Stress

Stress triggers a response from the body to maintain a steady state, Claude Bernard (studied pancreas and liver) “Fixity of the internal milieu”

Stress is a known trigger of disease

**External environment**: everything outside of the body, shares information with internal mechanisms to maintain homeostasis

**Cells-organ systems are subsystems to maintain "steady. State" homeostasis**

Stress reactions
"compensatory mechanisms" can be good or bad

**Bad**: dysfunctional / maladaptive "HF - retain sodium and H2O"

**Good**: Breathing with exercise to blow off C02 to maintain proper PH balance

MALADAPTIVE RESPONSE TO STRESS
{Stress is a known disease trigger}
A chronic, recurrent response to stress that does not support adaption.

**Faulty appraisals**: Blowing something out of proportion can cause an issue to become worse {thinking a headache is a migraine all the time}

**Inappropriate coping**: "dysfunctional" over eating, screaming, distancing, "non compliant diabetic”, are all negative responses and considered inappropriate coping.

**Maladaptive Diseases** (Seyle's 1976): Hypertension, cardiovascular disease, renal disease, rheumatoid arthritis, allergic disease, nervous and mental disorders, sexual dysfunction, digestive/metabolic disease, and cancer (all triggered by stress)

**TYPES OF STRESSORS**

A stressor is a change that provokes a stimulus
The goal is to adapt to stress to maintain homeostasis

{ Any state produced by the environment, change that is perceived as challenging, damaging, or threatening to the equilibrium }

Physical: Cold, heat, chemical agents

Physiologic: Pain, Fatigue, sleep disturbance, insomnia } a protective mechanism , initiates the fight or flight response. The body releases serotonin and norepinephrine as well as activates neurological mechanisms "general adaption system"

{ local adaption syndrome: cut or small injury inflammation response"

Psychosocial: Fear, normal life transitions { marriage, baby, nursing school }

Nursing assessment: Pt may have high bp, palpitations, diarrhea, and/or emotional response

SELYES GENERAL ADAPTION THEORY

Emergency, short term changes, rapid and self limited
3 PHASES

1. ALARM: Fight or flight response catchecheolamines ( epi & norepinephrine ) are released from the adrenal medulla. Cortisol "stress hormone" is released from the adrenal cortex. Glucose is released from the liver, pupils dilate, mental sharpness increases, peripheral vasoconstriction to prefuse blood to vital organs. Increase HR, BP, BREATHING, & STRENGTH

2. Resistance: Adaption occurs to the stressor, cortisol is still increasing in relation to long term stress, constant increase in HR, BP, BLOOD GLUCOSE. Decrease in immune system. To decrease pain and inflammation

3. Exhaustion: Endocrine activity continues, body will fail exposure to stressor is prolonged; outcome is either disease or death depending on where you start.

HOW STRESS AFFECTS THE BODY REVIEW

CARDIAC : Increase BP, blood vessels become more rigid causing the heart to work harder, HF and Cardiomyopathy.
**HTN:** Vascular disease, blood slams against the vessel walls causing injury, thus allowing bad lipids to attach

**METABOLISM:** Increased glucose irregularities

**REPRODUCTIVE:** Inhibits reproductive hormones and causes ED in men

**IMMUNE:** Too much inflammation/ immune suppression (lupus)

**VASCULAR:** Causes vasoconstriction hormones to be released and decreases blood supply

**CHOLESTEROL LEVELS:** Increases cholesterol from the Cortisol

**CORTISOL LEVELS:** Increase to physical or emotional stress, steroid hormone rises with. Cholesterol levels. A serum level or 24 hour urine specimen can be taken to measure levels. Cholesterol is lowest in the night and highest in the morning.
PAIN MANAGEMENT

**Pain**: the 5th vital sign; an unpleasant sensory, emotional experience with potential or actual tissue damage and is the most common reason to seek medical care.

The joint commission states that pain is assessed in all patients and all patients have the rights to appropriate assessment and treatment of their pain. Pain is whatever the patient states that it is. You as the nurse do not decide the pain level of a patient.

