painful pinprick may hurt more, which is hyperalgesia. A light brush may hurt despite usually being non-painful, which is allodynia. Repeated stimuli may feel progressively stronger.

These mechanisms do not form a universal stamp for chronic pain. Central sensitisation can be demonstrated when neural input and output are measured under controlled conditions. In ordinary clinical work it is inferred indirectly, and no questionnaire, tender point, scan or single sensory test proves that it explains one person's whole condition. Persistent inflammation, structural disease, neural lesions, endocrine or immune disorders and other causes remain possible. Mechanistic language should narrow uncertainty rather than close investigation.

Nerve injury supplies another route. Damaged sensory neurons can fire abnormally, alter channel expression and change connections in the spinal cord. Normal touch input may gain access to circuits that now contribute to pain. Loss of sensation can coexist with burning or electric shocks. The combination makes little sense under a simple damage meter and good sense once the measuring system can itself be diseased.

Learning and behaviour can join the sequence without becoming its sole cause. Guarding and avoidance are often sensible early responses. If every movement continues to predict reinjury, activity may shrink beyond what tissue state requires. Strength, tolerance, sleep, work and confidence can decline, while the next attempt carries more uncertainty and produces more pain. This process may maintain disability and sometimes pain. It is conditional. Avoidance can still protect unstable tissue, and forcing activity through progressive symptoms can cause harm.

Persistent pain also changes the person's environment. Interrupted sleep can increase sensitivity and reduce control. Lost work creates financial pressure. Repeated disbelief increases vigilance and reduces trust. Medication effects, low mood, isolation and diagnostic uncertainty narrow choices. Biopsychosocial is useful only when it identifies such interactions. Used as a euphemism for normal tests, it explains nothing.

The same properties that make pain forceful at the beginning can complicate its later course. Urgent, memorable experience can change behaviour before certainty exists. The same capacity for priority, learning and plasticity means later pain need not remain a proportional readout of the initiating tissue event. Yet it does not follow that the system is always protecting, overprotecting or making a false alarm. Chronic pain may itself be pathological, continuing disease may still generate input, and both can coexist.

Persistence is not the same as permanence. Nociceptive drivers can be treated. Neural excitability and inhibition can change. Movement can become safer through tolerable exposure when the diagnosis permits it. Sleep, work and social conditions can improve. Progress may first appear as wider activity, shorter flares or reduced interference before intensity falls. Some conditions remain severe despite excellent care, and promises to retrain the brain can turn scientific possibility into blame.

The mature question is therefore neither whether the pain is real nor whether the tissue healed. It is what processes now sustain the experience, which of them are established, which remain plausible and which can be altered without ignoring danger. Pain began as a force that reorganised action. When it persists, treatment must decide whether that force is still tracking a cause, has become part of the disease or is doing both at once.

How It Actually Works

Contact

Take an illustrative event: a bare foot lands on a sharp fragment hidden in grass. The point deforms the skin hard enough to activate mechanically sensitive nerve endings and may begin to tear cells. Positive ions enter through opened channels, the membrane voltage changes and, if threshold is reached, voltage-gated sodium channels generate an action potential. The impulse is renewed along the axon rather than draining away like current through a loose wire.

The cell body sits in a dorsal-root ganglion beside the spinal cord. One branch reaches the foot and another enters the dorsal horn. Nothing in this sequence is yet the person's pain. It is transduction and conduction: a noxious event has been converted into neural activity and carried towards circuits that can organise withdrawal, attention and later pain.

Threshold is not a fixed line engraved in the ending. Recent activity, tissue chemistry and inherited channel variants change how much stimulation is needed. Local anaesthetic works upstream of conscious pain by blocking sodium channels needed for action-potential propagation. The person has not been persuaded that the foot is safe. A required link in the evidence chain has been interrupted.

The signal is selective rather than complete. Pressure, temperature and damaged-cell chemistry recruit overlapping populations of nociceptors. The exact pattern depends on force, location, prior inflammation, genetics and the recent state of the ending. Touch and position fibres send their own information. The central nervous system receives several partial accounts of what happened, not a microscopic image of the skin.

Withdrawal

Fast A-delta activity reaches spinal circuits and often contributes to a quick, sharp first sensation. Interneurons excite muscles that lift the foot and inhibit muscles that would oppose the movement. Weight shifts to the other leg. In a more forceful event, crossed circuits help the opposite side support the body. The withdrawal can begin before the person has located and described the hurt.

Reflex does not mean mindless perfection. Pulling away from a hot surface is usually useful. Pulling a hand while holding a surgical instrument could be dangerous, and spinal output is shaped by posture, task and signals descending from above. Withdrawal circuits are fast because they use local organisation, but they remain part of a larger system.

Within the dorsal horn, the incoming activity meets inhibitory and excitatory networks. Touch fibres from the same area, signals from neighbouring tissues and recent nociceptor activity affect projection neurons. Repetition can make later pulses produce a larger response. Inhibition can damp them. The output of the spinal cord therefore depends on the incoming nociceptive activity, competing sensory input, local inhibition, recent history and descending control.

Primary afferents release glutamate and, during stronger or sustained activity, several neuropeptides. Different receptors respond over different timescales. Local inhibitory neurons use transmitters such as GABA and glycine to restrain excitation. The chemistry matters because pain is shaped by the balance among routes, not by one substance whose concentration can be read as the answer.

The hurt appears

Projection neurons send activity through ascending pathways towards the brainstem, thalamus and several cortical regions. The route branches. Some systems help locate the stimulus and distinguish pricking from burning. Others recruit arousal, autonomic change, unpleasantness, attention and action. The person looks down, stops walking and inspects the foot. A coherent pain appears from coordinated operations that have no single command centre.

Location feels delivered by the foot because the system has a reliable map built from development and experience. Most of the time, activity entering through particular nerves corresponds to events in particular tissues, so the inference is useful. The map can fail after amputation, nerve injury or convergence from internal organs. The experience remains somewhere in the body even when the original source is elsewhere or absent.

The event acquires meaning rapidly. Was it clean or contaminated? Is the person alone? Can weight be placed on the foot? Did the same spot hurt before? A sharp sensation during a safe laboratory task, an unexplained puncture in a field and a needle used during treatment do not predict the same future. Memory and context alter the pain and action response before deliberate reasoning has finished.

Several outputs arrive together. Attention narrows. Heart rate and sweating may change. The face and voice communicate distress. Movement is reorganised, and memory stores the event with its location and consequences. These are not optional decorations around a sensory core. They are part of why pain changes behaviour more forcefully than neutral information about tissue could.

The second wave

Slower C-fibre activity often contributes to a spreading burn or ache after the first sharp pain. If cells have been injured, potassium, protons, prostaglandins, cytokines and other mediators change the local chemical environment. Blood vessels and immune cells join the response.

