put together by Alex Yartsev: Sorry if i used your images or data and forgot to reference you. Tell me who you are.
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Vasovagal Syncope and Depression

History of Presenting Illness

The patient will be complaining of **having fallen unconscious**.
On-lookers will report a flaccid drop to the ground, **possibly preceded by an emotional event**, and possibly **followed by some twitching** (though not epileptiform convulsions)

Differential Diagnoses

- **Orthostatic syncope**
- **Cardiac syncope (AF, VF, Asystole)**
- **Stroke**
- **Seizure**
- **Aneurysm**
- **Hypoglycaemia**
- **Drug toxicity**
- **DVT and/or pulmonary embolus**
- **Transient ischaemic attack**

!! RED FLAG SYMPTOMS !!

preceding or following episode

- **chest pain**
- **dyspnea**
- **low back pain**
- **palpitations**
- **severe headache**
- **focal neuro deficits**
- **diplopia**
- **ataxia**
- **dysarthria**

WAS THERE DOPYNESS AND CONFUSION AFTER THE EVENT? YES?? THEN IT WAS A !! SEIZURE !!

Pertinent Findings on History

!! OBTAIN EXACT HISTORY OF THE EVENT !!
including reports of bystanders

- Precipitated by **emotional event** → vasovagal
- Precipitated by **straining** (eg. defecation etc) → vascular causes eg. stroke
- Precipitated by **sleep or food deprivation, hot environment, alcohol consumption, and pain.**
- Prior faintness, dizziness, or light-headedness occurs in 70% of patients
- Other symptoms, such as vertigo, weakness, diaphoresis, epigastric discomfort, nausea, blurred or faded vision, pallor, or paresthesias, also may occur in the presyncopal period.
- Symptoms of nausea or diaphoresis prior to the event may suggest syncope rather than seizure when the episode was not witnessed.
- The duration of symptoms preceding a syncopal episode has been reported to be an average of 2.5 minutes in vasovagal syncope and an average of 3 seconds in cardiac syncope.
- Syncope is associated with patient estimates ranging from seconds up to 1 minute
- Postictal phase? were they confused afterwards? (yes = seizure)
- Right after getting up quickly? = orthostatic hypotension
- ASK ABOUT MEDICATIONS eg. antihypertensives = these can cause orthostatic hypotension

Pertinent Findings on Examination

- **Blood pressure (orthostatic drop?)**
- **Mental state (still confused? Stroke-ish deficits?)**
- **Neuro exam (focal neuro deficits?)**
- **Heart examination (abnormal sounds, particularly AF?)**
- **Carotid Bruits**
- **May consider hematological exam if pt. looks anaemic**

Tests and Investigations

- **FBC** to look for ANAEMIA
- **SERUM BIOCHEMISTRY** to look for electrolyte abnormalities eg. hyponatremia
- **ECG** to assess heart function

How is this diagnosis made ?

→ By exclusion of anything more sinister

Disease Definition

Syncope (or fainting) is triggered by an abrupt decrease in arterial blood pressure, which in turn may result from a variety of causes. The most common cause (vasovagal syndrome) is associated with changes in sympathetic and cardiac vagal activity.

Management: Treat the cause!!

Eg. antidepressants, cognitive behaviour therapy, etc. …Assuming its vasovagal syncope.
ACUTELY, put the patients feet up. No other management is necessary

Prognosis

- Vasovagal syncope carries a uniformly excellent prognosis.
- This condition does not increase mortality rate, and recurrences are infrequent.

