PAIN

Severe pain demands attention. It warns of potential disaster. Reflex responses guard against more injury. They include a display of suffering that has its effect on others. In a compassionate society the display generates comfort and aid. It drove the medicine, which has grown out of the ancient arts for alleviating pain.

The most ancient medications such as opium alleviate pain whatever the cause, but temporarily. The brain develops a tolerance that gradually diminishes this benefit. All pain-relieving medications fail in time if the cause remains unaddressed. A more critical factor than the development of tolerance underlies this failure. The greatest weaknesses of medicine exist where it lacks the capacity to identify cause and can only address the symptoms of disease. Effect, in the case of pain no more than the display of suffering, does not distinguish possible causes that range from life-threatening disease to the faking of illness.

On the broader canvas of life the same hazard besets all human effort to counter the painful consequences of poorly understood causes. It may be completely misdirected. The effectiveness of modern evidence-based medicine began in 1890 when Robert Koch devised the principles of identifying the cause of disease.

Cause and Effect

The practical difficulties of managing pain result from the effort to alter an effect without knowing the cause. Over the millennia we have succeeded so well in turning cause to good effect that we have the conceit we can manage effects without knowing the cause. The conceit blinds us to the absolute barrier posed by the one-way direction of time. Using hindsight to reason backwards from an effect cannot penetrate the barrier of time passed to discern which possible cause of the many on offer contributed. The knowledge we have derives not from a capacity to discern the cause of the effect we see, but from the experience that repeatedly revealed what effects follow known causes. It bears repeating that pain like its display of suffering is an effect.

More than 250 years ago David Hume observed, in what I propose we recognise as Hume's law (Bell, 2015, see Chap.5), that we cannot know the connection between cause and effect. If we could we would know how everything about us came about. And yet humans have exercised that incredible vanity from time immemorial in myth, folklore and religion. Each group does not even learn from what we can recognise as the arrant nonsense of other groups, much less our own. Not surprisingly, Hume risked the wrath of the Inquisition. To this day at law the exercise of hindsight brings all too common failure such as the conviction of innocents (ibid.).

We do not need hindsight to learn what causes an effect. We succeed with experiments that allow us to observe what follows a cause. Long ago we reached whatever mastery we have in precisely that way with experiments that obey the one-way direction of time. During more than a million years of the stone-age our ancestors did precisely that, experimenting with the blows that shape stone with stone into the weapons that made sapiens so powerful. Their sustained deliberate efforts directed our evolution of bodies and brains and our ability to communicate what we learn in language (Blackmore, 1999).

Now in a supposedly enlightened age pain management poses the temptation to address effect not knowing the cause that brings about its characteristic display. Of course, when the doctor has no way to gather knowledge of the cause, temptation becomes forced necessity. Ignorance

creates the principal hazard of pain management, the encouragement of psychological maladaptation and drug addiction.

All pain-relieving drugs generate euphoria, the gain to be had from the mere display of suffering. The prescriber has no other guide than a display that can be readily contrived. Of course the accumulation of observations about what manner of pain follows a known cause gives a clue to the likely cause. It also provides clues to the different display seen in the absence of disease. This text concerns the assessment of pain behaviour in the relatively affluent peaceful community, where the simulation of the complaint for gain has become a common hazard for the diagnostician and the courts and an increasing burden for the compassionate society.

The Classification of Pain

The fundamental barrier to diagnosis based on a symptom such as pain creates great uncertainty (Illich, 1976, p.140). Certain patterns of complaint such as that of renal colic provide helpful clues to the likely cause, but few conditions present so characteristically. Even it can be faked easily (see the case of Carlo in Chap.7 of Bell, 2015). The diagnostician can never be certain about the question of gain, which may be as obscure as the motivation of people, who fabricate complaint to harm themselves through unnecessary surgery that inflicts real injury.

The commonest error comes from confusing effect with cause. The International Association for the Study of Pain (IASP) has done just that. It adopted a principle, which converts the doctor's empathetic reception of complaint into a duty to believe the patient. The Association holds (p.S217) that if people report the perception of pain "it should be accepted as pain" (Merskey 1986b). Algologists carry this form of blind trust further by allowing the complainant to calibrate the severity of pain through ratings from one to ten. Ten years down the track not the objective signs of illness, but the patient's claim of how much pain they have predicts the duration of sick leave (Grossi *et al*., 1999).