Nociceptors become easier to activate. Pressure from standing, which was harmless moments earlier, now hurts.

This tenderness often has adaptive value because it can reduce loading while the wound closes and contamination is controlled. The sensitive zone may extend beyond the exact point of damage. Yet neither its size nor its intensity maps neatly onto the number of injured cells. Inflammation changes neural responsiveness as well as tissue state, and excessive tenderness can obstruct recovery rather than improve it.

Sensitivity at the wound is often called primary hyperalgesia. Increased sensitivity in nearby uninjured tissue can reflect altered central processing as well as peripheral change. The border between them is not visible to the person. Both can feel as though the surrounding body has become damaged, showing how a widened sensitive area can be experienced as a map of pathology.

A clinician may call this nociceptive pain because potentially damaging events in non-neural tissue and activation of nociceptors are the main drivers. That label remains a hypothesis about mechanism, not a guarantee that every sensation originates at the wound or that greater pain proves greater injury.

Control returns from above

While signals ascend, control descends through pathways from the cortex, hypothalamus and brainstem. These pathways can inhibit or facilitate dorsal-horn processing. Endogenous opioid, noradrenergic, serotonergic and other systems contribute. Their effects depend on receptor, circuit, timing and state, which is why the loose phrase natural painkillers explains less than it seems.

Attention to an urgent escape route may suppress pain long enough to move. Fear and uncertainty can facilitate it. A person given control over the timing of an experimental stimulus may tolerate it better than someone receiving the same stimulus unpredictably. Deliberate strategies such as distraction or reappraisal can sometimes alter pain, but descending control is neither a switch nor proof of superior character.

Treatment enters these circuits too. A local anaesthetic can block action-potential conduction in peripheral nerves. Anti-inflammatory medicines can reduce production of some sensitising mediators. Opioids can act at peripheral, spinal and brain sites. Expectation can strengthen or weaken observed drug effects. Each intervention changes part of the system, which is why an identical tablet can have different consequences across people and occasions.

One painful stimulus can also inhibit another through descending control, a laboratory phenomenon often studied as conditioned pain modulation. The effect varies across people and conditions and cannot diagnose one mechanism at the bedside. It nevertheless reveals an important principle: pain processing reflects the state of a regulatory network, not the arithmetic sum of peripheral impulses.

The next day

The fragment has been removed and the wound cleaned. Tissue repair proceeds through overlapping inflammatory, proliferative and remodelling processes. Pain may fall quickly, fluctuate or remain while the skin is still sensitive. The person protects the foot, then tests it through ordinary movement. Successful, tolerable use provides evidence that greater loading may be possible without equivalent harm. A renewed sharp increase may indicate irritation, excessive demand or a complication worth reassessing.

Healing does not require pain to track every stage. Some wounds hurt little. Others remain tender after basic closure because nociceptors and central circuits take longer to settle. Sleep, prior injury, infection, medication, metabolic health and the location of the wound alter the course. A calendar

estimate is useful only as a range tied to the tissue and event.

What happens next can alter later responses. If each step produces severe pain, guarding and avoidance are reinforced. If carefully increased use remains tolerable and the feared injury does not occur, confidence and capacity may grow. The change is not guaranteed. A damaged nerve, unstable tissue or repeated flare may keep supplying evidence that caution remains necessary.

Too little pain control can obstruct recovery as well. Severe postoperative pain may discourage deep breathing, coughing, sleep and early movement. Total numbness can remove useful warning and permit overload. Clinical care therefore aims for adequate relief while preserving safety and function, not maximal sensation or maximal suppression as abstract goals. The correct balance changes with the procedure and person.

When pain persists

Now change the example. Months after an ankle sprain thought to have healed, walking remains painful. Several broad routes could produce that outcome.

The first is continuing nociceptive input. The original diagnosis may have missed a lesion, inflammation may persist or a related structure may now be overloaded. The second is neuropathic pain. A lesion or disease affecting the somatosensory system can create ectopic firing, loss of normal sensation, altered sodium-channel expression and changed spinal processing. Burning, shocks, numbness and pain from light touch may appear together.

The third route involves altered nociception that is not adequately explained by continuing tissue injury or a confirmed somatosensory lesion. Sensitivity may have widened, inhibition may be reduced and central circuits may respond more strongly to ordinary input. When the clinical pattern supports it, nociplastic pain can be a useful descriptor. It does not identify one lesion, one cause or one treatment.

These routes frequently mix. Osteoarthritis can supply nociceptive input while the nervous system becomes more responsive. A nerve lesion can provoke central sensitisation, guarding and avoidance. Persistent pain can disturb sleep, mood, work and movement, which then alter sensitivity and opportunity for recovery. Classification is most useful when it preserves overlap rather than forcing one pure explanation.

The observable pattern may include hyperalgesia, allodynia or a spreading painful area. Repeated input may feel progressively stronger. Those findings can support a sensitisation model, but they are not exclusive to one diagnosis and depend on testing method, attention and comparison site. A mechanism gains credibility from convergence, not from one dramatic bedside sign.

Assessment without a painometer

Pain assessment begins with the report. Where is it? What does it feel like? When did it begin? What changes it? What can the person no longer do? What happened before it started, and what has happened since? Intensity matters, but so do duration, distribution, interference, sleep, function, sensory loss, weakness, autonomic changes and the meaning attached to the symptom.

Examination tests competing explanations. Swelling, temperature, movement, strength, reflexes, sensation and nerve distribution can reveal patterns. Blood tests, imaging or nerve studies may identify inflammation, fracture, tumour, infection, nerve damage or another cause. They can also find abnormalities that are common in people without pain. A scan becomes useful when its finding fits the history and examination and changes a decision.

The task is Bayesian even when nobody uses the word. A common age-related disc change has limited explanatory power by itself. The same finding alongside a matching neurological deficit, distribution and time course carries more weight. A normal test can reduce the probability of some conditions without proving that no pain mechanism exists.

Credibility should not be made contingent on a visible lesion. Self-report remains the best direct evidence of the experience because every external measure captures a correlate, response or possible cause. That principle does not forbid careful diagnosis. It separates two questions: does this person hurt, and what processes explain it?

Numerical scales help track one person's change but create false precision when treated as universal units. A fall from eight to six may matter greatly to one person and little to another. The same rating can accompany walking to work or being unable to leave bed. Good assessment records intensity beside interference, function, distress, sleep, adverse effects and goals.

Matching treatment to the problem

Treatment starts with causes that can and should be changed. A fracture may need stabilisation. Infection needs specific care. Compression producing progressive neurological loss may require urgent intervention. Inflammatory disease, cancer and vascular problems have their own pathways. Pain education cannot substitute for identifying a dangerous or treatable process.