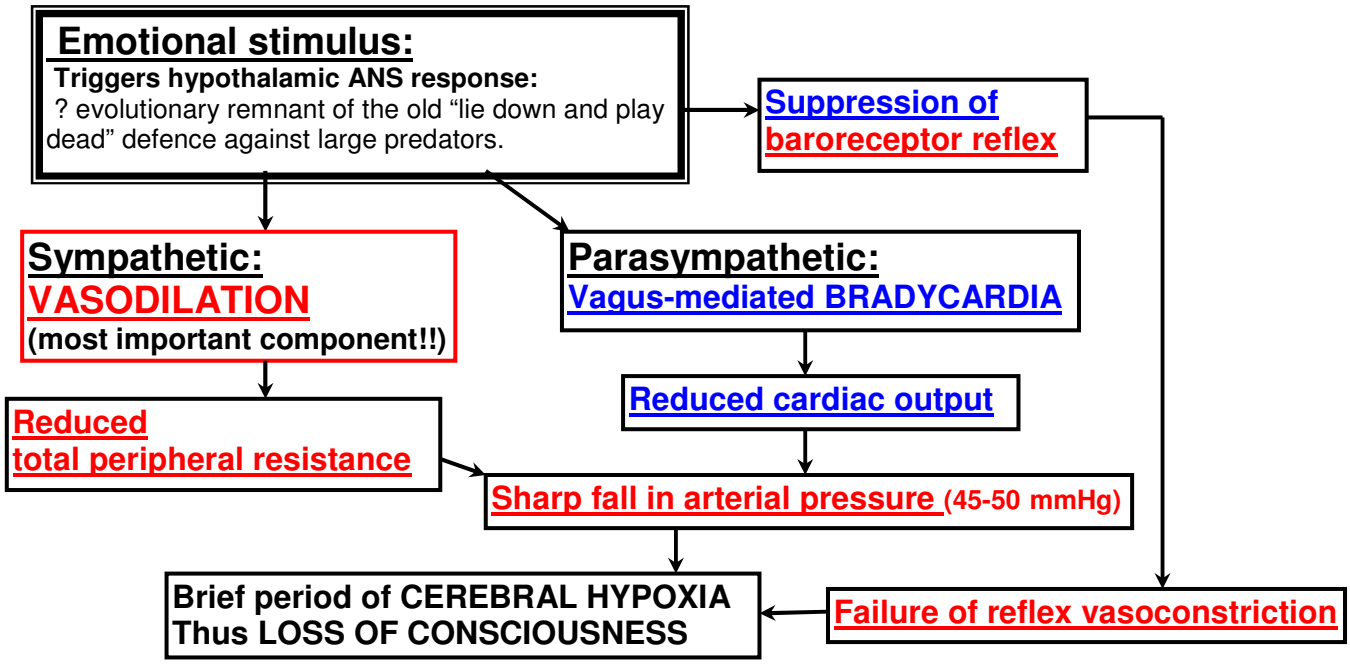
HOWEVER: if a cardiac patient has any type of syncope the prognosis is VERY BAD as it implies an asystole or a travelling atrial fibrillation thrombus

Epidemiology :

- **at least 3% of the population will have a syncopal episode within a 26-year period.**
- **Thirty percent will have recurrences.**

(In the US, Findings from the Framingham study)

Aetiology / Pathophysiology: mechanisms of syncope



WHAT SHOULD HAPPEN HERE: the BARORECEPTOR REFLEX

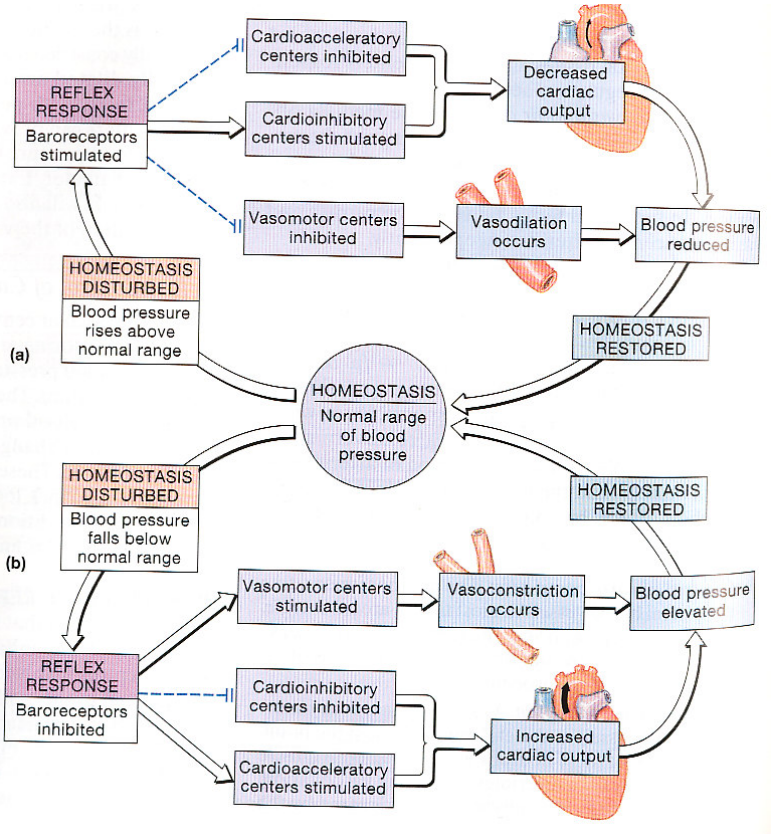
Falling blood pressure:
→ sensed by aortic arch and carotid sinus baroreceptors;