The IASP justified its stance with an internally contradictory definition of pain. It begins by defining pain in unexceptional terms as an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage (Merskey, 1986b). It recognises equally correctly that injury causes pain. Elsewhere it concedes another obvious, namely, that pain is subjective. This is the setting in which it asserts, in what I will refer to from now on as *the assumption*, that the complaint "should be accepted as pain. This definition avoids tying pain to the stimulus."

Of course it poses contradiction. It had already tied the complaint to a specific cause, injury, before admitting that cannot be done, but without acknowledging the opening it creates for contradiction. To harbour contradiction equates incompatible opposites. The device provides the algologist with the tautology, which justifies any chosen cause. Because the mere say so of the patient establishes the authenticity of the complaint, the assumption takes from the clinician the professional responsibility for the key task of assessment. At the same time it enforces the medical imprimatur of approval. It has the effect of equating all pain, whether real, the result of psychological disorder, fancied or malingered. It provided a rationale to put aside responsible diagnosis.

How could the medical profession, and in turn the law, accept this misleading sophistry? They

fail to recognise that although the logical order from antecedent to consequent establishes that injury causes pain, it does not justify reasoning in reverse to hold that the complaint of pain proves the presence of injury. The same physicians would not dare argue that because infection causes fever, the complaint of feeling hot proves the presence of an infection. The reasoning from the consequent leads back to a great variety of antecedents without providing a guide for making the correct choice.

Pain specialists seriously maintain that the distinction serves no purpose. They hold that the pain of psychological origin feels the same as the pain of physical origin. They have absolutely no evidence to support their contention and ignore much that disproves it. Taken to its logical conclusion, the reasoning equates the pain produced by torture with the pain reported by people connected to a sham stimulator of electrical shock. Both even have the dose-response correlation which forms one of the indicia of a causal relationship. The imaginary pain increases in parallel with the setting of a sham gauge (Bayer *et al*., 1991). Real pain inflicted by electricity also increases in parallel with the settings of a voltmeter.

Not only are they different, but the sham state does not amount to malingering. It reveals a very different phenomenon. Ideas can evoke perception. The person need not have had the experience in the past. People who have never had an infestation of body or hair lice simply have an idea rather than experience. Given the belief alone of an exposure to lice, they soon begin to itch and scratch. Here we have the very opposite of the common understanding that awareness begins with perception. It also demonstrates that a simulated display of suffering does not necessarily mean that the person doing so set about faking deliberately.

Similarly, suggestion readily elicits other sensations as well such as fatigue, dizziness or a feeling of swelling. A hypochondriacally directed introspection will soon make any person start to feel pains here and there in the body. To equate these imagined sensations with the real prevents the distinction of illness from fake. It creates a gap which speculative notions and fashionable diagnoses fill. The passing scene provides a fascinating record of this medical mythology which comes and goes, eventually damned by the recognition of its falsity.

The IASP definition does not admit to the existence of feigned pain. The paradigm produces a selective blindness in this direction. After it appeared, for decades articles on the chronic pain syndrome almost totally disregarded the simulation or malingering of pain. In parallel, simulation expanded enormously. Aronoff (1991) estimated that one third of people attending the Boston Pain Clinic had "learned how to be pain patients" and as well another third complaining of pain did not deserve health care. Some pain clinics will not treat people with pending litigation.

The definition does not allow for "painmanship" (Milligan & Atkinson, 1991). Nor can it accommodate the "two week syndrome", shown by many receiving regular injections for their alleged pain. On next presenting for treatment, regardless of the interval since their last treatment, they claim the pain had returned between one and three weeks earlier. It makes no allowance for the vagaries of memory. People in treatment report gaining relief from pain even if it became worse (Feine *et al*., 1998). Those with the least pretreatment pain in recollection later exaggerate its intensity and those with the most pretreatment pain later underestimate it.

The Chronic Pain Syndrome

The label *chronic pain syndrome* indicates the persistent complaint of pain, which has no detectable cause. It does not distinguish real from imaginary pain. Rather, it conceals the failure to make the attempt. The act of awarding a name gives the misleading impression that the label indicates knowledge of real illness in its own right, a condition which has its own unique causes and is amenable to specific treatment and management. It achieves much the same trick as the word *idiopathic*, which means "no known cause". Paradoxically, in their case the label conceals the ignorance of the diagnostician with a label that declares it. The law in its turn succumbs to the magic of a name, accepting it as a real entity and rewarding its display with compensation.

