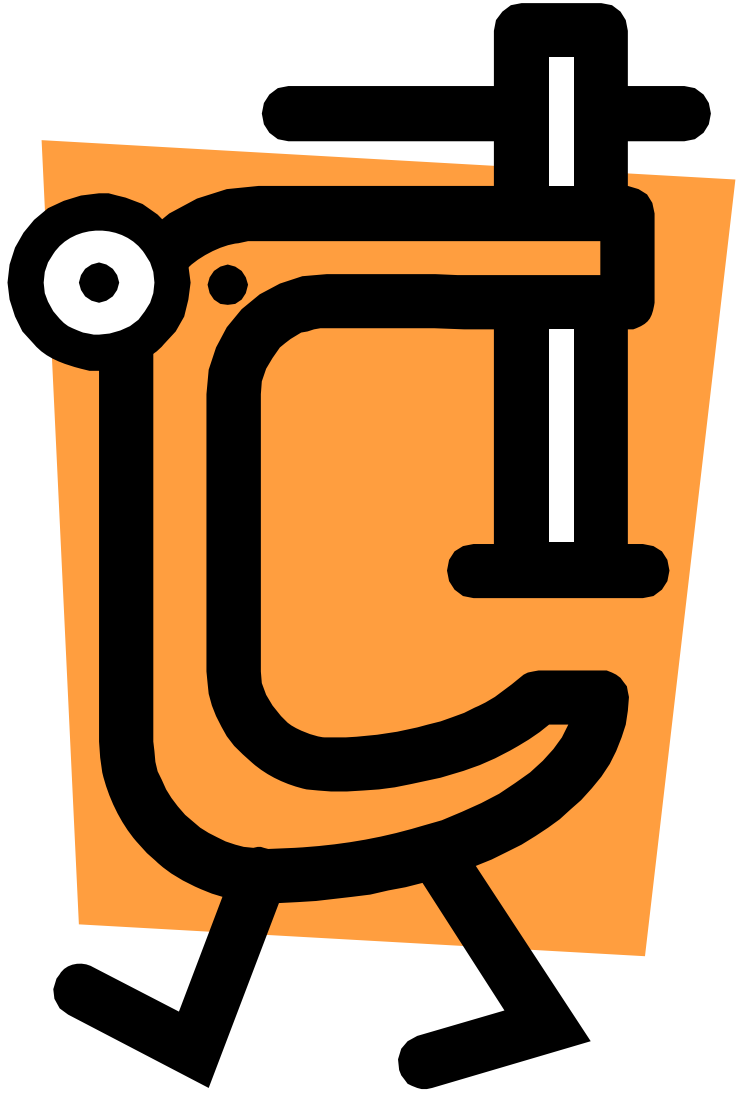


# The Chronic pain patient in acute pain in acute hospital

Dr Jane Trinca



# Nurses caring for patients with severe pain in an acute hospital

- 2009 Susan Slaytor
  - APS APRA PhD scholarship holder
  - Research nurse Sir Charles Gardiner in Perth
- Behaviour of nurse: balance of wellbeing for patient and wellbeing of nurse which effects practice
- Most nurses approach to Acute severe pain: medication, promote wellbeing, patient compliant.
- If patient non compliant or pain not controlled???

# Practice point 1

- When a nurse was unable to control patients pain and improve wellbeing over several hours, they often retreat because powerless

# Severe acute pain: uncontrolled

“ it was horrible

You don't know how to tackle the problem.

Its really a challenge to look after such patients....you get drained...you don't want to go there...in that room...You don't want to go because it, you come to a certain level when you can't take it...you know,...look at the patient's suffering”

From Slayter presentation APS 2009 quoting ward nurse

# Severe acute pain in chronic pain patient: Practice point 2

- Don't see distressed non functioning patient but ignore this as if patient not in pain
  - Less inclined to give medications
  - Go in to patient protection mode to avoid dependence/ addiction/
  - Are conscious of perceived manipulation

# Patient with history chronic LBP

- Bilateral # ankles
- “she actually would tell one of the nurses:  
“ I think I need my pain killer” She’s literally timing it. I really can’t see anyone who’s in pain and can be so rational as that....A woman whose not in pain and not doing her best to get to sleep”

From Slayter presentation APS 2009 quoting ward nurse

# Acute pain on Chronic in hospital setting

“They don’t show any symptoms of pain. They don’t show any grimacing, guarding, bead sweating; there’s no increase in pulse rate, resps and BP is fine..”I’m in pain” and they’re talking to Joe Blo on the phone “can I have my morphine?” ...Um “No”

# Chronic pain is not all the same

- Different causes in.....
  - Nerve injury
  - Ongoing inflammatory
  - Tumour
  - Generalized pain hyperalgesia
  - Localized pain hyperalgesia
  - Post surgery
  - Loss of body part
  - Gene mutation
  - De-conditioning...etc
- different individuals
  - Age
  - Genetics
  - Nutrition/substance
  - Co-morbid conditions
  - Medications
  - Personality factors
  - Coping strategies
  - Mood states
  - Beliefs/ cognitions/ fears
  - Previous pain problem

# But how about the social context?

- Work
- Family
- Friends
- Alone
- Compensation

# Previous learning and experiences

- Important determinant of chronic pain
- Aversive memories can enhance pain perception
- Chronic pain treatments (should involve)
  - Extinction of aversive memory traces
  - Relearning of positive associations

Herta Flor “Extinction of pain memories: Importance for treatment of Chronic pain” Proceedings IASP ASM Glasgow 2008

# Social influences

- Education standard
- Culture
- Type of family
- Disability support
- Anger/ sense of entitlement

# Current State

- General health
- Activity level
- Weather
- Distraction
- Attention
- Belief in a cure (placebo)
- Association with adverse/ good past experience
- Comfort and confidence in environment/  
caregiver

# And everyone will behave differently to pain

- All behaviour that results from impact of pain is “pain behaviour”
  - Screaming
  - Not screaming despite pain
  - Lying in bed
  - Getting up despite pain
  - Limping
  - Not limping despite pain
  - Seeing a doctor, taking pills and not doing so