After that, mechanism and context guide the mix. Nociceptive inflammatory pain may respond to time, protection, appropriate movement, local treatment, anti-inflammatory measures or analgesia. Neuropathic pain may require medicines and procedures that differ from those used for a sprain. Chronic primary pain may call for supported exercise, cognitive or behavioural treatment, selected medicines and attention to sleep, work and social barriers. Guidelines differ by condition, age, pregnancy, comorbidity and country.

Medicines do different jobs. An anti-inflammatory drug aims partly at the chemistry sensitising tissue; a local anaesthetic interrupts nerve signalling. Some medicines known as antidepressants can help selected pain conditions even without depression. The name records one use of the drug, not a verdict on the origin of the patient's pain. Evidence and harms still depend on the condition: a medicine helpful after surgery may be poor

long-term policy for chronic primary pain. Intensity alone does not choose the drug.

Opioids can provide important relief in selected acute, cancer and palliative settings. Long-term use can bring tolerance, physical dependence, sedation, constipation and overdose risk, with limited benefit for many chronic non-cancer presentations. Physical dependence means adaptation that can produce withdrawal when the drug is reduced or stopped; it is not the same as addiction, which involves impaired control and continued use despite harm. Both risks require attention. Stopping prescribed opioids abruptly can be harmful; changes need a plan with the prescriber.

Procedures face the same test. An injection, nerve block, ablation or operation is rational when a plausible target, expected benefit and risk profile align. A visible abnormality is not enough. The intervention should answer a question about mechanism or change a function that matters. Repeated procedures without durable benefit are evidence too.

Rehabilitation works through several routes. Movement maintains capacity, confidence and participation. Graded exposure can reduce fear and expand capacity when a chosen activity has become safer than expected. Pacing can help some people avoid repeated overextension followed by prolonged withdrawal. Cognitive behavioural therapy and acceptance and commitment therapy can reduce distress, avoidance and interference without claiming that thoughts caused the original pain. Social and occupational changes may remove loads no nervous system can reframe away.

The target is wider than zero. Pain reduction matters, and some causes can be removed. In persistent conditions, progress may also mean sleeping longer, walking farther, working more reliably, using fewer risky medicines or recovering from flares faster. Function should not be used to deny symptoms, and a person should not be forced through escalating pain to prove motivation. It offers another axis on which improvement can appear.

Every treatment is also a test of the model. A nerve block that briefly abolishes a matching pain supports one source without proving that every

future episode shares it. Failure of one drug may reflect wrong mechanism, inadequate exposure, intolerance or an unrealistic target. Plans should state what improvement would count, when harms will be reviewed and what result would trigger a different explanation.

When the pattern changes

A sophisticated model can create a new danger: explaining away a changed symptom. Some patterns require emergency care, not reassurance about a sensitive nervous system. Sudden chest discomfort that does not go away, especially with pain spreading to an arm or jaw, sweating or breathlessness, warrants an immediate emergency call. Back pain with new loss of feeling around the genitals or anus, difficulty passing urine or loss of bladder or bowel control also needs emergency assessment. In the UK, call 999 for chest-pain emergencies and seek A&E or 999 help for these back-pain signs. Elsewhere, use the local emergency service.

Severe rapidly worsening pain, progressive weakness, major trauma or fever with marked illness may also change urgency. The relevant signs depend on the body region, history and person. These examples are not a complete checklist, and absence of a listed feature does not establish safety.

Persistent familiar pain can also change for a new reason. A person with chronic back pain can still develop a kidney stone, infection or fracture. Previous nociplastic or neuropathic mechanisms do not immunise anyone against fresh disease. The correct lesson is not to distrust pain. It is to interpret the pattern with proportion, history and readiness to revise.

That revision can move in either direction. An alarming sensation may prove benign after assessment, allowing activity to resume with greater confidence. A familiar ache may acquire a new neurological or systemic feature that changes urgency. Safety lies in noticing the altered pattern, not in deciding once that the nervous system is overprotective or that every flare marks new damage.

How we know

Pain science combines evidence that answers different questions.

Recordings from single sensory fibres show how mechanical, thermal and chemical events are transduced. Animal studies permit invasive work on cells and circuits but cannot reproduce a person's full report. Human psychophysics relates controlled stimuli to sensation, while nerve blocks, lesions and medicines test causal pathways. Clinical patterns reveal what disease and injury do outside the laboratory.

Brain imaging maps distributed activity; personalised models can also track pain within some individuals. That is not yet a general clinical substitute for report. Placebo and nocebo experiments show that expectation changes processing, though tightly controlled heat studies do not represent every chronic condition. Longitudinal studies of persistent pain are vulnerable to mixed diagnoses, changing treatment and uncertain direction of causation.

Classification has improved by separating nociceptive, neuropathic and nociplastic mechanisms and chronic primary from secondary pain. The boundaries remain working tools rather than natural boxes. No method answers every level of the problem, so agreement across methods carries more weight than one vivid result. Across all methods, self-report stays central. The evidence can explain how pain is generated and altered. It cannot replace the person experiencing it.

What People Get Wrong

"Pain is a signal sent by damaged tissue"

The wire model is built into ordinary language. We speak of pain receptors, pain fibres and pain signals travelling to the brain. A cut activates peripheral neurons, activity reaches the spinal cord and pain follows, so the shorthand seems earned.

What travels is nociceptive activity. It contains information about intense mechanical, thermal and chemical conditions, then encounters spinal and

brain systems that regulate, locate and value it. Damage can occur with little immediate pain. Nociception can continue during unconsciousness without a reported experience. Pain can arise in a missing limb or from normally harmless touch after nerve injury.

This does not make tissue irrelevant. A fracture, burn or inflamed organ can provide decisive input. The correction concerns identity: the input is one cause of pain, not pain itself. Calling nociceptors pain receptors hides every point at which the system can amplify, inhibit, mislocate or reshape the input.

The practical cost is large. A clinician may keep searching for tissue damage because pain persists, or dismiss the person once the search fails. The better question is which peripheral, neural and contextual processes are producing the experience now.

"More pain means more damage"

Pain often rises as a controlled harmful stimulus intensifies, which is why the rule feels dependable. Press harder on a bruise and it hurts more. Increase heat and withdrawal becomes urgent. In a new injury, sudden severe pain can be an important warning.

The relationship is neither fixed nor universal. Inflammation lowers nociceptor thresholds. Nerve injury can make a light brush excruciating. Attention and uncertainty can increase pain, while emergency conditions can suppress it. Some serious diseases remain quiet until late. Tissue type, location, duration, prior injury, sleep and the nervous system's state all alter the response.