In the past three decades the complaint has grown at an enormous rate (see "The Pain Empire" *http://brainaction.com/psychogenic-pain/the-pain-empire*). Paradoxically, it grows at a time when the medical ability to identify and treat the physical causes of pain improves rapidly in the very communities increasingly protected from injury and disease. The focus on pain has brought about an escalating deployment of health care resources for management of the symptom rather than the far more effective tackling of disease. As pain clinics have multiplied since the early 1970s (Williams, 1988), the "disability epidemic" of chronic pain has grown enormously. The sequence illustrates the disabling potential of the professions (Illich, 1977). The cases appear "where entitlement programs are viewed as appealing alternatives to gainful employment" (Aronoff, 1989).

A specific form of the chronic pain syndrome, the *regional pain syndrome*, illustrates how readily reason bypasses established knowledge. The regional distribution indicates pain localised to the lay idea of the body's regions such as a limb. Nerves supply an entirely different plan of regions known as dermatomes and the brain has yet another plan. The anatomical rules have such precision that the neurologist can predict the position of some lesions in the brain within a range of millimetres. In contrast the ideas of regions have the distribution that reveals their origin in the mind. For that reason the regional pain syndromes can have no other basis than psychogenesis. How is it that doctors ignore this basic fact? Epidemics of pseudo-illness such as repetitive strain injury (RSI) have involved large numbers of people in mass hysteria promoted by doctors (Bell, 1989; Bell, 2000).

The IASP definition incorrectly invests the chronic pain syndrome with the ominous significance attaching to the acute pain of disease. The distinction between acute and chronic separates two distinctly different entities. Acute and chronic pain are "not only different in time scale but are fundamentally different in kind" (Waddell, 1987). Most cases of chronic pain have no recognisable cause, they fail to present with the syndromes known to result from physical or mental illness and they certainly have a "psychological" component (Loeser & Cousins, 1990).

The so-called chronic pain of cancer and other chronic physical diseases more correctly belongs to the class of recurrent acute pain. Comparison with the chronic pain syndrome establishes that cancer brings about pain without the manipulative elements accompanying the chronic pain syndrome. It brings about a more unpleasant and intense sensation, but a lower level of emotional distress than chronic "benign" pain and a higher expectation of relief (Cohen *et al*., 1986). It correlates with the medical facts rather than the psychosocial features of the complainant (Syrjala & Chapko, 1995).

Despite its obvious faults, the IASP definition survives, perhaps because the definition serves the interests of the main players. The uncritical acceptance of a complaint assists the person

intent on gaining unjustified compensation, invites political favour and provides employment for lawyers, algologists and a host of paramedical workers.

In contrast to the acceptance of the concept by algologists, the Institute of Medicine (1987) concluded that the manifest deficiencies of the label "chronic pain syndrome" should exclude it from the regulatory listing of impairments eligible for disability payments in the United States. "There has been no demonstration of a common etiology, a predictable natural history, a clearly defined constellation of symptoms, or a specific treatment for the various pain conditions that would suggest a basis for positing a single chronic pain syndrome."

Chronic Pain in the Lower Back

The absence from work justified by the complaint of pain in the lower back has increased dramatically since the 1950s (Waddell, 1987). More than 30 years into this escalation the prevalence in the USA continued to increase at 14 times the rate of the population growth (Aronoff, 1991). In Sweden the rate of disability attributed to back pain increased eightfold from 1970 to 1987 and the number entitled to permanent disability pensions increased 60 times. When the cost reached the prohibitive level of 5% of gross national product, the government reduced compensation payments and Swedish backs suddenly improved (Malleson, 2002, p.78). Malleson reviews reports of equally illuminating extensive fraud committed in the USA.

The characteristic complaints exclude work as the likely cause. Those injured on duty have longer periods of disability compared to those injured off duty (Sander & Meyers, 1986). Their disorder does not correlate with the physical stress of the work (Bigos *et al*., 1986a). As a group they have a lower than average IQ, a shorter education and less satisfaction with their work (Bergenudd & Nilsson, 1988). They are likely to have earned a poor employee appraisal rating within the six months before injury (Bigos *et al*., 1986b). Resentment cripples them. Those who blame the employer for their pain have greater mood disorder, more behaviour disorder and a poorer response to treatment (DeGood & Kiernan, 1996).