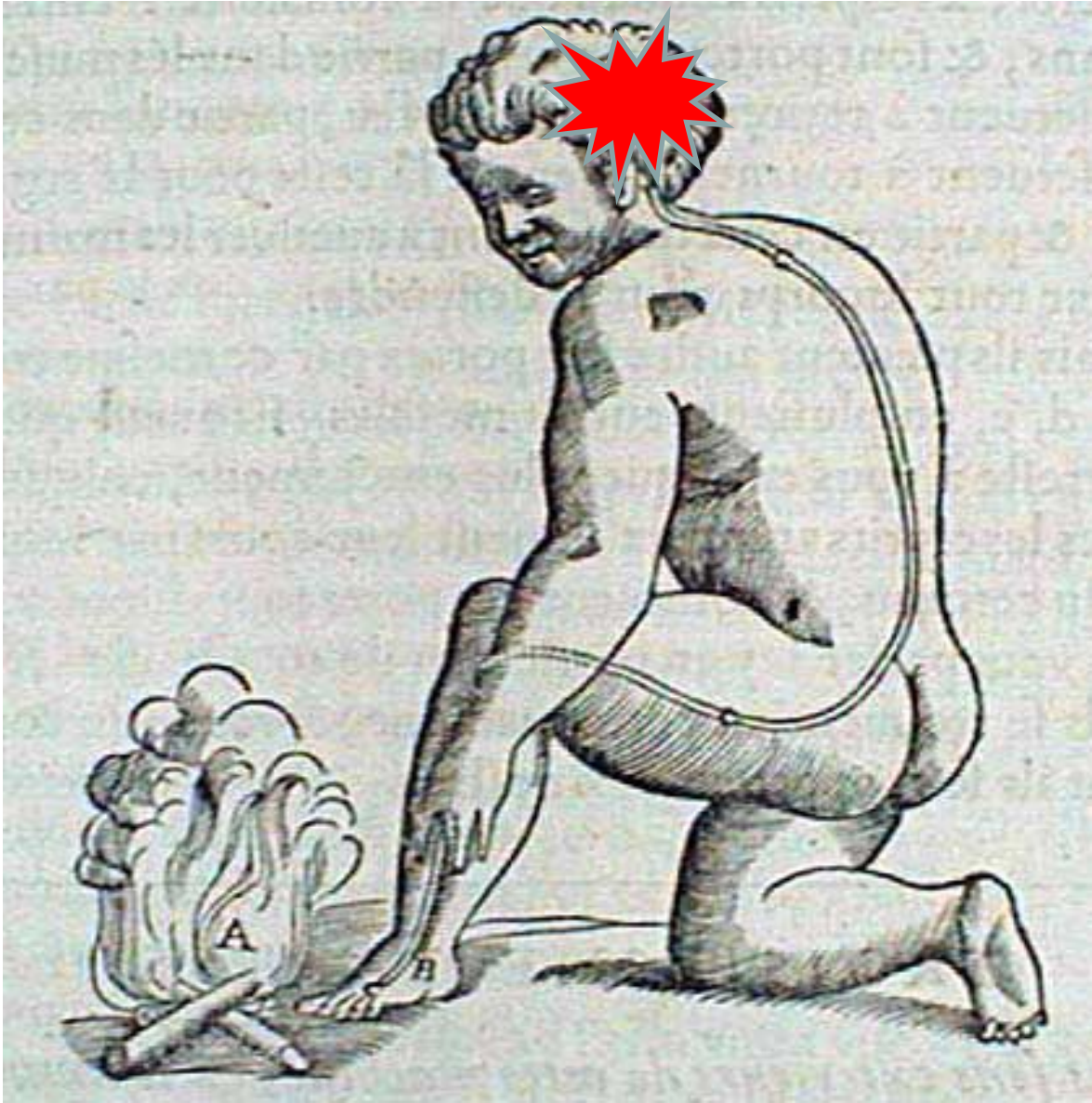
# Care givers will respond differently to pain behaviours

- Ignore
- Believe/ not believe
- Respond with medications/ support
- Etc.....