AORTIC signals via VAGUS,
CAROTID signals via GLOSSOPHARYNGEAL
→ feeds back to the heart rate control centre at the medulla: nucleus of solitary tract

→ **medulla controls**
heart rate (via SA node, under vagus control)
and stroke volume (via heart muscle sympathetic efferents)
and arteriole diameter (via sympathetic NS)

thus HR and Stroke Volume increase in response to falling blood pressure,
and the peripheral arterioles constrict

→ all of which increases the blood pressure



Overview of heart function: pressure and volume relationships

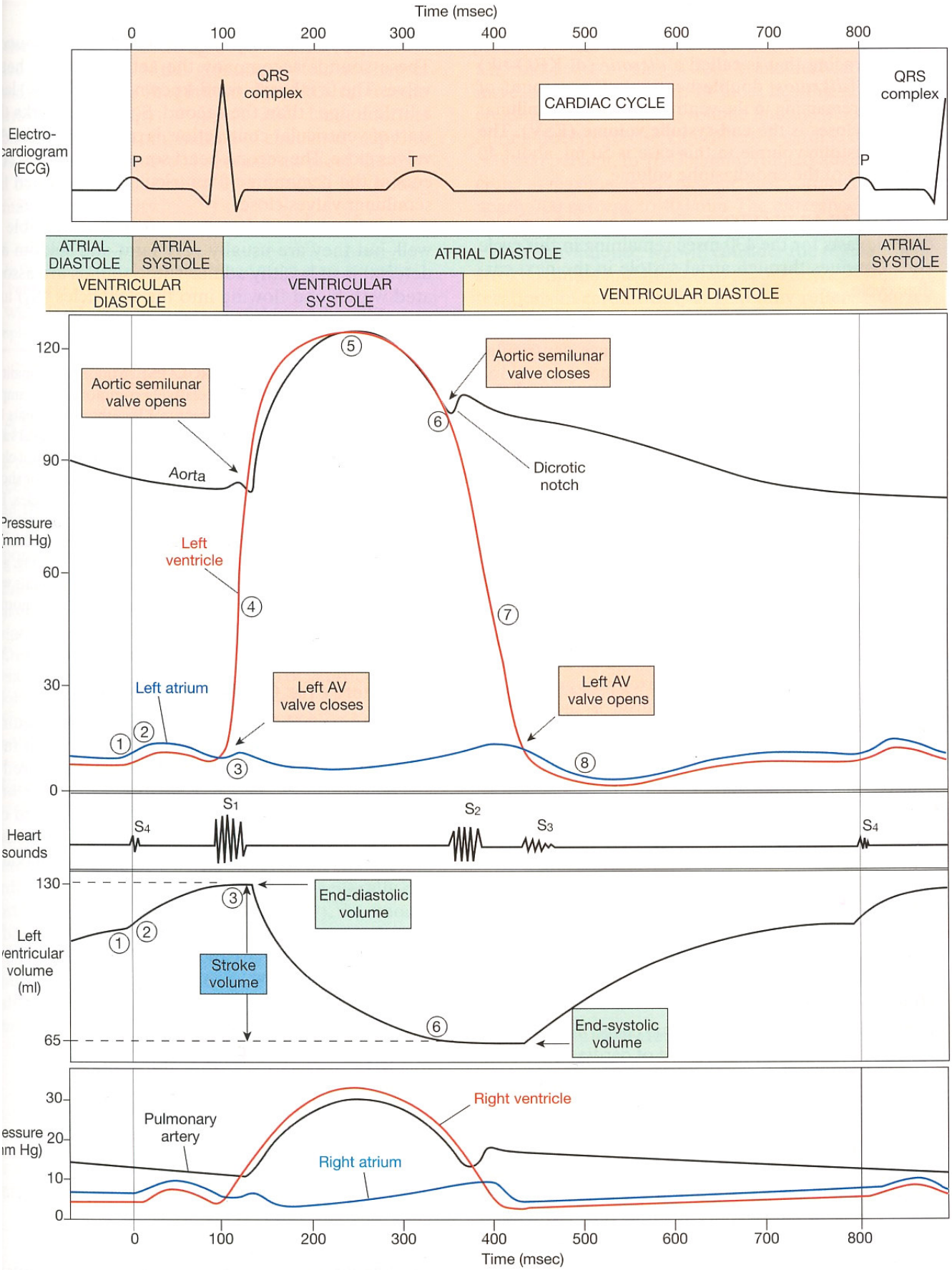


FIGURE 20-16 Pressure and Volume Relationships in the Cardiac Cycle. Major features of the cardiac cycle are shown for a heart rate of 75 bpm. The circled numbers correspond to the numbered list on pages 696 and 698.