**Pain is categorized in 3 ways**
1. **Duration**: how long have they had the pain
2. **Location**: where is it starting and where does it stop
3. **Etiology**: what's causing the pain?

**TYPES OF PAIN**

*Nociceptive pain* { normal transmission of pain}
"pain chain"

**Transduction** { touching something hot}
- bradykinin, substance p, and prostaglandins are released to transmit the feeling of pain to the ascending pathways.

**Transmission**
- The message is sent to the brain

**Perception**
- the brain perceives pain message

**Modulation** { response to pain}
- what the body does in reaction to the painful stimulus { pull hand back}

*Neuropathic Pain* { abnormal process of painful stimuli, doesn't have to involve tissue damage}
Most commonly seen in diabetics

**Peripheral mechanisms:**
- **neuroplasticity**: damaged nerve reorganization
- **Peripheral Sensitization**: increased response to stimuli increased sensitivity

**Central mechanisms:**
- **Central Sensitization**: Increased excitability in the spinal cord
- **Allodynia**: pain from a stimulus that normally doesn’t cause pain
PATHOPHYSIOLOGY OF PAIN

**Nociceptors**: pain receptors on nerve endings that respond selectively to painful stimulus

**Nociception** is the transmission of pain

**Chemical substances**: Some increase sensitivity to pain while others decrease it.

**Increase sensation of pain**: prostaglandins initiate an inflammatory response, they get broken down to archidonic acid. Cyclo-oxygenase become prostaglandins. Cox 1 and cox 2 are iso enzymes of cyclo-oxygenase.

**COX 1**: Present in most body tissue, mediates prostaglandin formation, platelet formation, provides gut protection from ulcers. By inhibiting COX-1 the patient is at risk for GI bleeding and Kidney dysfunction. 2nd leading cause of peptic ulcers.

**COX-2**: present in inflammation, pain and fever. Inhibition of COX-2 will reduce the symptoms of fever, inflammation and pain ( inhibits substance p )

**Decrease sensation of pain**: Enprohpins and enkephalins. Act as endogenous opioids to suppress pain.

**NSAIDs**:
- Can be selective or non selective
- Non selective binds to both COX-1/COX-2: ibuprofen, ketoralac, naperson, aspirin
- Celebrex is a selective NSAID that binds to COX-2 only.
- You can take NSAIDS w/ Tylenol
- Tylenol( non salicylate) and aspirin compete for binding sites
- When giving Tylenol make sure other analgesics ordered do not contain Tylenol to the point where it can overdose the patient.

**GATE CONTROL THEORY**
The first theory to suggest the physiological factors influence pain

3 systems transmit nerve impulses that are influenced by a gating mechanism that is influenced by nerve impulses that defend from the brain.
1. Substantia gangliosa in the dorsal horn
2. Dorsal column fibers
3. Central transmission cells

**Stimulation of C fibers ( larger fibers) closes the gate; massage, visual imagery, distraction, placebo**
These will have an excitatory effect

**Stimulation of the A-DELTA fibers causes the gate to open**
These will have an inhibitory effect
FACTORS THAT INFLUENCE THE PAIN RESPONSE

1. Past experience
2. Anxiety: decreases the pain threshold
3. Depression: decreases the pain threshold
4. Age
5. Gender

**Culture: cultural factors must be taken into account when treating pain.** Early in childhood people learn from those around them the appropriate response to pain. The child also learns what stimuli are expected to be painful. Some patients may feel powerless if they feel their clinicians do not appreciate the magnitude their pain. Some may avoid reporting pain, grimacing, moaning, crying or seeking relief of pain. The nurse must react to the perception of pain and not the behavior.