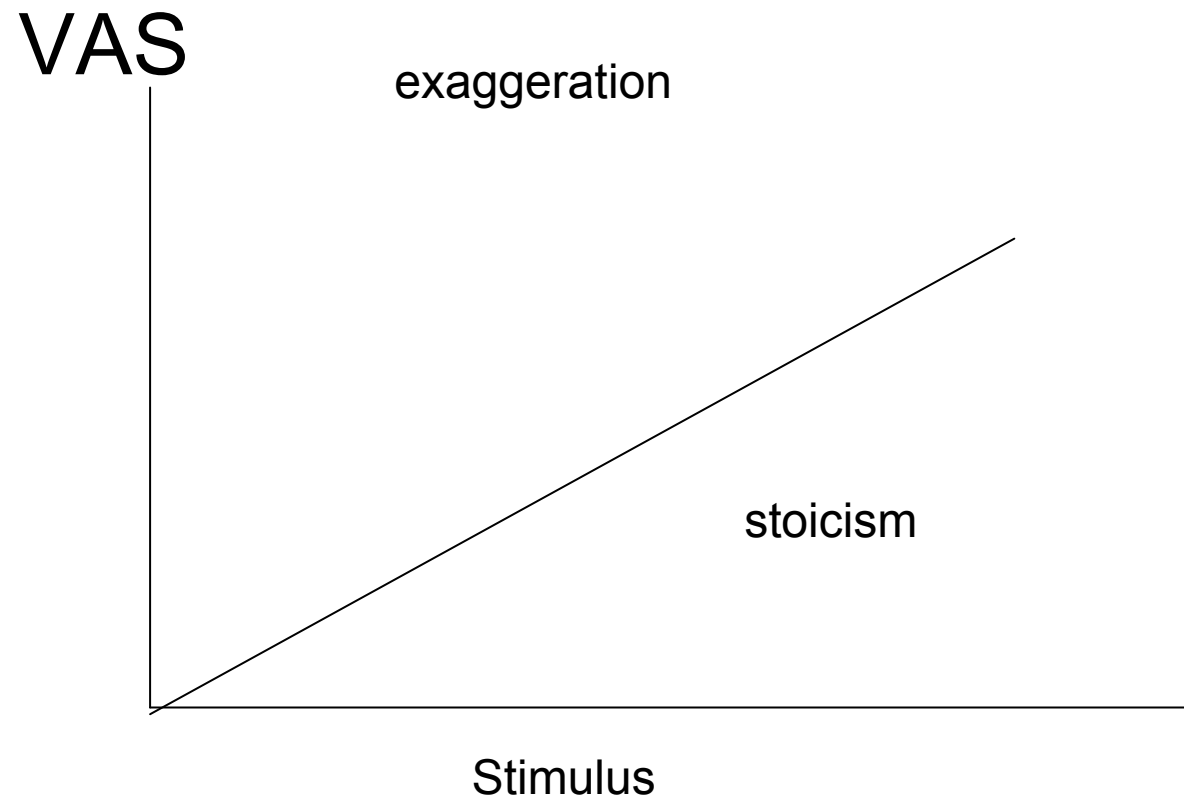
Which has impact on pain

# Therefore the consequences will be different:

- Job
- Family/ partners
- Benefits
- Finances
- Activity level
- Mood
- Self efficacy
- Medications/ surgery/ healthcare reliance
- Frustration, anger, response to healthcare workers



# Cartesian model pain



“Our tendency to invoke psychological explanation for situations that do not conform to Cartesian world view is a factor that has undoubtedly contributed to defensive reactions in many of our patients” Pain Management 2<sup>nd</sup> edn Main et al 2008

# Is pain a hard-wired sensation?

- “The classic teaching on pain had such enchanting simplicity that it was easy to learn and hard to forget. It therefore haunts us.” PATRICK WALL

# Traditional concepts

- Hardwired, modality specific single pathway from stimulus to sensation
  - High threshold nociceptors (Sherrington)
  - Low threshold mechanoreceptors (LTM)  
e.g. Touch  $A\beta$  – pain when sensitised
  - Dorsal horn cells that only respond to noxious stimuli (pain cells)
  - These cells are too slow to correlate with behavioural response in animal (C Fibres)

# Traditional concepts

- Spinothalamic tract to thalamus because analgesia produced by ventrolateral cordotomy
- Ignored fact that pain usually returns sometime after cordotomy
- This ignored the large number of spinal tracts that ended in brain stem

# Traditional concepts

- The Thalamus and cortex were chosen as site of pain sensation based on untested Victorian assumption that therein lay the basis of all sensation.
- However surgical lesions of forebrain to produce analgesia failed dismally

# **War casualties felt little pain. Why?**

- Beecher - distraction

**When we cut nerves to relieve pain,  
the pain returns.**

**Why?**

- Regrowth, sprouting in brain and at nerve endings
- Ectopic foci

# Why pain after thalamic stroke?

- Theory: Lack of inhibitory input

# Why pain after spinal cord injury?

- Lack of inhibition below lesion –
  - de-afferentation / phantom
- At level pain –
  - Neuropathic
- Often muscle pain at and above level of lesion

# Why do we feel pain in remote sites?

- Thoracic muscle injury in groin
- Adductor injury in vagina
- Back muscle injury in testis
- Diaphragm irritation in the shoulder tip
- Trigger points

# Why pain after shingles?

- Post herpetic neuralgia
- An example of neuropathic pain

# What causes profound pain and autonomic disturbance after some nerve injuries?

- CRPS
- Autonomic receptors growing on somatic nerves
- Central sensitization
- Neglect: “Lost limb syndrome” - cortical changes
- Dystonia – association pathways
- Oedema: neurogenic oedema, immune system

**Some individuals feel experimental pain more than others.**

**Why?**

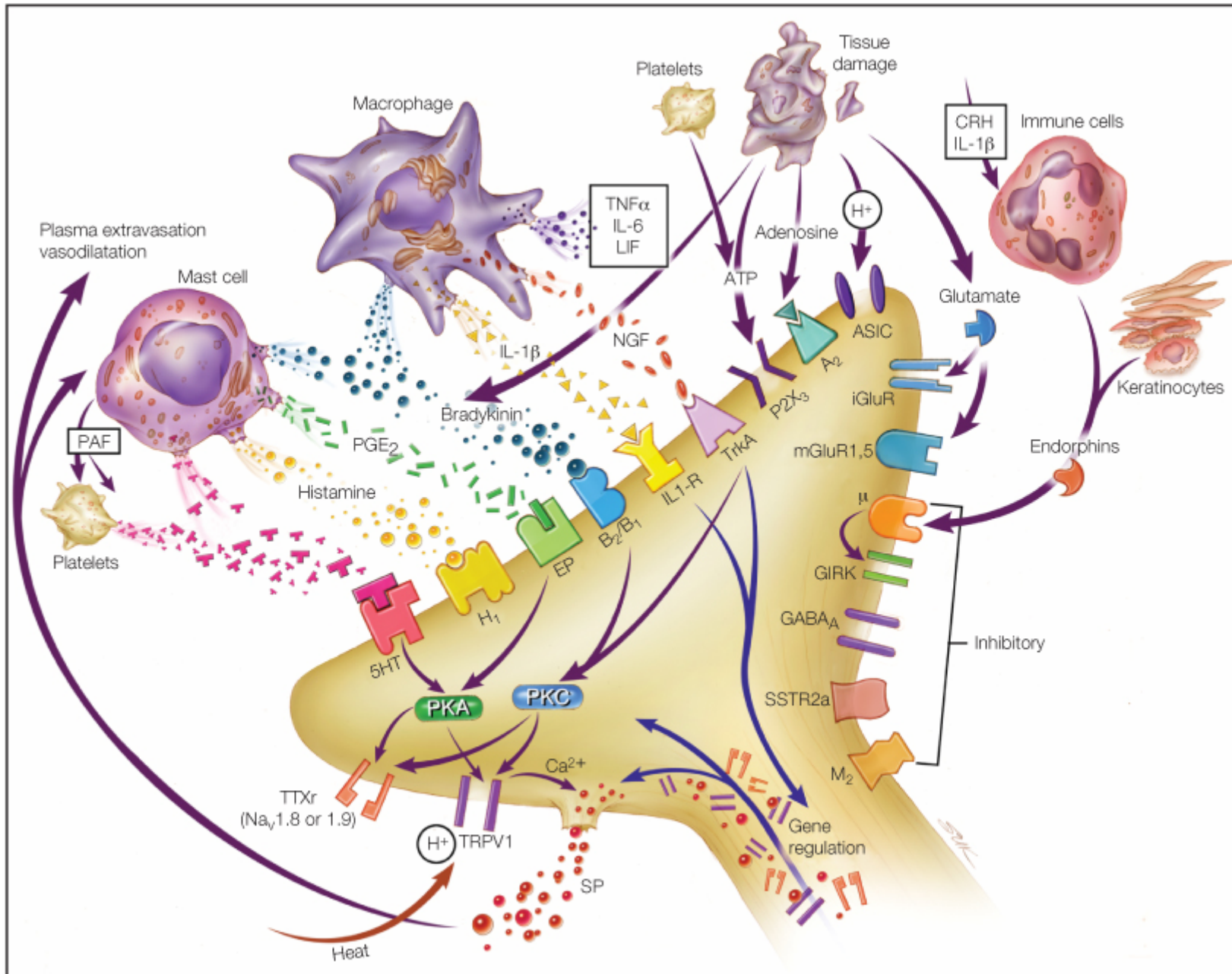
- Pain threshold difference
- Genetic
- Female – ?Oestrogens