A pain score therefore measures reported intensity, not millimetres of damage. It can be useful for following one person under comparable conditions. It becomes misleading when used to compare injuries, decide that severe pain must reveal severe pathology or infer that improving pain proves complete healing.

The correction cuts both ways. Low pain does not certify safety. High pain

does not justify disbelief when tests look modest. Intensity is evidence about the experience and a reason to investigate in context, not a ruler laid across tissue.

"A scan can prove what hurts"

Images have an advantage in an argument: everyone in the room can look at them. A disc bulge, torn tendon or arthritic joint appears on a screen, while the patient's pain reaches the clinician chiefly through an account. The picture can seem to settle both credibility and cause. Those are different questions.

Imaging can identify fractures, tumours, infection, inflammation, nerve compression and other conditions that matter greatly. It can also reveal age-related and degenerative changes in people who have no pain. Systematic reviews of symptom-free populations find many spinal abnormalities become more common with age. Conversely, a normal scan does not show that nociception, nerve dysfunction or sensitisation is absent.

A finding earns causal weight when it fits the location, time course, examination and known biology. That fit supports an explanation; it does not prove it. A scan showing a disc bulge is one observation. Establishing that this bulge explains this person's symptoms requires the rest of the case. Functional brain imaging adds another distinction: predicting pain under a research protocol is not independently certifying a patient's suffering in ordinary care.

The myth persists because medicine prefers visible objects and patients deserve explanations. A named abnormality can feel kinder than uncertainty. It becomes harmful when incidental findings create fear, treatment chases the picture or an unremarkable image is used to deny suffering.

"Chronic pain is an injury that never healed"

Sometimes it is. Ongoing inflammation, tumour, joint disease, repeated tissue stress or another continuing process can keep activating nociceptors. Chronic secondary pain exists precisely because an underlying condition remains clinically important.

The model fails when it becomes the only possibility. Nerves can be injured and fire abnormally. Spinal and brain circuits can become more responsive. Pain can persist as a condition in its own right when no continuing lesion adequately explains its severity or impact. Chronic primary and chronic secondary pain can also coexist, so finding one diagnosis does not reveal every maintaining process.

Three months is a classification threshold, not the usual healing time for every tissue and not the moment pain becomes psychological. Bone, tendon, skin, nerve and disease follow different clocks. A persistent symptom may justify renewed investigation, a revised mechanism or both.

The unhealed-injury story encourages endless structural searches and permanent guarding. The opposite story, that all chronic pain is a mere false alarm, can miss disease. The useful account asks what remains active: nociceptive input, neural lesion, altered nociception, learned responses, sleep disruption, social strain or a mixture.

"Pain is either physical or psychological"

The binary offers a clean sorting rule. Find a lesion and the pain is physical. Fail to find one and emotion, stress or personality must be responsible. Patients learn that psychological explanation means fabricated, while clinicians use physical to mean respectable.

Every pain is physical in the plain sense that it depends on a living nervous system. Psychological processes such as attention, expectation, fear and learning are also biological processes with neural, endocrine, autonomic and behavioural effects. Social conditions alter exposure, safety, sleep, work and access to care. None occupies a separate substance outside the

body.

A broken ankle can hurt more when a person is frightened and alone. Neuropathic pain can be worsened by poor sleep. Persistent pain can produce depression, and depression can alter pain. Direction runs both ways. Recognising these links neither removes the fracture nor blames the person.

The binary damages care in two directions. It can send people through procedures because a psychological contribution feels insulting, or deny medical assessment because a mental-health history is visible. A complete explanation asks how biological, psychological and social processes interact without using one category to disqualify the others.

"Placebo relief means the pain was imaginary"

A placebo response is often treated as a lie detector. If expectation or a treatment ritual reduces pain, the original symptom must have lacked a physical cause. But an effect on pain does not identify what started it. Relief answers one question; diagnosis answers another.

Expectation can alter attention and descending pain control. Conditioning can recruit learned physiological responses. Endogenous opioids contribute to some placebo analgesia, and blocking opioid receptors can reduce those effects in certain experiments. These are changes in living tissue, not an admission extracted from an unreliable witness.

There is a second confusion hidden in the word response. Improvement after receiving a placebo can include natural recovery, fluctuating symptoms and other changes that would have happened anyway. To isolate an effect of treatment context, an experiment needs an appropriate comparison. A patient feeling better is welcome news, but the sequence alone does not tell us why.

Where a context effect is established, it still does not identify the original cause or promise lasting relief. The ethical lesson is to use explanation, trust and expectation honestly alongside treatments with defensible benefits.

Neither dismissing the patient nor selling a cure on the strength of a short laboratory effect follows from the evidence.

"The aim is to silence pain completely"

Relief matters. Severe pain can prevent sleep, breathing, movement, work and clear thought. Some causes can be removed, and good acute care should not preserve suffering to prove that pain is useful.

Zero becomes a dangerous universal target when every residual sensation is treated as treatment failure. Medicines are escalated despite diminishing benefit. Procedures chase abnormalities with weak causal links. Activity is postponed until a body feels perfect. In persistent pain, the pursuit can enlarge side effects, dependence, fear and disability while the original condition changes little.

A better target has several dimensions: reduce pain where possible, treat the cause, improve sleep and function, widen safe activity, shorten flares and limit treatment harm. The balance differs for a postoperative wound, cancer, nerve injury, migraine and chronic primary pain. No single ladder applies to all.

Function is not a consolation prize and should not be used to dismiss intensity. A person may live better before pain disappears, and greater activity can be meaningful even when symptoms remain. The system can also produce valuable warning during recovery. The aim is proportionate care: treat what can be treated, reduce suffering, preserve safety and function, and avoid creating more harm in pursuit of a perfect score.

Use It

Treat pain as evidence, not a verdict

Pain deserves attention because it is direct evidence of a person's experience and a forceful change in bodily priority. The mistake is letting the experience settle every question at once. It can establish that a person hurts. It cannot, by intensity alone, establish what is damaged, where the cause sits, whether movement is unsafe or which treatment will work.

Use two columns in thought. The first contains the observed pattern: location, onset, character, triggers, associated changes and urgency. The second contains the apparent conclusion: the joint is ruined, the nerve is trapped, the exercise caused fresh injury, the scan must show it. The pattern is data. The conclusion is a hypothesis.

This separation prevents two opposite errors. It stops a person from assuming that every flare proves harm. It also stops a clinician from assuming that a poor match between pain and imaging proves exaggeration. Both parties can take the symptom seriously while remaining willing to revise its explanation.

The habit is useful beyond medicine. Strong internal signals often arrive with interpretations attached. Fear says danger is certain. Fatigue says continuation is impossible. Pain says the place that hurts is damaged now. Respect the experience first. Test the embedded claim second.