They have as well the features of people who fail at other pursuits of life. Those with the most complaint are distinguished by a high rate of psychopathology (Bigos *et al*., 1986b; Grossi *et al*., 1999), which also applies to those who fail to respond to the conventional exhibition of analgesics (Magni, 1987). The explanation may lie in their response to pain. The magnitude of their fear of pain rather than the severity of the pain determines the severity of their disability (Crombez *et al*., 1999).

Psychiatric disorder plainly causes the disability of some. In one study about 12% seemed to have sufficient impairment to warrant total disability. Of this subgroup 50% had a psychiatric disorder unrelated to the work (Aronoff, 1991). Patients with chronic pain smoke more heavily (Deyo & Bass, 1989). Although they receive more treatment than other categories of illness, they show less benefit (Grossi *et al*., 1999; Reesor & Craig, 1988). Those pursuing a compensation claim respond poorly to surgery and rehabilitation.

Fittingly, the opposite qualities predict early improvement from pain in the lower back. Those who do well compared to the patients with the chronic pain syndrome have a low level of psychological distress, undertake more physical activity and are satisfied with their work. Their pain begins suddenly, has been present for a short duration when they seek treatment and is restricted to the lower back (Macfarlane *et al*., 1999).

Waddell (1987) reviewed the pitfalls that beset treatment. Not diagnosis, but abnormal illness behaviour determines the vigour with which treatment is pursued. Considering that no form of treatment has proven to be better than a placebo, the correlation makes sense. Not the cause, but the display of suffering decides the treatment. Even worse, certain treatments generate real disability where none had existed and most reinforce the sick role. Contrary to the general principle which applies to illness, rest increases the duration of the complaint and the insistence on a prompt return to work hastens recovery.

The situation since has not changed. Morley *et al*. (1999) join many others who maintain that cognitive behaviour therapy (CBT) provides real benefit. Their meta-analysis discloses that its only significant effect is the reduction of complaint. In short, the treated have learned how to behave better. Their mood disorder and catastrophization persist. No study demonstrates objective improvement in occupation, the use of drugs or the utilisation of health services. Rather, the settlement of the claim for compensation brings about these objective improvements (Guest & Drummond, 1992). Few return to the pain clinic after the resolution of litigation (Aronoff, 1991).

Psychogenic Pain

In the setting created by the IASP definition, the label of "psychogenic" pain implies that a disturbance of the psyche has brought about the same quality of pain and suffering as does injury. Its thrust depends on that assumption. No element of established knowledge supports it. Rather, its qualities and objective effects indicate a complaint that is "all in the mind." Furthermore, the people who advance this type of complaint have in common the proclivity to maladaptive behaviour and its consequences.

The DSM-III definition of **psychogenic pain disorder** required positive evidence of a psychological cause. No specific mechanism has surfaced (Ginzburg *et al*., 1988). The studies purporting to have found a specific background psychological disorder lack validity (Gamsa, 1994b). Because "such positive evidence is most often lacking in patients with intractable pain that has no adequate medical explanation" and the judgements "tend to be neither reliable nor valid" (Barsky, 1989, p.1022), the DSM-III-R discarded the need for positive evidence with the change in name to **somatoform pain disorder**.

Logically, the absence of evidence should have excluded the original speculation. Instead, the altered name signified a return to the speculative and unproven notions about hysteria renamed somatoform disorder, but still retaining the original unprovable concept of a causal unconscious mental conflict. The inexcusably speculative notions common to the concepts of hysteria and somatoform disorder persist despite their absurdity. Taylor (1986) explained this extraordinary aberration as the preference for any system of classification to none at all.

The results reveal their motivation. The rationalisations justify keeping as clients those feigning disorder. Pilowsky (1990) warned: "There is no way better calculated to alienate a patient with a somatoform pain disorder than to imply, directly or indirectly, that one does not accept the validity of the patient's complaint of pain." The doctor has to appease the patient and avoid the mention of psychogenesis (Shorter, 1992, p.217). Pilowsky (1994) illustrated a bleak alternative with the case of a patient who murdered the doctor. Effective alienation would have a far more likely consequence. It would largely empty the pain clinics of clients, reduce surgery on the lower back to miniscule proportions, deprive physiotherapists of the

greater bulk of their work and diminish greatly the call on medico-legal assessments.