# **Why does some pain not go away despite healing?**

- Continued nociceptive input
- Changes in spinal cord: Central sensitization
- Continued firing of nerve endings in periphery and cell body
- De-afferentation: lack of inhibition
- Secondary postural change & deconditioning

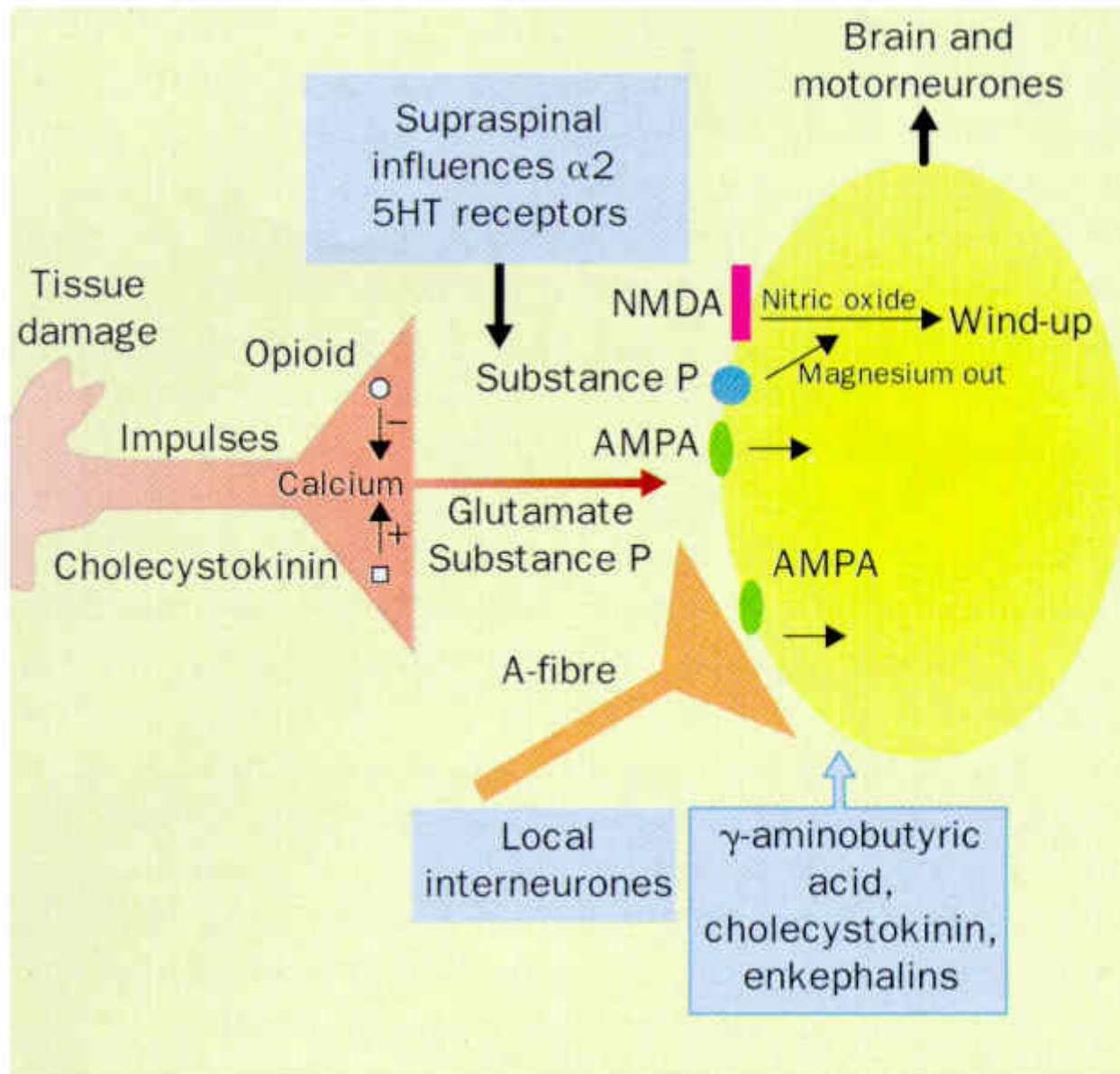
# Peripheral Sensitisation

- Following injury:
  - Lowering threshold of C fibres locally by biochemical soup – inflammatory mediators
  - Hyperalgesia
  - Widening of receptive field
  - Can facilitate central sensitisation



# Wind up

- Persistent, repetitive, intense stimulus
- Temporal Summation in SC leading to NMDA receptor activation – pain amplification
- Short lasting
- Precursor to CS