6. **Categories of pain**

**ACUTE PAIN**
- Sudden onset of pain and specific to injury
- Teaches you to stay away from potentially harmful/dangerous situations
- Usually will decrease as healing occurs
- Lasts from seconds to 6 months although most acute injuries heal within a few weeks
- If pain persists past healing time allotted the pain will be considered chronic

**Chronic pain (non malignant)**
- Persistent intermittent pain that lasts beyond healing time
- Seldom attributed to a specific cause
- Poorly defined onset, hard to treat because origin is usually unknown
- Lasts 6 months or longer
- If chronic pain continues it may become the patient's primary disorder

**Cancer Related Pain (malignant)**
- Can be acute or chronic
- Pain is the most feared outcome of cancer
- Can be a result of the cancer or the treatment, most pain is a direct result of tumor involvement.
Harmful Effects of Pain

- If inadequately treated pain can have harmful effects such as: Sleep alteration (insomnia), fatigue, depression, and psychological implications

Effects of Acute Pain

- Pulmonary(atélectasies) cough, sputum and risk for lung infection, cardiac, endocrine, and immune systems can all be affected
- Stress response usually causes
  I. Increased cardiac output and work load
  II. Impaired insulin response (hyperglycemia) high blood glucose
  III. Increase cortisol production, immune suppression
  IV. Increase fluid retention (hypervolemia, cushingoid state) causing increased risks for patients with CHF AND Diabetes, and pulmonary edema.

- Stress response may also increase the physiologic response (MI, Pulmonary infection, thromboembolism, and prolong paralytic ileus and increase metabolic rate as well as cardiac output

- May hamper healing time in debilitated or critical patients
- Effective pain relief prompts healing

Effects of Chronic Pain (longer than 6 months)

- Suppression of immune system function from chronic pain may lead to tumor growth
- Depression, disability, fatigue, anger, and inability to perform ADLs
- It is safe to gradually increase opioid dose for progressive chronic pain
- Educate patients on pain management and resources
- Most common is lower back pain

CHARACTERISTICS OF PAIN

1. **Intensity**: Pain scale 0-10/1-10
2. **Timing**: When did the pain start? What was going on when it started
3. **Location**: Where is the pain, does it radiate
4. **Quality**: Is it dull, sharp, burning?
5. **Aggravating and Alleviating Factors**: What helps it, what makes it worse?
Physiologic Basis for Pharmacological Pain Relief

- **Opioid analgesics** act on MU and Kappa receptors in the CNS to inhibit the activity of ascending nociceptive pathways.
- **NSAIDs** block COX-1/2
- **Local anesthetics** block nerve conduction when applied to nerve fibers (dental work)

**Opioid Tolerance and Addiction**

- MAX safe opioid dosage must be individually addressed, every patient is different.
- Tolerance develops in all patients who take opioids for prolonged periods (as little as a few days) increased amounts may be needed.
- Dependence occurs with tolerance, withdrawal symptoms may occur when opioid use is discontinued.
- **Addiction**: not common from therapeutic opioid use.
- **Pseudoaddiction**: occurs when you haven't properly controlled the pain, increase dose and frequency.
- **Opioid induced hyperalgesia**: paradoxical effect, increased sensitivity to pain when given opioids.

**Pain Relief Interventions [Pharmacological]**

- **Multi-Modal** "balanced analgesia": use of more than one drug to cause pain relief.
- **PRN Medication**: Given as needed for breakthrough pain.
- **Routine Administration**: Given around the clock or a preventative approach (persistent pain is pain that lasts longer than 12 hours in a 24-hour period). (post surgery its ok to wake the PT up to give pain meds to prevent severe pain)
- **PCA** 'Patient controlled analgesia': Only the patient can push the button, set up so that the PT can't overdose.
- **Local anesthesia**: Shot given to numb a specific area.
- **Topical/patches**: Cream diclofenac is used for arthritis, lidocaine patches can be used for postherpetic neuralgia (shingles). Transdermal patches are absorbed systemically. You must clean the area and remove the old patch before administering next dose usually 72 hours, place old patch folded and in the sharps container. Lidocaine patched should be given 12 hours on and 12 hours off.
- **Intraspinal administration**: Epidural is given in the epidural space of the spine. Intrathecal is given in the subarachnoid space. When giving intraspinal medications the patient is at an increased risks of spinal fluid loss and spinal headache, to relieve spinal headache lay the patient flat on their back, some Dr.'s will order a blood patch. Push fluids.
- **PERINEURIAL ANESTHESIA**: A continuous infusion into the pained area, decreases the need for other pain medications.
- **Peak Respiratory depression**: usually 6-12 hours after administration up to 24 hours.
NONOPIOID ANALGESIC AGENTS