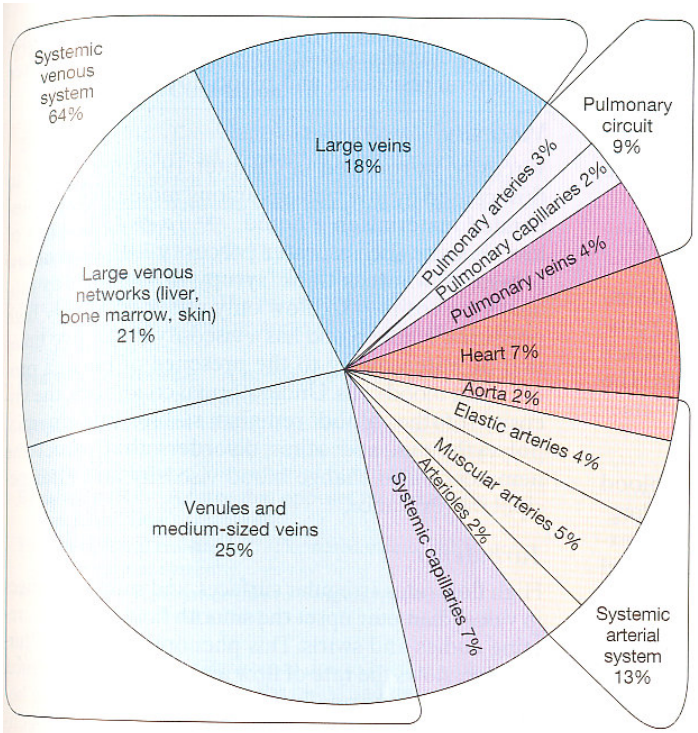


FIGURE 21-8 The Distribution of Blood in the Cardiovascular System

← FACTOIDS →

heart stops for more than 10 seconds, = syncope;
more than 10 minutes = brain damage

the heart beats 2 billion times in 70 years.