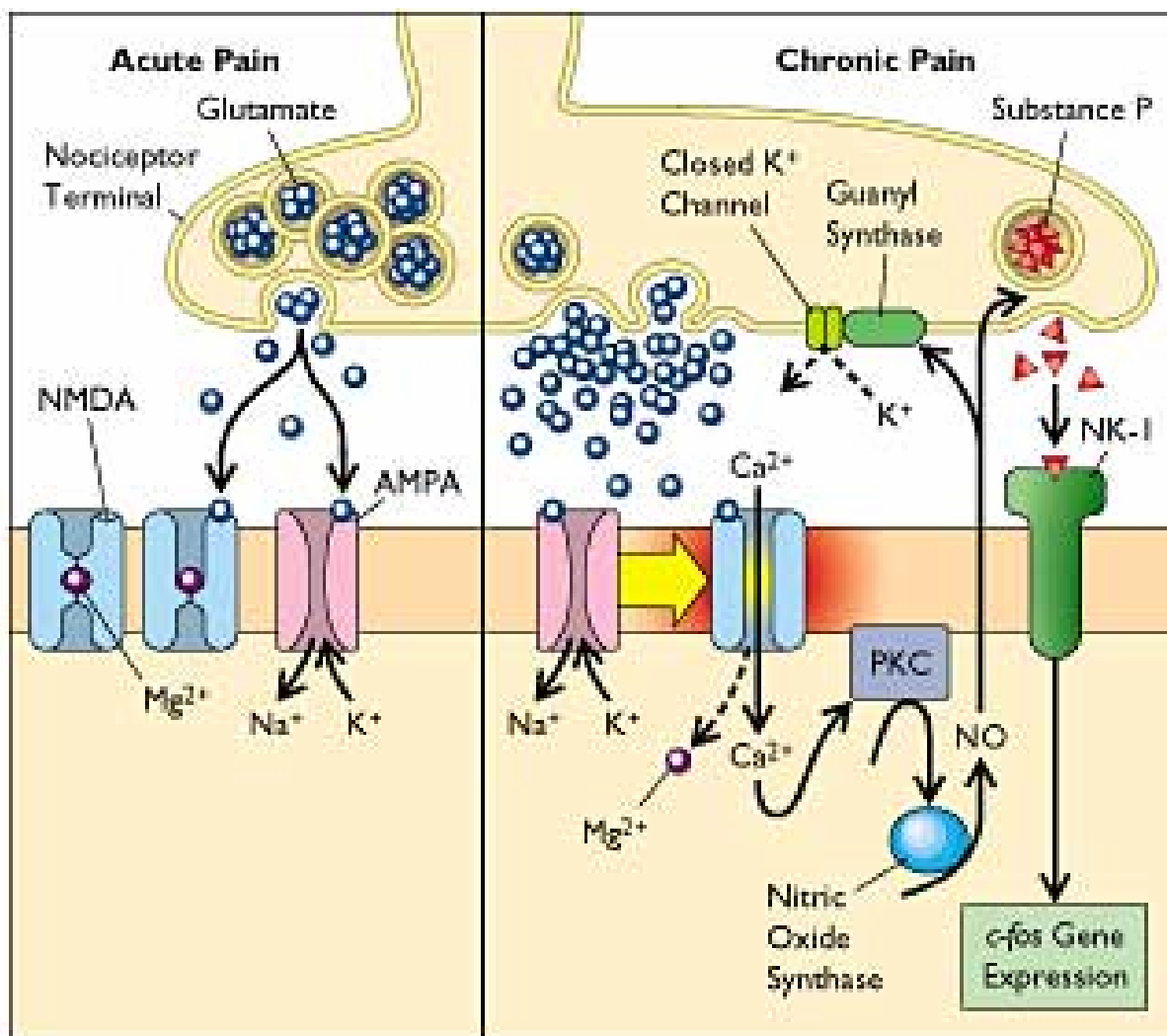


Illustration: Seward Hung

# Neuropathic pain & immune system

- Immune system involved in hyperalgesia
- Neutrophils macrophages recruited from circulation and enter damaged nerve
- Inflammatory mediators are produced which have toxic effects on nerves causing excitation, pain and damaged nerve/brain barrier

# Nociceptor

- Specific nerve that transmits noxious stimulation once the stimulus reaches a certain threshold
- Innocuous sensations like light touch are carried in non-nociceptor fibres  $A\beta$
- Question: What is this phenomenon called when light touch is painful?
- Why is light touch sometimes painful?

# GATE Theory

- Could the fast fibres travel to the spinal cord and block the slow fibres?
  - Could other areas of the brain and sensations from other body parts have an effect on this system as well?
- 
- Melzack & Wall 1962 Nature

# **Pain a balance of inhibition and excitation**

- E.g. Brachial plexus avulsion remove inhibitory pathway
- We would be in a state of constant pain without the sensation from the rest of our body inhibiting the pain system
- Inhibitory transmitters: endorphins, GABA

# **What happens when we electrically stimulate the PAG of spinal cord?**

- Analgesia

# Descending modulation

- Explains the effect of emotional state and attention on pain
- Nor adrenaline and serotonin involved in this pathway
- e.g. Endep (TCA), Cymbalta (SNRI), Clonidine ( $\alpha_2$  agonist)

# Gate Theory

- Yes MODULATION of the transmission of nociception occurs thru CNS
- Movement – inhibition
- Cognition
- Attention – focus/distraction
- Fear
- Anticipation
- Immune system