The failure to admit the possibility of simulated illness makes an enigma of the data on the chronic pain syndrome. Many have wrestled earnestly with the unnecessary puzzles which ensue. Alter the paradigm and the findings fit well. They fit the characteristics of antisocial behaviour. In an extensive review, Turk *et al*. (1987) brought together the research which indicates that pain tends to cluster in families and to be associated with child abuse, aggression, marital conflict, sexual dissatisfaction, depression and anxiety. Had they not confused the "complaint of pain" with pain itself, the data would become more realistically oriented. For comparison they had studies dealing with undoubted pain; the families of those with painful diseases such as rheumatoid arthritis handled the illness very well and did not have the clustering of antisocial behaviour characteristic of pain without recognisable disease.

Research continues to produce much of the same. Fishbain *et al*. (1988a) found that, among a large group of consecutive admissions for chronic pain, those on compensation tended to have conversion and personality disorders. Constricted as they were by their conceptual approach, like Turk *et al*. (1987) they could draw no conclusion from the data. In a group of 135 cases of chronic pain, 28% had been subjected to childhood sexual abuse (Wurtele *et al*., 1990), but among women with some forms of chronic pain the rate of childhood physical and sexual abuse rises as high as 66% (Harness & Donlon, 1988). A comparison of various diagnoses confirmed the clustering of familial psychosocial disorder with psychogenic pain (Adler *et al*., 1989).

The search for evidence to support the paradigmatic blindness produces the inevitable burgeoning of inconclusive facts coupled with the rationalisations needed to put anomaly aside. Noting the excessive rates of a past history of major depression, family psychiatric disorder and sexual abuse in childhood, Gamsa (1994b) interpreted them to define the subgroups "at risk", but at risk of what? As antecedents those factors indicate a risk of recurrence or a risk for other people.

The comorbidity with depression indicates both conditions have a common causal trait of psychological vulnerability (Von Korff & Simon, 1996). Some faced with the evidence of simulation abandon conventional wisdom. Rosomoff *et al*. (1989) argued from findings which included non-anatomical sensory loss that the complaints resulted from musculoskeletal disease. They argued that even the deliberate and admitted fabrication of the complaint for a deliberate purpose could "be not wholly under conscious control" (Fishbain *et al*., 1988b).

Various euphemisms such as "medically incongruent pain" (Reesor & Craig, 1988) and "indeterminate pain" (Magni, 1987) assist the turning of the blind eye towards simulation. In a series of landmark studies, the signs of simulation were termed "nonorganic physical signs" (Waddell *et al*., 1980), "inappropriate descriptions of symptoms" and "inappropriate responses to physical examination" (Waddell *et al*., 1984). The label of "inappropriate" could only apply in the context of genuine illness. In the absence of disease the same complaints and signs point appropriately enough to the simulation of illness. The labels "abnormal illness behaviour" and "sick role" strike closer to the mark, but avoid the issue by implying that the problem is one of psychological illness.

McNeil *et al*. (1986) found the Pain Drawing Test and the Back Pain Classification Scale between them effectively identified the "psychologically disturbed". The authors acknowledged that they could not identify by the usual means the psychological disturbance.

In reality both tests only identify aspects of behaviour incongruent with genuine illness. They have no direct bearing on psychological disorder. Not psychological disturbance but simulation fits the observations.

The complainants volunteered "minimal information" about emotional and personal issues, they did not present with the usual behaviour or appearance of the psychologically disturbed nor did the mental status examination reveal the usual distinctive features. Attempts to administer a special investigation failed because "patients typically find these measures objectionable and often fill them out with extreme reluctance and defensiveness." Revealingly, these subjects claimed treatment had been effective and yet the "pain continues to remain fairly high and incapacitating to many in this group as judged by the level and quality of pain that continues to be reported."

Subsequent research reports use similar devices to maintain the illusion. Udén *et al*. (1988) equated the "nonorganic pain" revealed by pain drawings with "psychological disturbances" even though the usual psychometric instruments failed to reveal psychological disorder. They justified their failure to find the necessary supporting evidence with the argument that everyone knows "psychological testing is a rather crude and inexact instrument". Similarly, they dismissed the fact that these supposedly disturbed people had not sought the appropriate treatment on the grounds that consultation with a psychiatrist is "a rather blunt criterion of emotional disturbance".

These obtuse devices allow their adherents to deny the real significance of the clinical phenomena. The *Guides to the Evaluation of Permanent Impairment* (American Medical Association, 1990) contains the criticism of the medical profession "as a whole" for being slow to identify the chronic pain syndrome as a specific medical disorder requiring the attentions of the new specialty, algology. It holds that two or more of the six "D"s (ibid, p.249) establishes the diagnosis. My analysis of the six "D"s leads to the conclusion that they identify the simulation of pain.