Follow the pattern, not one number

A zero-to-ten score compresses a changing system into one digit. It can help track one person's pain under comparable conditions, but it hides what changed. A six that permits work and sleep differs from a six that wakes the person hourly. A brief eight after a known movement differs from a new eight accompanied by weakness or fever.

Patterns carry more information. Record onset, duration, distribution, triggers, recovery time, sensory changes, sleep, function and treatment

effects. Ask whether the painful area is fixed or spreading, whether the same load produces the same response and whether a flare returns to baseline. The trend across days or weeks often matters more than the worst minute.

Pattern also improves communication. Saying that pain rose after ten minutes of walking and settled within an hour gives a clinician more to test than saying the leg is always bad. Saying that a medicine reduced pain by one point but restored sleep while causing dizziness reveals both value and cost.

Numbers feel objective because they are easy to compare. Pain measurement becomes more honest when several dimensions remain visible. Intensity, interference, function, distress and safety are related outcomes, not interchangeable units.

Ask what mechanism the treatment is targeting

Every intervention tells a causal story. An anti-inflammatory treatment assumes inflammatory mediators matter. A nerve block assumes a particular route carries important input. Graded exposure assumes a chosen activity can be expanded safely enough for repeated experience to reduce fear and avoidance. Different stories should lead to different expectations.

Imagine being offered an injection for a persistent pain. It is reasonable to ask which structure or pathway is being targeted, why the clinician thinks it matters here and what a useful result would look like. Brief relief might help answer a diagnostic question or make movement easier. That is a different promise from lasting relief. Knowing which promise is being made prevents one result from being quietly substituted for the other.

The same discipline applies to tablets, exercise and psychological treatment. Agree what benefit would count, when to assess it and what harm or failed result would prompt a change. An intervention that cannot fail any test is difficult to distinguish from a ritual with an invoice.

Intensity alone is a poor selector. Stronger pain does not automatically

require stronger medicine, and gentle does not automatically mean effective or low-risk. The intervention has to earn its place in this person's care, not merely sound appropriate to the severity of the complaint.

Make uncertainty informative

Pain produces an appetite for certainty. A structural label can satisfy it, even when the finding is common in people without symptoms. A clinician may offer reassurance by saying nothing is wrong, which leaves the patient with severe pain and no model. Both responses close the case too early.

Useful uncertainty has boundaries. State what dangerous causes have been considered, what evidence supports the leading explanation, what remains unknown and which change would trigger review. A normal scan can make some diagnoses less likely without proving that the nervous system is normal. A plausible disc or tendon finding can guide care without becoming the whole person.

Language alters threat. Catastrophic metaphors such as crumbling, bone on bone or worn out can turn ordinary movement into a test of structural survival. Empty reassurance can do the same by forcing the person to monitor symptoms alone. Better explanations connect findings to probability, function and a plan.

The aim is not certainty at any cost. It is enough shared understanding to act, observe and update. A provisional model that predicts what happens next is more useful than a confident label that explains every outcome after it occurs.

Test capacity with tolerable evidence

When pain, fear and avoidance have narrowed activity beyond what the diagnosis requires, argument alone rarely restores capacity. A feared movement performed at a tolerable dose, with an understood purpose and a recoverable response, can provide evidence that greater activity is possible. Repetition matters because one tolerable attempt is weak evidence, while a stable pattern supplies more.

This is not an instruction to push through every pain. The relevant dose depends on diagnosis, tissue state, neurological signs, health, medication and the person's goals. Some conditions require protection, surgery or medical control before exposure is sensible. Worsening function or new symptoms can change the plan.

The useful distinction is between hurt and harm without pretending they are always separable in advance. A planned increase can be followed by agreed criteria: how much symptom change is acceptable, how long recovery should take and what would count as excessive. Capacity then grows through controlled tests rather than bravery contests.

Improvement may first appear around the pain. The person sleeps better, walks farther, worries less about a flare or returns to work while intensity moves slowly. Those changes are not evidence that the pain never mattered. They show that pain no longer controls the entire life.

The limits

This book cannot diagnose a symptom, identify a safe exercise dose or choose a medicine. Pain biology describes mechanisms shared across people; clinical decisions depend on age, pregnancy, disease, injury, medication, examination and local guidance. A familiar pattern can acquire a new cause, and a person with chronic pain remains capable of fracture, infection, vascular disease, cancer and every other ordinary pathology.

The protective model also has rhetorical risks. Calling pain an output can sound as though it is optional. Calling chronic pain an overprotective alarm can erase ongoing disease, nerve damage and social conditions. Calling treatment retraining can imply that failure reflects poor effort. No mechanism justifies disbelief.

The categories remain incomplete. Nociceptive, neuropathic and nociplastic pain overlap. Central sensitisation has no single routine clinical test that explains every case. Brain imaging has not supplied a general clinical replacement for individual pain reports. Treatment evidence is often based on group averages with modest effects, short follow-up and populations that

do not represent everyone.

Pain can be useful and still deserve relief. It can be disproportionate and still signal something serious. A model earns trust by preserving both possibilities until evidence narrows them.

The one thing to keep

The hot pan at the beginning has not become less dangerous. You should still take your hand away. Understanding pain does not require a seminar before every sensible act of self-preservation.

What changes is what you infer afterwards. The speed and certainty of the hurt can make it feel like a complete explanation: something is damaged here, by this much, and it will remain dangerous until the sensation stops. Sometimes that account is close enough. Sometimes the pain persists after the first cause has changed, or the cause lies somewhere else, or the sensory system itself is part of the problem. The experience does not announce which case you are in.

That gap matters most when you encounter someone whose suffering you cannot see. A normal scan does not settle the question by making the person wrong. Nor does believing the person require believing every explanation offered for the pain. You can take the experience seriously while continuing to investigate its cause. In fact, serious attention requires both.

The aim is not to win an argument with a nervous system. It is to understand enough to act: remove danger where possible, relieve suffering and recover room to live. Pain can command attention without supplying the final verdict. A person who has spent months being told either that nothing is wrong or that everything is damaged deserves more than a choice between those answers.

Terms

Pain. A personal, unpleasant experience with sensory and emotional dimensions, linked to or resembling the experience of actual or possible tissue harm. Nociceptor activity alone cannot establish it.

Nociception. The neural process of encoding noxious events. It includes transduction, conduction and central processing, and can occur without conscious pain. Pain can also occur without continuing peripheral nociception.

Noxious stimulus. A mechanical, thermal or chemical event that damages or threatens damage to normal tissue. Threatens matters: nociceptors can respond near the boundary of injury, before measurable destruction has occurred.