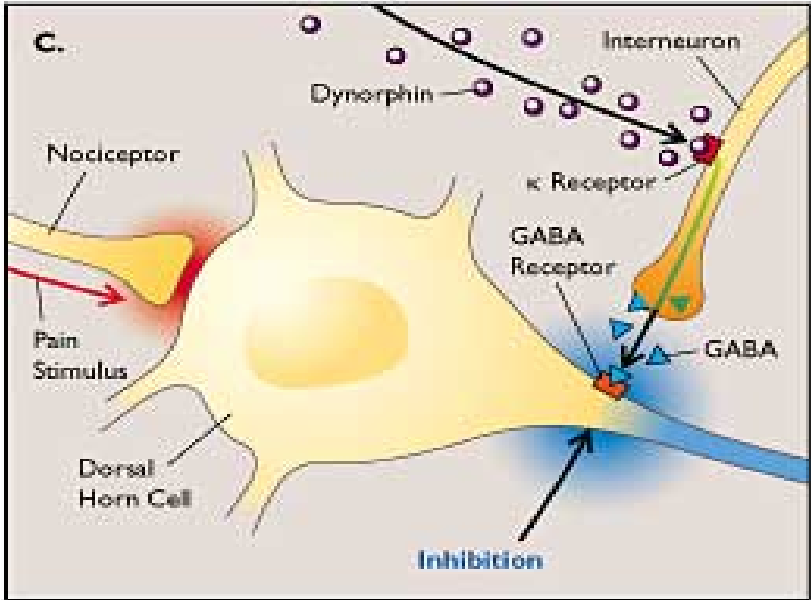
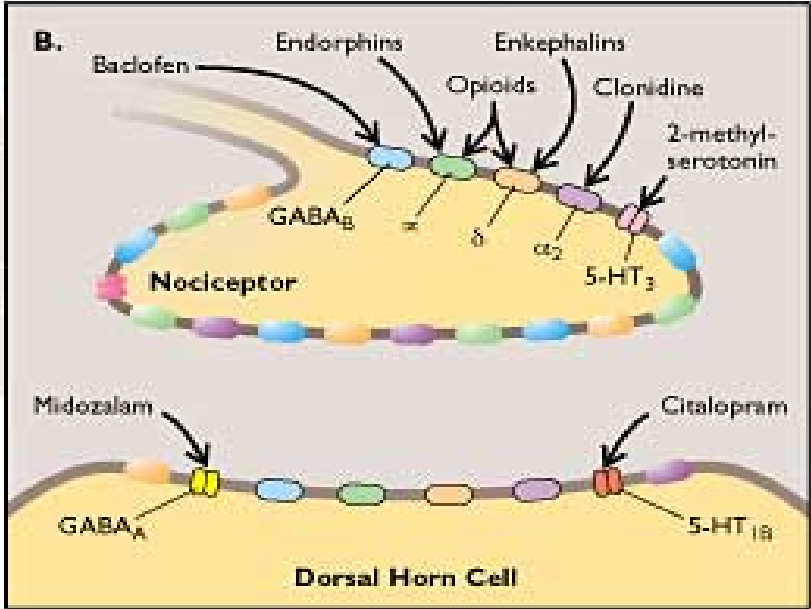


Illustration: Seward Hung

# More than the gate?

- The pain system is not hard wired it has
- **PLASTICITY**
  - Inflammation changes pain threshold of nociceptors.  
“Silent” receptors become active nociceptors
    - E.g. In bladder, 40% of nociceptors inactive until bladder becomes inflamed
  - Regrowth & sprouting of nerves periph/central
  - New connections - SC
  - New transmitters manufactured in nerve body and transmitted to nerve endings e.g. TRPV1 – increased pain receptors - bladder
  - Chemical structure of central nerves change: **NMDA** receptor change=influx  $\text{Ca}^{2+}$

# Central sensitization of pain system

- Described by Clifford Woolf
  - Upregulation of pain system with repeated stimulation
  - 1983
  - Published in Nature

# Neuromatrix Theory

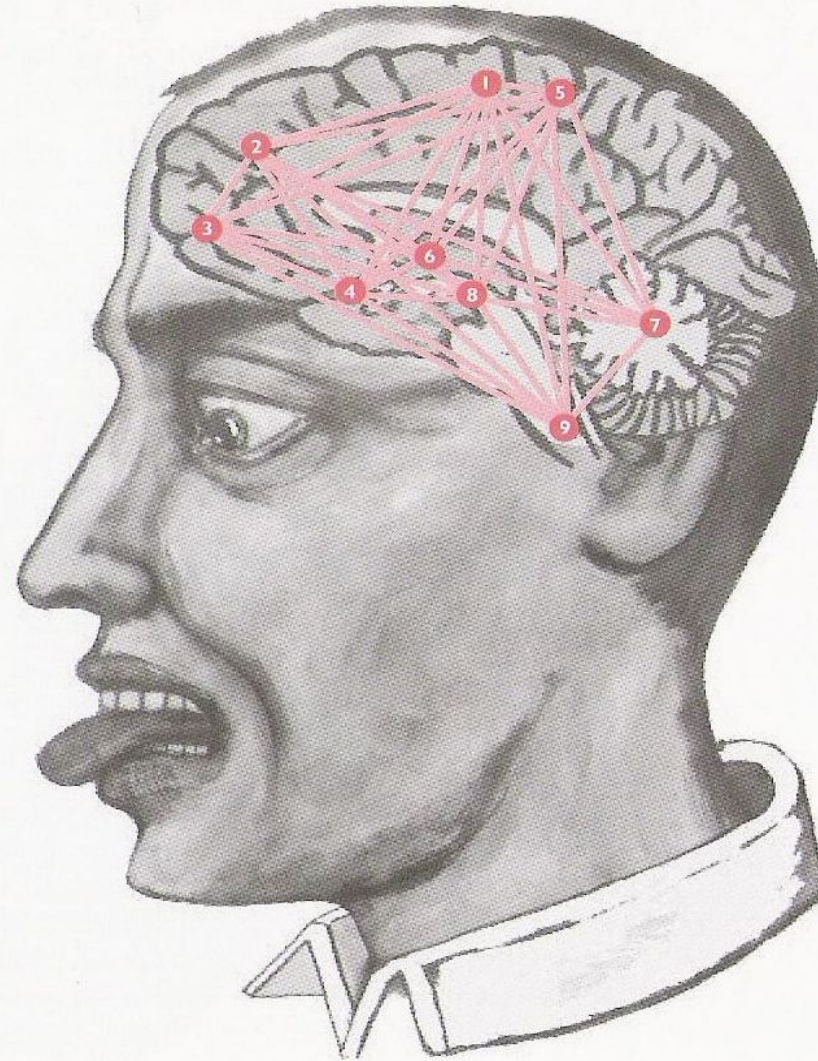
- In the brain
- The “body-self” neuromatrix theory of pain proposes:
  - that pain is a multidimensional experience
  - produced by characteristic "neurosignature" patterns of nerve impulses
  - modified by sensory experience
  - genetically determined
  - The neuromatrix is the primary mechanism that generates the neural pattern that produces pain