Duration: Chronic pain persists and progresses long after tissue damage has healed, probably as the result of "underlying predisposing factors".

Reality: The absence of the time sequence diagnostic of tissue damage excludes it as a likely cause. The "underlying predisposing factors" are all mental.

Dramatisation: The patients describe their problem with emotionally charged words and exaggerated histrionic deportment, reflecting their "maladaptive conditioned behavior."

Reality: This behaviour reveals the motivation to impress with the display of contrived suffering and differs from that shown by the those with severe illness.

Drugs: Dependence on alcohol and/or prescription drugs forms part of their excessive use of health care. They submit to repetitive diagnostic studies, which generally are inconclusive or contradictory. They demand passive modalities of physical therapy, "which, although pleasant for the moment, provide no lasting benefit." The drugs produce their own disabling effects and, when all else fails, surgery all too often contributes iatrogenic pain and disability. In the "terminal" phase they are referred to the psychiatrist.

Reality: All too true. The display of suffering guarrantees the prescription of addictive drugs

such as the opiates for a long time. The misguided therapist gives up only when the failure of treatment becomes unavoidably obvious.

Despair: The manifestations include depression, apprehension, irritability and hostility leading to an embittered, defensive, rigid state.

Reality: These psychiatric complications do not complicate painful chronic physical illnesses. In the chronic pain syndrome they reflect a lifelong maladaptation that becomes increasingly evident to the disillusioned therapist.

Disuse: Prolonged excessive immobilisation and self-imposed splinting produces disability of its own and poor physical condition. The prolonged disuse may generate pain.

Reality: The efforts of those with genuine disability to overcome their handicap stand in remarkable contrast to acceptance of splinting by these patients.

Dysfunction: Withdrawal from social life, work and recreation. They alienate friends and family.

Reality: Severe chronic pain of physical origin can also bring about withdrawal, but in a different way, conspicuously without the anger and bitterness that alienates.

The Guides repeat the consensus of algologists that the alternative of malingering is "readily detected with appropriate medical and psychological tests." The Guides maintain that malingering is infrequent among those with chronic pain.

Reality: There are no specific medical tests for malingering and those used by the psychologists have gross limitations. When a simple observation such as looking at the signs of wear in the palms of the hand would provide a decisive answer, rarely does the physician record it, or even look for it. That algologists fail to even consider the possibility of malingering explains why they find it so infrequently.

Pain as a Depressive Equivalent

Like all fashion, the ruling of pseudo-illness changes regularly. These days chronic pain and neurotic depression usually occur together. A century ago classical hysteria presented with no apparent mood at all, *la grande belle indifference*. Some with the chronic pain syndrome still have this bland presentation, but since the diagnosis of depression became so popular in the second half of the 20th century it permeates most cases of pseudo-illness. It leads to the reasoning that one brings about the other. The interaction postulates a vortex of self-feeding disaster regardless of which comes first.

Magni *et al*. (1994) extracted from the National Health and Nutrition Survey the evidence that depression promotes pain and pain promotes depression, but, in contrast to the common co-existence of the two, the connection accounted for only a small proportion of the variance. The bulk of the comorbidity requires some other explanation.

Most instances of comorbidity reflect the consequence of "drawing borders where no genuine distinctions exist" (Van Praag, 1996). During a lifetime most neuroses prove interchangeable. The special interest of the diagnostician may decide which becomes the label. In the middle of

the 20th century the enthusiasm for the anti-anxiety drug amytal decided the preponderance of anxiety states until the advent of the newer antidepressants brought about the upswell of depression instead. The diagnoses which classify effects give diagnosticians this procrustean flexibility. A classification of effects rarely hits upon a cause.

Pain and depression are effects, which are entirely subjective and highly variable. Either can have any one of a great number of causes ranging from serious injury, mental or physical, to the opposite extreme, the simulation of illness. The diagnosis does not identify which of these vastly disparate causes have operated. The notion of comorbidity fails because of the fundamental error inherent in trying to turn a classification of effects to a purpose it cannot satisfy.

Similarly, the notion of the "depressive equivalent" lacks credence (Magni, 1987; Rudy *et al*., 1988). Pain does not bring about depressive illness. Depression occurs at a relatively low rate among those who have pain of undoubted organic origin (Magni, 1987). As an example of the contrary argument, Atkinson *et al*. (1991) claim that chronic pain in the lower back provokes major depression, increasing the risk of its appearance nine-fold. They also found an association with alcoholism, which clearly preceded the onset of the pain. They need not have gone any further. They had found an adequate cause of the depression in their cases.