NSAIDS:
1. ketoralac " Toradol"
2. Sprix: nasal prep
3. Calador: Cox 2 inhibitor , Ibprofen, given P.O or I.M but still can cause GI ulcers

Acetaminophen
Can cause Hepatotoxicity, can be given with an NSAID , given oral, or rectal, the max daily recommended dose is 3000 mg / day { adult}

Ofirmez: IV version and is a newly approved short term piggy back

{ You can give a pain pill in the rectum as along as the patient does not have decreased neutrophils, platelets, perianal abcess, or rectal surgery, or low blood pressure or diarrhea. }

OPIOID ANALGESIC AGENTS

TRAMADOL " ULTRAM": Blocks the reuptake of serotonin and norepinephrine

Tapenatadol: Blocks the reuptake of norepinephrine

Tylenol 3

Meperidine " Demerol"

Propoxyphene " darvon"

Propoxyphene with Tylenol " Darvocet"

LIVER AND KIDNEY DISEASE: Increase hepatotoxicity risk

Meperidine: Rapidly accumulates to toxic levels, constantly check labs

Hypothyroid: A decreased metabolism will increase the effects of the drug because it isn't being metabolized as quickly.

Hyperthyroid: and increased metabolism of the drug will decrease its therapeutic effects/ window

Decreased Respiratory Resverve: can cause further respiratory depression

Dehydration: Increased risk of orthostatic hypotension

MAOI/PHENOTHIAZINE/TRICYCLIC ANTIDEPRESSANTS: If taken with an opiate you could have an increased opioid response. ( phenagren is a phenothiazide)
**Adjuvant Analgesic Agents**

**Local anesthetics:** can cause toxicity "ringing in the ears, metallic taste, seizures, cardiotoxicity "dysrhythmia" as characterized by circumoral tingling and numbness, bradycardia, CV collapse, mouth tingling, allergies are rare.

**Anticonvulsants:** Gabapentin, lyrica

**Antidepressants:** Tricyclics can cause orthostatic hypotension and cardiotoxicity

**Ketamine:** "special K" used when children need stitches, the person is there but it seems like nobody's home, common street drug due to the effects like disassociation and hallucinations.

**Placebo:** the person thinks they are getting the drug, but actually getting something else

**ADVERSE EFFECTS OF ANALGESIC AGENTS**
1. **RESPIRATORY DEPRESSION:** the antagonist is narcan/ can cause withdrawal s/sx
2. **SEDATION**
3. **NAUSEA/ VOMITING:** morphine is usually given with zofran
4. **CONSTIPATION**
5. **PURITIS:** considered an adverse effect unless accompanied by angioedema

**GERONTOLOGIC CONSIDERATIONS**

- They are more likely to have adverse effects of drug interactions
- More sensitive to drugs that produce CNS sedation, usually start with 1/4-1/2 the normal dose
- Increased risk of NSAID-induced GI toxicity, a COX-2 is usually prescribed with a proton pump inhibitor of these patients to prevent Peptic ulcer
- May need to have more time in between doses due to decreased excretion and metabolism
- *They are at an increased risk for heart attack, stroke, heart failure, high BP, destabilization of BP due to NSAID interference.*

*If they are diabetic or have decreased renal fx or decreased perfusion do not give NSAID, If they ask educate them to increase fluids*
Nonpharmacologic Interventions

- Cutaneous stimulation: "close the gate"
- massage
- Thermal therapies: heat or cold, assess the skin before and after application { never but heat on acute injury }
- TENS Machine
- Contralateral stimulation: stimulation of opposite side of effected area of injury

Distraction

Relaxation techniques: breathing, lamaze for pregnancy

Guided imagery

Hypnosis

Music therapy

Alternative therapies: acupuncture

Nursing interventions: cold wash cloth, repositioning, pillow under the head, back massage.