Nociceptor. A high-threshold peripheral sensory receptor capable of transducing and encoding noxious stimuli. Calling it a pain receptor is misleading because it supplies input rather than containing the experience.

Transduction. Conversion of mechanical force, temperature or chemical change into electrical activity at a sensory ending. Ion channels perform the first translation from a bodily event into neural evidence.

A-delta fibre. A thin, lightly myelinated sensory fibre that conducts faster than an unmyelinated C fibre. Its activity often contributes to the first pricking sensation after an abrupt noxious event.

C fibre. A thin, unmyelinated sensory fibre with slower conduction. C fibres often contribute to burning, aching and later pain, though fibre type never determines the complete experience by itself.

Dorsal horn. Grey matter at the back of the spinal cord where sensory afferents first meet central circuits. Excitation, inhibition and descending control begin reshaping nociceptive input here.

Withdrawal reflex. Rapid spinal organisation of muscles away from a noxious stimulus. It can begin before conscious pain is fully formed, showing

that motor defence and experience are related but separable.

Spinothalamic tract. A major ascending route carrying information about noxious stimulation, temperature and touch towards the brain. Pain depends on wider networks, so the tract is not a dedicated pain cable.

Descending modulation. Control travelling from cortical and brainstem systems towards the spinal cord. It can inhibit or facilitate nociceptive processing according to attention, expectation, threat, context and behavioural state.

Somatosensory cortex. Cortical regions contributing to the location and physical qualities of bodily sensation. They help map pain but do not alone generate its unpleasantness, meaning or behavioural priority.

Insula. A cortical region involved in representing internal bodily state, salience and feeling. It is often active during pain but also contributes to many non-painful experiences and decisions.

Anterior cingulate cortex. A set of medial frontal regions involved in unpleasantness, attention, motivation and action. Activity here is neither unique to pain nor an objective measure of suffering.

Endogenous opioids. Opioid peptides produced within the body, including endorphins and enkephalins. They influence pain processing, stress responses and some placebo effects through opioid receptors in several neural locations.

Gate-control theory. Melzack and Wall's 1965 proposal that spinal transmission depends on competing peripheral and central inputs. Modern circuitry is more complex, but the passive pain-wire model did not survive.

Referred pain. Pain felt at a site different from its principal source, often when visceral and somatic inputs converge. The experienced location is a nervous-system interpretation, not random imagination.

Phantom-limb pain. Pain experienced in an amputated or absent limb. Peripheral, spinal and brain mechanisms can contribute, demonstrating that

the body's experienced map can persist after anatomy changes.

Hyperalgesia. Increased pain from stimulation that normally provokes pain. It can arise through peripheral or central sensitisation and must be distinguished from a more dangerous underlying process where relevant.

Allodynia. Pain caused by stimulation that does not normally provoke pain, such as light brushing. It is common in several neuropathic and sensitised states and reveals altered processing.

Peripheral sensitisation. Increased responsiveness and reduced threshold of peripheral nociceptive neurons, often driven by inflammatory mediators. It can reduce use of injured tissue while also contributing to tenderness and hyperalgesia.

Central sensitisation. Heightened responsiveness in central nociceptive neurons, including responses to input that was previously normal or below threshold. It is a supported mechanism, not a universal clinical diagnosis.

Temporal summation. A progressive increase in perceived pain during repeated identical stimulation. It reflects time-dependent central processing and can help study sensitivity, though it does not identify one disease.

Nociceptive pain. Pain arising when actual or threatened damage to non-neural tissue activates normally functioning nociceptors. Intensity still does not measure damage precisely.

Neuropathic pain. Pain resulting when disease or a lesion affects the somatosensory nervous system. Diagnosis requires a plausible neural distribution and supporting evidence, not merely burning or electric descriptive language.

Nociplastic pain. Pain attributed to altered nociception without clear evidence that actual or threatened non-neural tissue damage, or somatosensory disease or lesion, adequately explains it. It can coexist with other mechanisms; it never means imaginary pain.

Acute pain. Pain linked to a recent injury, illness, noxious event or

procedure, usually over a limited period. It often has adaptive value but may still require strong relief and active treatment.

Chronic pain. Pain that continues or returns beyond three months in current international classification. The threshold aids recognition and coding but does not define one mechanism or prognosis.

Chronic primary pain. Chronic pain recognised as a condition in its own right, accompanied by significant distress or functional disability, when another diagnosis does not better account for the presentation.

Chronic secondary pain. Chronic pain associated with an underlying condition, including cancer, surgery, nerve disease, secondary headache, visceral or musculoskeletal disorders. It can coexist with chronic primary pain in one person.

Go Deeper

The accessible overview. Monty Lyman, *The Painful Truth: The New Science of Why We Hurt and How We Can Heal* (Bantam Press, 2021). Lyman is a doctor and researcher who explains the protective model through patients, experiments and his own encounters with pain. It is inviting, clear and especially good on why context and learning change biology. Read it as a route into the science, not as a promise that one explanatory model or self-directed programme can resolve every chronic condition. It works best after this book because the recurring stories supply lived detail while the broader classification and evidential limits remain visible.

The major synthesis. Patrick Wall, *Pain: The Science of Suffering* (Columbia University Press, 2000). Wall helped overturn the passive-wire account and spent a career studying how pain is regulated. This book ranges from peripheral nerves to war injury, placebo, treatment and the moral problem of another person's suffering. Some terminology and evidence have aged, but the questions remain sharp. It shows how a scientist can defend mechanism without reducing the person to circuitry.

Wall also writes with unusual impatience towards tidy divisions between tissue, mind and society, making the book a useful bridge between laboratory explanation and the clinical encounter.

The original intervention. Ronald Melzack and Patrick D. Wall, “Pain Mechanisms: A New Theory” (*Science*, 1965). The paper is short, technical and historically decisive. Its gate-control proposal made room for touch, spinal inhibition and descending influence inside pain biology, changing both research and treatment. Read it for the break it created rather than as a current wiring diagram. Later work revised many details while leaving the old direct-transmission model untenable. Pair it with a modern review of dorsal-horn circuitry if the cellular details matter to you. The reward here is seeing a field change its governing picture in real time.

The cultural history. Joanna Bourke, *The Story of Pain: From Prayer to Painkillers* (Oxford University Press, 2014). Bourke follows how people have described, displayed, judged and treated pain across medicine, religion, war and ordinary life. The book is strongest on the social problem neuroscience cannot settle: how one person's private experience becomes credible to another. It is a corrective to accounts that make receptors and scans the whole subject, though it is history rather than a clinical guide. Bourke also shows why stoicism, gender, class, race and professional authority affect which suffering receives language, belief and relief.