Alcoholism is a potent cause of depression. Alcoholics have a suicide rate between 60 and 120 times that of the general population (Murphy & Wetzel, 1990). Atkinson *et al*. (1991) found that the risk for alcoholism did not increase after the onset of the complaints. The contradictory indications of the findings and their disparity with other studies reflects the confusion created by a failure to separate pain of undoubted physical origin from pain without recognisable cause.

The comorbidity of depression and the chronic pain disorder reveals their common constitutional origins. Both have in common mental vulnerability (Von Korff & Simon, 1996). Like those who have depressive illness, people complaining of chronic pain without obvious cause have high rates of mental aberrations such as hypochondriasis, neurosis, personality disorder and hysteria in a setting of family disorder and discord (Benjamin *et al*., 1988; Dworkin *et al*., 1990; Turk *et al*., 1987; Von Korff *et al*., 1988). They have a common bond of maladaptive behaviour (Rudy *et al*., 1988). Commonly the depression begins before the onset of the pain or both occur simultaneously (Sullivan *et al*., 1992).

Pseudo-illness

To ignore the mental origins of a complaint creates a gap which speculation readily fills. The passing scene provides a fascinating record of transient medical mythology, damned by the eventual recognition of its falsity. In most pain is a prominent complaint. Some notions endure for relatively short periods. As an example, epidemics of RSI move from one part of the world to another (Bell, 1997), but in any one place usually last for less than ten years (Bell, 1989). Change its name to regional pain has allowed it to rumble on in a less dramatic endemic form. *Carotidynia* received official approval by the International Headache Society Classification Committee in 1988 after hundreds of cases were reported in the 1960s and 1970s. Official approval proved the kiss of death. It vanished entirely in the next five years (Biousse & Bousser, 1994).

The chronic fatigue syndrome (CFS) disguises psychological complaint with in physical terms

including pain. It has diagnostic criteria so flexible that the notion becomes meaningless and yet its proponents claim it has a pathophysiology, diagnostic tests and a treatment. Neurasthenia fulfilled that purpose for earlier generations. Indeed, the label did not fall into disuse until its mental nature became clearly evident (Wessely, 1990). Similarly, the speculation about a physical basis to CFS reinforces denial of its mental nature (Wessely & Powell, 1989). The sufferers have a high rate of depression and the same characteristics as people complaining of chronic pain without recognisable cause, except that they tend to have more deviant personalities (Blakely *et al*., 1991).

The most durable pseudo-illness, *reflex sympathetic dystrophy*, has lasted for more than a century even though it is "neither reflex nor sympathetic nor dystrophy" (Ochoa, 1995). It still has currency, although isolated to use by "non-academic" clinicians, among whom its role "is almost accepted as dogma" (Jänig, 1991). Oddly enough, the beginning of disillusionment coincided with its official recognition by the IASP, once again in the 1980s. Behind dogmatic assertions about its validity lie ambiguities, contradictions and paradoxes (Bennett, 1991).

The flexibility of the supposed signs makes it possible to diagnose it in any case of unexplained pain. The skin may be hot or cold. The limb may have been injured or not. Signs may be totally absent. The definition alters from time to time, place to place or person to person (Ochoa, 1995). The variations rob the term of any potential scientific value (Jänig, 1991). The label could fit any of a variety of real organic disorders, a purely psychogenic condition or plain malingering (Ochoa, 1995). Nevertheless, it has accumulated a supposed physiopathology, x-ray signs, specific diagnostic tests and a treatment with destructive surgery. Critical evaluation establishes that they have no validity (ibid).

The notion of sympathetically mediated pain has its basis in erroneous observation (Ochoa, 1999). The test with phentolamine block brings about less effect than a placebo (Verdugo & Ochoa, 1994). It cannot identify genuine painful polyneuropathy (Verdugo *et al*., 1994). At best the treatments are useless, at worst they produce real injury (Ochoa, 1995).

Despite the difficulty of assessing those who present with subjective complaints alone, responsible assessment is possible. Acute and chronic pain caused by undoubted physical disorder are readily recognised. The patient’s description of pain reliably identifies syndromes such as sciatica, pain originating from the axial skeleton and referred pain (Bogduk, 1992). A careful analysis of key words assists (Waddell, 1987); a limited range of sensory-thermal terms points to pain of physical origin, as do physical precipitants, physical relieving factors (Boyd & Merskey, 1978) and a reactivity to movement (Adler, 1981).

