+ Other symptoms, adverse effects of treatment

DEPRESSION TOTAL PAIN ANGER

Loss of role etc. Delay in diagnosis etc.
Insomnia
Fatigue
Helplessness ANXIETY
Disfigurement fear of pain/death
Worry re: finances/future

Nowadays, the so-called Biopsychosocial model has superceded the stimuluse-response theory.

Gatchel and Turk include affective (emotional) factors, personality, social and learning, cognitive(beliefs about pain) and self-efficiency as making up the pain experience.

An abbreviated chronic pain cycle might look like this:

PREMORBID PERSONALITY

ACUTE ILLNESS hospital; mild domestic crisis

Pain persists

Depression
Despair syndrome

multiple doctor

Large-scale
investigation

anger, distrust

Failure of
Alternative treatment, Surgery,
Medication
Physical
Seek alternative
Treatments
Psychosocial distress

deconditioning

We can learn more about pain by looking at its Evolution. This was discussed by Professor Broom who is an expert in animal behaviour.

Pain has an adaptive function, as it may result in relevant learning and can qualitatively change behaviour to facilitate objectives.

The advantage of experiencing pain is therefore the opportunity to change behaviour to avoid pain-anticipation, avoidance and behaviour modification are all evolutionary advantages.

As a subjective experience, pain is, of course, difficult to evaluate. Different animal species

respond differently to pain.

In animals, it is often necessary to observe abnormal behaviour in order to assess pain. Some species will vocalise about pain, which has the function of signalling to their group who may help.

Humans, other primates, dogs and pigs do this (although monkeys do not vocalise when giving birth, perhaps because this could attract the unwelcome attention of predators). Many animals do not vocalise in this way as perhaps this would be maladaptive.

Following on from this, we can look at pain in pre-term new-born babies (neonates). Professor Fitzgerald, an expert in developmental neurobiology, opened her talk ("Pain in the foetus and neonate") by saying that up until relatively recently, children in pain were grossly undertreated.

In 1976, Swafford and Allen stated "Paediatric patients seldom need medication for relief of pain. They tolerate discomfort well." This seems scarcely credible as a viewpoint in the latter part of the twentieth century!

In 1993, Southall et al looked at 181 outpatients who underwent an invasive procedure. 50 had no analgesia (pain relief) and 89 (unsurprisingly) showed signs of distress.

Cummings et al, more recently (1996), looked at 200 inpatients in Canada (which is known as the best country in the world for pain treatment) and found that many endured unacceptable levels of pain.

Thankfully, nowadays, the work of Professor Fitzgerald and others is changing this.
As the professor said in her talk, we will probably have to accept that we will never know how much pain very young children experience.

However, using behavioural patterns, one can discern how much analgesic to give and whether it is working.

Although blood levels of stress hormones have been taken, these do not always correlate well with the pain level. Neither do spinal reflexes, which are unconscious, protective reflex movements: so we do not know how much information is passed on to the cortex of the brain. Paraplegics will show a withdrawal reflex but feel nothing.

Pain perception, of course, occurs not at the site of the perceived pain, but in the brain, just as it is the brain that "sees" an image picked up by the retina of the eye.

It is worthwhile to look briefly at what we know about infant pain mechanisms as this may have some bearing on problems experienced later in life.

From what can be measured, it seems that infants have less selectivity in the information they pass on: they have a temporary wiring of touch neurones which does not persist. Sensory receptive fields in the CNS (central nervous system: brain and spinal cord) are much larger than in the adult.

Interestingly, these fields are similar to those seen in patients with central pain which has developed due to sensitisation of the CNS, sometimes after a discrete incident, and more commonly when there is unalleviated chronic pain.

One of the reasons that these sensory fields are larger is because, unlike in the adult, inhibitory messages from the brainstem are not modifying the sensory input.

This seems to develop slowly postnatally.

Preterm infants (and animals) have very little descending control. As cognitive and cortical aspects develop, reflexes tend to become dampened. It is possible that this is the reverse of the

CNS sensitisation (?wind-up') that can occur in chronic pain.

Infant pain reactions are more disorganised than those of an adult, less selective and with poorer spatial localisation; again, this is similar to allodynia seen in central pain.

This correlates with Professor Broom's assertion that there is an incorrect assumption that a greater cognitive (thinking) ability suggests that greater pain is felt: the more complex animal may in fact be better equipped to deal with pain than the less complex, by virtue of its ability to use cortical activity (thinking in humans) to dampen pain perception.

There is some evidence that very early pain experiences can result in longer-term consequences: changes in the nervous system. Animal models suggest that the plasticity (ability to change) of the nervous system can result in lifelong changes.

It is, however, unknown to what degree adult nervous system plasticity can be affected by the experience of pain. Certainly, the current notion of "wind-up" suggests that chronic pain can sensitise the nervous system and affect the way in which sensations other than pain are perceived.

Pain is seen as essentially a conscious sensation, however, even in the unconscious patient, there may be signs of physical consequences such as raised heart rate and blood pressure.

Professor Bond expressed the opinion that there may be damage due to chronic pain, as injury can lead to changes in the spinal cord and prolong the pain experience.

As stated before, pain is a subjective, conscious sensation, so it is obviously essential to look at which parts of the brain are involved.

Recently available imaging techniques such as PET and fMRI (functional MRI) scans have

shown that there is no single pain centre but rather several areas that are activated by pain. The thalamus, limbic system and various cortical areas (inferior parieto-temporal, prefrontal, insular and anterior cingulate) are all involved.

It is likely that NMDA involves the limbic system, which is also responsible for the link between sensation, emotions and the endocrine (hormonal) system.

The brain not only receives incoming information but also sends descending inhibitory messages to "dampen" the system.

In summary, pain is initiated by nociceptors which detect damage. The message is transmitted via numerous neurotransmitters along neurones to the central nervous system (CNS).

In the spinal cord, there may be a "sensitisation" which results in any descending control of pain (from the brain) being over-ridden. Pain is perceived in the brain and may affect a number of different parts of the brain, which has a bearing on other effects such as emotional and endocrine .