Notes and Sources

Key mechanistic claims, clinical guidance and bibliographic details were rechecked on 5 September 2026. Pain terminology continues to develop, particularly around nociplastic pain, chronic primary pain and the clinical interpretation of sensitisation. The manuscript therefore uses current international definitions while keeping disputed mechanisms and diagnostic limits visible.

Definition and the title

The International Association for the Study of Pain revised its definition in 2020. The definition treats pain as a personal sensory and emotional experience associated with, or resembling that associated with, actual or potential tissue damage. Its accompanying notes state that pain and nociception are different phenomena, that biological, psychological and social factors influence pain, and that a person's report should be respected. Raja and colleagues explain the reasoning and compromises behind the revision.

The subtitle's claim that pain “lies” is a metaphor for mismatch, not deceit. The manuscript does not imply that a person has misreported an experience. It argues that pain can misrepresent location, scale, persistence, mechanism or urgency because the experience integrates nociceptive input with wider bodily and contextual processing rather than directly measuring tissue. IASP notes that pain usually has an adaptive role but may also harm function and well-being. The exact phrase “pain lies” remains the book's organising judgement rather than an IASP term.

Protective function and congenital painlessness

Cox and colleagues described six affected people in three consanguineous families in northern Pakistan with congenital inability to experience pain caused by loss-of-function mutations in SCN9A, which encodes the voltage-gated sodium channel Nav1.7. The paper reports an index case who performed street entertainment involving knives and hot coals and died after jumping from a house roof on his fourteenth birthday, before the researchers could examine him. The affected people who were examined had histories including fractures, burns, cuts and injuries to the tongue or lips. The manuscript preserves the paper's sequence and does not assign a motive to the fatal jump.

Congenital painlessness is rare and does not prove that every pain experience is necessary, adaptive or correctly calibrated. It establishes the narrower point that losing pain can expose tissue and joints to repeated

injury. Goodwin and McMahon review how Nav1.7 and other sodium channels contribute to peripheral excitability while warning against treating one channel as the whole pain system.

Nociceptors, transduction and the first signals

Julius and Basbaum and the later review by Basbaum and colleagues support the account of specialised peripheral sensory neurons, transduction channels, inflammatory mediators, A-delta fibres, C fibres and central pathways. Nociceptors are high-threshold peripheral sensory receptors capable of transducing and encoding noxious stimuli, including damaging or potentially damaging mechanical, thermal and chemical events. They do not contain a conscious experience or encode a universal damage score.

Capsaicin activates TRPV1, a channel also sensitive to damaging heat and other chemical conditions. That shared molecular entry point supports the chilli example without implying that eating chilli produces the same tissue state as a burn. The contrast between faster, often sharper first pain and slower, often burning or aching second pain is a useful teaching pattern, not a law that assigns one quality to every A-delta or C fibre.

Inflammation and peripheral sensitisation

Inflammatory mediators can lower nociceptor thresholds and increase responsiveness around threatened tissue. Basbaum and colleagues and Woolf support the distinction between peripheral sensitisation near tissue and central sensitisation within the central nervous system. Tenderness after injury can reduce loading while healing proceeds, though excessive pain can obstruct recovery. The manuscript avoids claiming that all inflammation is painful, all pain is inflammatory or every tender area is damaged.

The asymmetry in the book, that missing some dangerous events may cost more than overreacting to a harmless event, is a functional inference rather than a measured universal ratio. It helps explain why caution can be adaptive without implying that every pain episode has a current protective purpose.

The spinal cord, withdrawal and gate control

Melzack and Wall's 1965 paper proposed that spinal transmission depends on the interaction of peripheral input and central control rather than a passive line from tissue to brain. Later work revised the original circuitry, but the paper remains the decisive historical break with a fixed transmission model. The manuscript uses gate control as a conceptual inheritance, not as a complete current wiring diagram.

Basbaum and colleagues support the dorsal horn account, including local excitatory and inhibitory circuits. Fields and the review by Heinricher and colleagues describe descending control from brainstem and higher systems, including inhibition and facilitation. A withdrawal reflex can be organised through spinal circuits before the conscious experience is fully formed. This timing does not make pain irrelevant; it shows that rapid protection and conscious suffering are related processes rather than one event.

Rubbing an injured area can alter input through touch-sensitive afferents and spinal processing. It is used as a familiar illustration, not as a treatment claim or proof that touch will relieve every pain.

Distributed brain processing

Tracey and Mantyh review the cortical and subcortical systems contributing to sensory discrimination, unpleasantness, salience, motivation and modulation. Iannetti and Mouraux explain why the frequently repeated idea of a dedicated “pain matrix” is misleading: many regions activated during pain also respond to salient, threatening or behaviourally important non-painful events. The manuscript therefore describes a distributed contribution rather than one pain centre.

Davis and colleagues set out medical, legal and ethical constraints on pain imaging in 2017. Lee and colleagues' 2026 study adds personalised prediction of fluctuations from repeated scans in two people. The models did not generalise across patients. This advance does not establish a general clinical replacement for report or a pain lie detector.

Location, referred pain and phantom limbs

Referred pain is consistent with convergence and ambiguity in somatic and visceral pathways. The precise pattern depends on the organ, spinal level and person, so the manuscript avoids turning one classic map into a universal rule.

Flor, Nikolajsen and Staehelin Jensen review phantom-limb pain as a product of interacting peripheral, spinal and brain changes. The existence of pain in an absent limb establishes that the experienced body is neurally represented, but it does not prove that phantom pain has one cause or that cortical reorganisation alone explains it. The book keeps that plurality visible.

Attention, expectation, placebo and nocebo

Bingel and colleagues manipulated treatment expectation while healthy volunteers received remifentanyl during controlled heat pain. Positive expectation roughly doubled the observed analgesic benefit in that setting, while negative expectation abolished it. The study is memorable and causally strong within its design, but it concerns an experimental stimulus, a specific opioid and a small healthy-volunteer sample. The manuscript uses it to show that expectation can change drug effect, not to quantify every clinical placebo or nocebo response.

Eippert and colleagues provide evidence that placebo analgesia can recruit endogenous opioid and descending pain-control systems. Colloca and Barsky review learning, expectation, communication and clinical context across placebo and nocebo effects. These sources support the statement that context changes biology. They do not justify telling a patient that positive thinking will remove disease or that failure to improve reflects inadequate belief.

Rossettini and colleagues distinguish contextual effects from the other reasons symptoms may change during treatment, including natural history, regression towards the mean and concurrent care. The book uses that methodological distinction, not physiotherapy-specific results, to explain why

improvement in a placebo group is not itself a measure of a placebo mechanism.

Attention, fear, safety, perceived control, memory and present demands can alter pain through overlapping mechanisms. The book describes these as contributors whose weight varies. It does not claim that one cognitive variable determines a person's pain.