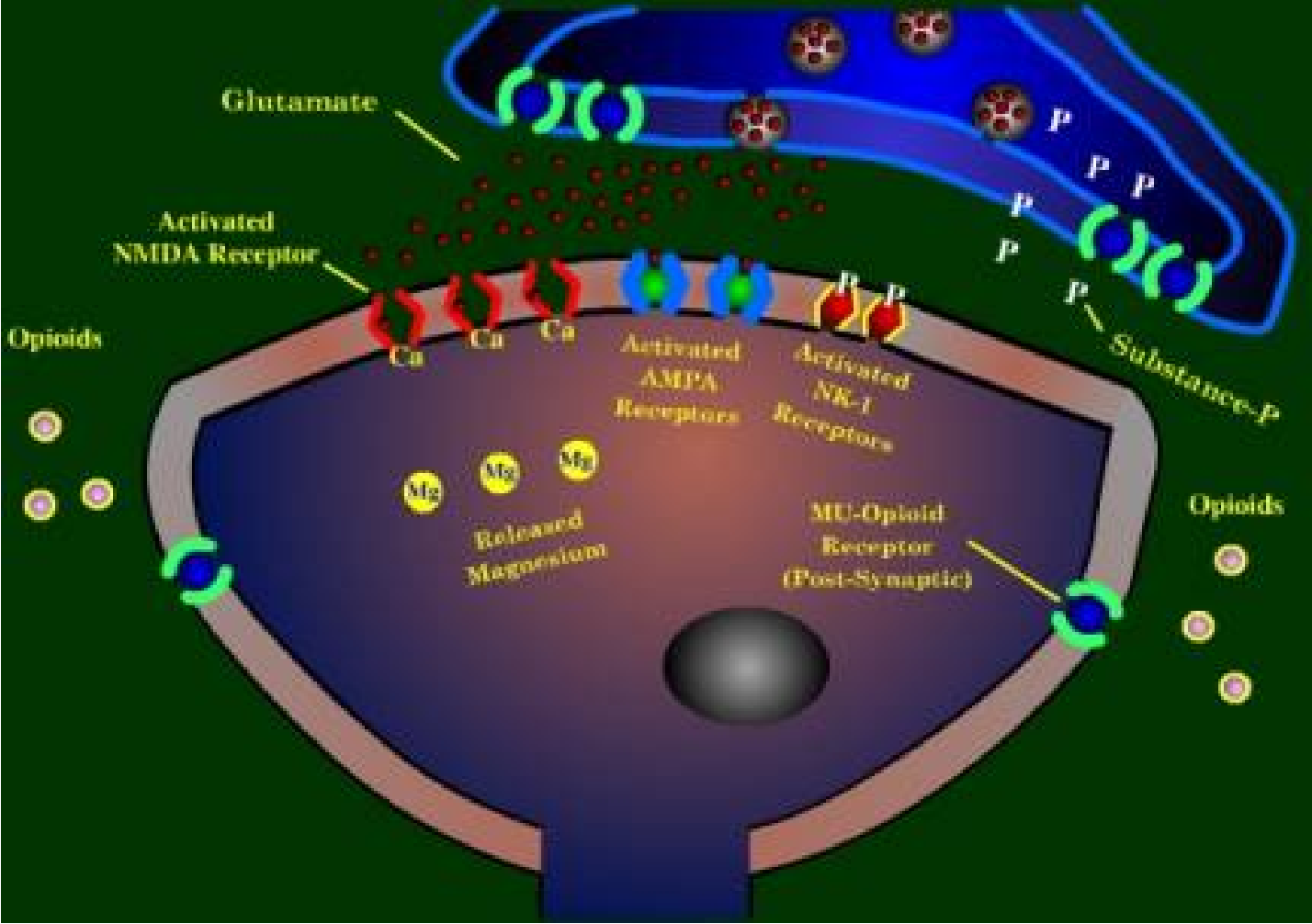
# Neuromatrix Theory

- These neurosignature patterns may be triggered by sensory inputs, but they may also be generated independently of them.
- It proposes that the output patterns of the body-self neuromatrix activate perceptual, homeostatic, and behavioural programs after injury, pathology, or chronic stress.
- Pain, then, is produced by the output of a widely distributed neural network in the brain rather than directly by sensory input evoked by injury, inflammation, or other pathology.
-

## A POSSIBLE PAIN NEUROTAG

1. **PREMOTOR / MOTOR CORTEX**  
*organise and prepare movements*
2. **CINGULATE CORTEX**  
*concentration, focussing*
3. **PREFRONTAL CORTEX**  
*problem solving, memory*
4. **AMYGDALA**  
*fear, fear conditioning, addiction*
5. **SENSORY CORTEX**  
*sensory discrimination*
6. **HYPOTHALAMUS / THALAMUS**  
*stress responses, autonomic regulation, motivation*
7. **CEREBELLUM**  
*movement and cognition*
8. **HIPPOCAMPUS**  
*memory, spacial cognition, fear conditioning*
9. **SPINAL CORD**  
*gating from the periphery*





# Chronic pain “a disease”

- 20% of population of Sydney has chronic pain (Blyth F Pain 2001)

# Prevalence Chronic pain in hospitalized patients

- 62% medical inpatients reported pain (non joint)
  - 36 %chronic regional
  - 21% chronic generalized
  - 5% transient

– *Seminars in Arthritis and Rheumatism*, Volume 30, Issue 6, 2001, Pages 411-417  
D.Buskila

# Identified risk factors in hospital patients (medical)

- 59% pain
- 26% severe
- Sickle cell most likely
- Weight
- Female
- > 65 years
- Education level higher than high school

– Chad et al, Arch Intern Med. 2004;164:175-180.