An account of an improbable nature (Golla, 1949) or of an excessively wide distribution of the pain (Toomey *et al*., 1983) with the use of affective words (Tyrer, 1986), the picturesque and dramatic indicate the reverse (Guthkelch, 1980; Leavitt & Garron, 1982). During the interview, the person with learned pain behaviour may exaggerate the movements and expressions suggesting the experience of pain (Tyrer, 1986). The person in real pain becomes submissive, apologetic and subject to an irrational guilt (Robinson, 1992) in contrast to the assertive and demanding attitude of the person with learned pain behaviour.

Surprisingly, a history of disturbed sleep argues against physical disorder, correlating with depression and hypochondriasis (Pilowsky *et al*., 1985). Ancillary methods such as the pain drawing test of its distribution help to distinguish physical disorder from its simulation (Waddell, 1987). Pain having a "clear organic cause" correlates well with scores on pain

scales unlike the poor correlation shown by cases with "little demonstrable pathology" (Perry *et al*., 1991). The persistent use of addictive analgesic and sedative drugs augurs for a poor outcome (Turner *et al*., 1982).

The study of back pain confirms the validity of the long established principles of diagnosis. Waddell *et al*. (1984) found the inappropriate symptoms to include the excessive distribution of pain and numbness, continuous unremitting pain, intolerance of treatment and emergency admissions to hospital for pain. To these may be added unexplained progression of the severity and extent over time (Tyrer, 1986). The inappropriate signs are tenderness in an abnormal superficial region or in a non-anatomical distribution, an abnormal regional distribution of weakness or sensory impairment, reaction to a simulated provocative test, overreaction in the case of a genuine test or lack of reaction when distracted (Waddell *et al*., 1980).

On the other hand certain signs have doubtful significance. As an example of the contentious, the fact that a patient with lower back pain can sit upright on the examination couch does not necessarily conflict with the finding that straight leg raising is restricted, despite the frequent assumption in medical reports that this is so. Caution should also be used in arriving at a conclusion about the words used to describe pain. Agnew and Merskey (1976) found more similarities than differences in the words used by groups with organic and non-organic disease. At least one group with genuine organic disorder tend to use bizarre terms to explain their unpleasant sensations, that is, those with damage to the spinal cord (Patten, 1982, p.143).

The Treatment of Pain

The inability to distinguish simulated from real pain misdirects considerable resources not merely to ineffective management, but to the unjustified payment of pensions and compensation. The uncertainty repercusses to the disadvantage of those with real pain. It leads to an undesirable caution in the administration of pain relieving drugs to those who need it. The escalation of demand for analgesia by the person who has unrelieved pain so resembles the behaviour of the addict it earned the label of “iatrogenic pseudoaddiction” (Weissman & Haddox, 1989).

The techniques available for the relief of pain caused by undoubted physical disease continue to advance, improving the management of some groups such as those with cancer. In contrast, the reports of improvement in the treatment of chronic pain without recognisable cause are dubious. They contradict the extraordinary growth of this group and the intractable nature of their complaints. The studies have fatal flaws such as the failure to consider the selective factors of referral patterns and attrition, the use of inappropriate controls (Turk & Rudy, 1990) and the failure to control for the placebo effect (Ochoa, 1999).

The poor results should not surprise. The treatment methods have doubtful validity and for practical purposes produce no benefit. Across the board most published trials of treatment fail to meet the standards of adequate methodology (Bloch, 1987; Ochoa, 1999; Turk & Rudy, 1991). The claims about the value of early treatment and rehabilitation remain untested (Institute of Medicine, 1987) or disproven (Pennie & Agambar, 1990).

Surprisingly, standard methods as respectable as physiotherapy belong to the treatments which have no proven value (British Association of Physical Medicine, 1966; Brooks, 1987; Flor & Turk, 1984). Less surprisingly, the treatments for the fallacious notion of

sympathetically mediated pain provide no more benefit than placebo (Ochoa, 1999). The gate control theory has produced a technical application, transcutaneous electrical nerve stimulation (TENS). Although no more effective than a placebo (Deyo *et al*., 1990; Marchand *et al*., 1993) but certainly far more expensive, it enjoys widespread use. At least experience has established that surgery generally makes the complaint of pain worse.

REFERENCES

Refer to separate download, References-for-resource-texts.pdf.