Nociceptive, neuropathic and nociplastic pain

The IASP terminology page supplies the definitions of nociceptive, neuropathic and nociplastic pain. Neuropathic pain requires a lesion or disease of the somatosensory nervous system; a burning or electric quality alone is insufficient. Nociplastic pain describes altered nociception without clear evidence that actual or threatened non-neural tissue damage, or a somatosensory lesion or disease, adequately explains the presentation.

Kosek and colleagues propose clinical criteria and a grading system for chronic nociplastic musculoskeletal pain. Clinical criteria exist for chronic nociplastic pain affecting musculoskeletal structures, while validation and transport to other presentations remain incomplete. It must not be used as a loose synonym for central sensitisation, medically unexplained symptoms or pain without biology. Häuser and Kosek's review, first published online in November 2025 and assigned to a 2026 issue, reinforces the distinction from central sensitisation and chronic primary pain and the need for further field validation. Mechanisms can coexist, and a classification can change as evidence changes.

Central sensitisation and persistence

Latrempoliere and Woolf and Woolf's later clinical review describe central sensitisation as increased responsiveness of nociceptive neurons in the central nervous system to normal or subthreshold input. Experimental signs include expanded receptive fields, reduced thresholds and enhanced responses to repeated input. Hyperalgesia, allodynia and temporal summation can be consistent with sensitisation but are not exclusive fingerprints of one mechanism or condition.

The book's account of persistence combines continuing peripheral input, nerve pathology, central plasticity, learning, sleep, distress, movement, work and social conditions. This is a systems account, not a claim that every factor is active in every person. Experimental and review evidence supports a two-way relationship between disturbed sleep and pain sensitivity, but transport to an individual chronic condition remains uncertain. The suggestion that avoidance can maintain disability and sometimes pain is conditional. Avoidance can also be sensible when tissue remains unstable or disease is active.

Chronic primary and chronic secondary pain

Treede and colleagues explain the ICD-11 classification of chronic pain and the distinction between chronic primary and chronic secondary categories. The three-month threshold is a classification convention for persistent or recurrent pain; it does not establish a single biological transition on day ninety-one or assume that every tissue should have healed by then.

Chronic primary pain recognises pain and related distress or disability as a condition in its own right when another diagnosis does not adequately account for the presentation. Chronic secondary pain remains associated with an underlying disease or injury category. The two can coexist. The manuscript rejects the false choice between continuing pathology and altered processing.

Scans, lesions and clinical explanation

Brinjikji and colleagues systematically reviewed degenerative spinal imaging findings in asymptomatic people. Several findings became more common with age, showing why an abnormal image can be real without explaining a person's pain. The review does not show that spinal findings never matter. The manuscript therefore requires fit among history, examination, distribution, timing and the decision a test would change.

The imaging sources distinguish experimental prediction from an established clinical pain meter. Tests can identify causes, correlates and consequences; self-report remains central where communication is

possible. A normal scan can lower the probability of some diagnoses without proving that no pain mechanism exists.

Report, communication and unequal credibility

The IASP definition says that a person's report should be respected. Schofield's UK guidance for older people treats self-report as the most valid direct indicator where communication is possible. Behaviour, physiology and observation matter when self-report is limited, but they are indirect and can miss quiet, masked or unfamiliar expressions of pain.

Anderson, Green and Payne review racial and ethnic disparities in pain care. Hoffman and colleagues experimentally linked false beliefs about biological differences to biased pain ratings and treatment recommendations among white medical students and residents in a United States study. Samulowitz and colleagues review gendered norms and bias in chronic pain care. Lor and colleagues review pain research involving people with limited English proficiency. Schofield addresses age, cognition, communication and cultural factors in older people. Together these sources support the bounded claim that identity, communication and clinician expectations can affect assessment and treatment. The evidence comes from different countries, settings and methods, so the book does not imply one identical disparity everywhere.

Treatment and current guidance

The National Institute for Health and Care Excellence guideline NG193 covers assessment of all chronic pain in people aged sixteen and over and the management of chronic primary pain. For chronic primary pain it recommends offering supervised group exercise and considering acceptance and commitment therapy or cognitive behavioural therapy for pain. Selected antidepressants may be considered for adults aged eighteen and over, with specialist advice needed at sixteen or seventeen. It advises against initiating several drug classes for chronic primary pain. Those recommendations must not be transported to acute pain, cancer pain, palliative care, confirmed neuropathic pain or every secondary pain condition.

The Faculty of Pain Medicine's current Opioids Aware resource, which states that its webpages are undergoing updates, supports the manuscript's bounded opioid account. Opioids have established roles in selected acute, cancer and palliative settings, while long-term use can bring tolerance, dependence, sedation, constipation, overdose risk and limited benefit for many chronic non-cancer presentations. MHRA guidance supports the distinction between physical dependence and addiction and the warning against abrupt, unsupported withdrawal. The book offers no individual prescribing or tapering instructions.

Movement, pacing, graded exposure and psychological therapies are described as routes to function, confidence, reduced avoidance or reduced interference when they fit the mechanism and person. They are not presented as proof that pain began in thought, nor as substitutes for investigation of progressive neurological loss, infection, fracture, cancer, inflammatory disease or vascular problems.

The NINDS overview and NHS pages on back pain and chest pain inform the safety passage. NHS advice specifies emergency action for relevant chest-pain patterns and for back pain with new genital or anal numbness or bladder or bowel dysfunction. The 999 and A&E instructions are explicitly UK-specific. These examples are not a complete triage rule.

Evidence across methods

The hierarchy in the final evidence section reflects the strengths and limits of each method. Cellular recording and molecular studies can identify transduction and excitability. Animal work permits invasive causal experiments but cannot supply a human report. Human psychophysics controls stimuli and expectations but compresses the social and clinical world. Lesions, nerve blocks and medicines can test pathways while remaining imperfectly selective. Imaging can map group patterns and, experimentally, predict within-person fluctuations; neither is independent certification of another person's experience. Clinical cohorts capture lived complexity while mixing mechanisms, treatments and directions of causation.

Convergence therefore carries more weight than one striking method. The manuscript treats the current classifications as useful working structures, not discovered natural boxes. It also keeps the distinction between explaining pain and measuring another person's pain.

Go Deeper editions

The cited editions were checked against publisher or bibliographic records. Lyman's book was published by Bantam Press in 2021. Wall's general-reader synthesis was published by Columbia University Press in 2000. Melzack and Wall's paper appeared in volume 150 of *Science* in 1965. Bourke's cultural history was published by Oxford University Press in 2014. Each recommendation is included because it performs a different job rather than because it agrees with every judgement in this manuscript.

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