# Pain prevalence in German teaching hospital

- 33% persistent pain for more than 3 months

**Barbara Strohbuecker et al**  
May 2005.

# Prevalence chronic pain in hospital

- 53% chronic pain
  - 83% pain in last 3 months
  - 64% pain reason for admission
- 
- Clinical J of Pain 2009, 25 Gerberschagen et al

# Point prevalence study Austin health

- analgesic prescribing across the acute inpatients on single day excluding ICU/ emergency/ oncology
- 206/278 receiving paracetamol+/- opioid
- Further 46 receiving opioid without paracetamol
- 252/278 prescribed an analgesic in acute tertiary hospital: incidence of pain on this basis: 90%

# Typical chronic pain patients

- Treatment or cure unlikely
- Search for pathology often unfruitful
- Most patients just want to “get rid of pain” so that they can get on with life
- Multiple cures offered with often short term benefits only
- Search for abnormal test results often fruitless

# Chronic pain patients often

Believe that:

- surgery will remove pain source
- That they need to rest or avoid activities that cause pain as any increase in pain is associated with damage
- That medications are required
- That tests will find the cause

# Chronic pain patients often

- Have high disability
- Cycles of overdoing and under-doing
- Heightened responses to stressors
- Associated anxiety, depression, PTSD, anger frustration
- High use of health services and medications
- High level of associated social dysfunction

# Are some more prone to develop chronic pain?

- Similar TRAUMATIC/ INFECTIOUS INSULTS : Ongoing neuropathic pain in small %
- Experimental pain in healthy volunteers:
  - Large range pressure/ pain thresholds, pain tolerance to cold, large range VAS with thermal
  - fMRI of this group: areas in brain higher activation with higher VAS

# Incidence Prone surgical groups

Amputn	30-50%	Severe 5-10%
Breast	20-30%	5-10%
Thoractomy	30-40%	10%
Ing hernia	10%	2-4%
CABG	30-50%	5-10%
CS	10%	4%

# Prevalence of substance abuse in hospitals

- Patients who abuse opioids have a higher incidence of pain and a greater rate of hospitalization.
- In 2004, 38.7 million Americans were hospitalized.
- It is estimated that substance abuse affected up to a quarter of these patients, with rates as high as 40% to 60% for those admitted secondary to trauma.
- There has been an almost 4-fold increase in the incidence
- Patients involved with substance abuse are generally undertreated for pain. The tendency is to "use opioids sparingly (for pain), resulting in both poor pain management and withdrawal phenomena resulting in:
  - **Increased length of stay;**
  - **Frequent readmissions; and**
  - **Increased outpatient and emergency visits.**

# Differences in clinical pain

- Patients with following conditions show increased pain sensitivity compared to controls:
  - Fibromyalgia
  - Irritable bowel
  - Musculoskeletal pain
  - Headache
  - TMD
  - Interstitial cystitis
- Mediating factor may be DNIC

# DNIC

- Diffuse Noxious Inhibitory Controls
  - In normal controls application of an intense somatic noxious stimulus leads to generalized whole body analgesia
  - Mediated by serotonergic and nor-adrenergic descending pathways
  - DNIC is reduced in a large % pts with Fibromyalgia and irritable bowel syndrome

# DNIC

- Opioid system changed in chronic pain: higher levels of enkephalins
- Higher occupancy of opioid receptors and thus fewer binding sites on PET scanning
- Thus opioids not effective and larger doses required
- In Fibromyalgia reduced NE and Serotonin
  - Thus drugs that increase these are effective

# Other neurochemical difference CSF of Fibromyalgia and HA

- More pronociceptive
  - Substance p
  - NGF
  - BDNF
  - Glutamate
- Less anti-nociceptive
  - NE
  - Serotonin

# fMRI findings in FM patients

- Increased activity with stimuli including pain in many brain areas
  - Leftward shift in stimulus response
  - Increased gain or volume setting
  - Also found in LBP, IBS

# Variability

- Nature
- Nuture
- interaction

# Neurobiology pain system

- Previously thought 3 areas of brain involved in pain processing
  - Sensory discriminative
    - Primary and secondary somatosensory cortex, thalamus, posterior insula
  - Emotional
    - Ant cingulate, ant insula, amygdala
  - Cognitive
    - Pre frontal cortex and affective areas

# Now

- No areas of brain solely devoted to pain processing and many other areas activated with pain

# Psychological factors and MRI changes

- Levels of depression did not correlate with enhanced in sensory levels of pain on testing or MRI sensory areas of pain
- Levels of depression did correlate with affective processing areas of pain processing whereas
- Levels of clinical pain were associated with both sensory and affective areas
- Therefore may be independent and parallel systems process affective and sensory aspects of pain

# Does stress lead to pain and other symptoms in fibromyalgia

- Strong familial and strong genetic factors
- Other external factors known to trigger FM presumably in those genetically pre-disposed
  - Traumatic events eg war, terrorism
  - MCA
  - Childhood hospitalizn associated with MCA
  - Infections eg EB, Hep c Brucellosis
  - Systemic disease eg Rh Arth

# Psychological distress

- Not good predictor
- Number of somatic complaints mod predictor
- Chronic pain predicts psych distress
- Early life trauma risk factor for chronic pain
- Baseline function of the stress response eg HPA is predictor independent of distress and other psych factors

# Infection and FM

- Premorbid factors not relevant
- Number of somatic complaints in acute phase predictive
- Eg 12 % develop FM
- 10% gastroenteritis IBs
- And Baldder Infection results in interstitial cystitis in a % of women
- Some vaccines eg rubella clear assocn

# Summary

- Genetic predisposition
- With biological; difference with augmented central processing of pain or sensations
- When exposed to infection/ signif trauma
- Main predictor is multiple somatic complaints

# Morphine resistant pain??

- Works at  $\mu$  receptor in spinal cord and brain
- In tolerance and central sensitization the central cells have developed a life of their own which is unaltered by blocking opioid receptors
- After nerve injury there is decreased manufacture of opioid receptors

# Trends in management of chronic pain

- Medications:
  - NSAIDS for inflammation
  - Tri-cyclic antidepressants
  - Anticonvulsants, local anaesthetics
  - NMDA receptor antagonists
  - Opioids
- Nerve blocks/ destruction
- Electrical stimulation: SCS, TENS

# Trends in Pain Management

- Restore function
- Cognitive behavioural treatment
- Interdisciplinary management
- Measure outcome re mood, medication use, function, QoL

# **Planning & Delivering appropriate care to patients in chronic pain**

- Mechanism
- Measurement
  - Function
  - Mood
  - Cognitions
  - Coping strategies
- Previous treatments
- Involve other disciplines

# Planning

- Undergo any evidence based treatments
- Develop goals re treatments
- Education
- Measure outcome
- If no treatment possible or effective
  - Consider CBT or functional restoration using multiple techniques