RATES OF DIFFUSION: to reach equilibrium

Across 1 micrometre takes 1 msec

Across 1 mm takes 15 min

Across 1 metre takes ~30 years

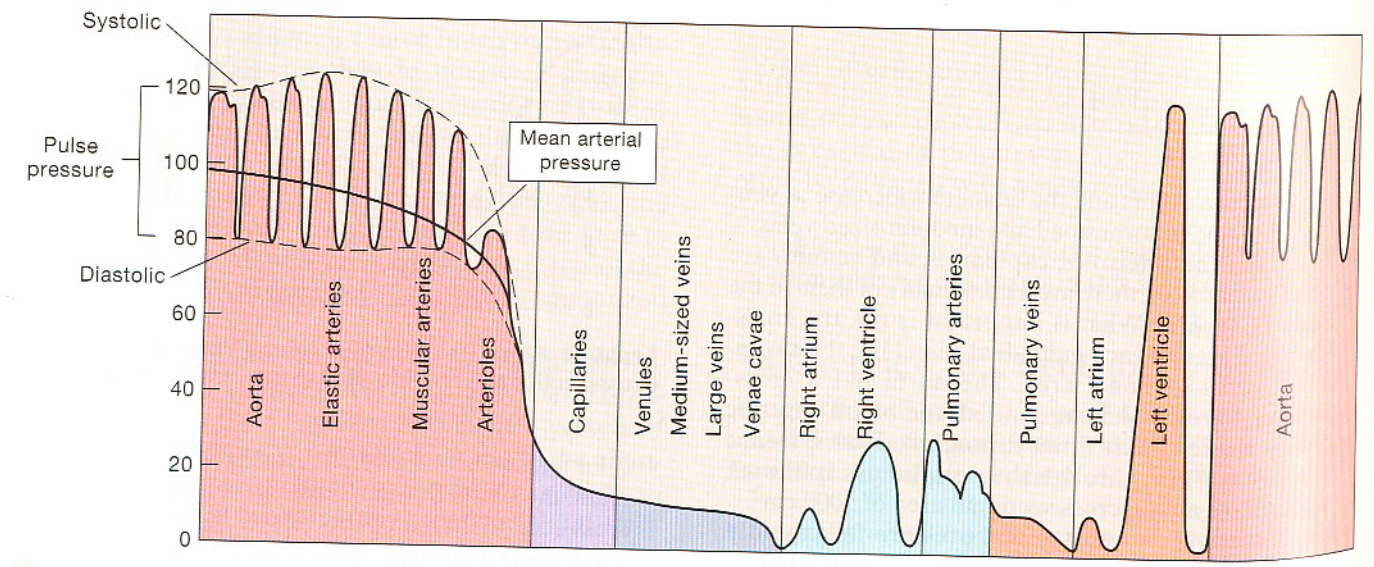


FIGURE 21-10 **Pressures within the Cardiovascular System.** Notice the general reduction in circulatory pressure within the systemic circuit and the elimination of the pulse pressure within the arterioles.

Muscle weakness and fatigue:

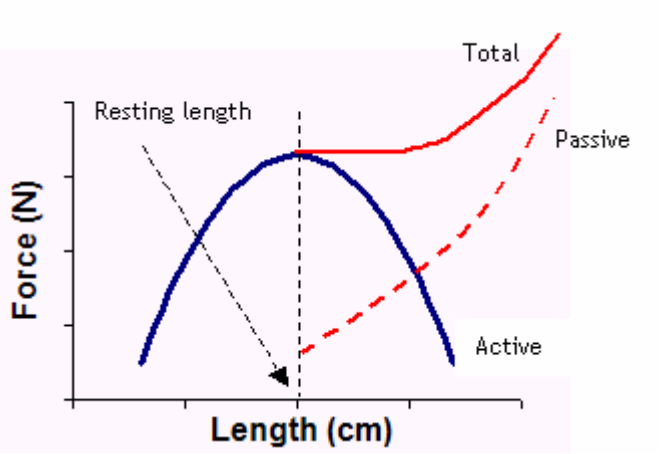
Force production by muscles: *remember the "sliding filament" theory*

Smaller motor units (fewer muscle fibers) have a small motor neuron and a low threshold for activation. These SMALL UNITS ARE RECRUITED FIRST

Effects of :

cross-sectional area: ***THE THICKER THE MUSCLE, THE GREATER THE FORCE***

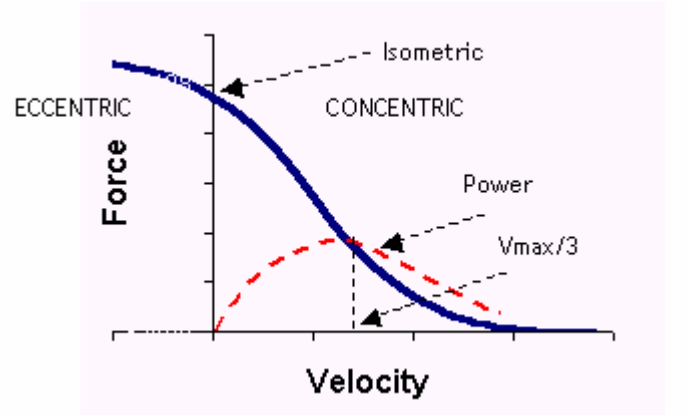
length



ELONGATED MUSCLE = = GREATER FORCE

AN ELONGATED MUSCLE DEVELOPS GREATER FORCE BECAUSE ITS CONNECTIVE TISSUE STRUCTURES CONTRIBUTE RELATIVELY LARGE PASSIVE (ELASTIC) FORCES.

shortening velocity



VELOCITY INCREASES = = FORCE DECREASES

Max power (Force X Velocity) at about ≈30% of max velocity

Central v. peripheral fatigue.

(a) **central involves proximal motor neurons (mainly in the brain)**

caused by inhibition of motor neurons; originates @ reticular formation of brainstem

This kind of fatigue should manifest itself by a decrease in the outflow of motor impulses to the muscles.

(b) **peripheral involves within the motor units**

(i.e., motor neurons, peripheral nerves, motor endplates, muscle fibers).

two different sites where repeated contractions may cause impairment:

the "transmission mechanism" *(neuromuscular jn, muscle membrane, and endoplasmic reticulum)*

and the "contractile mechanism" *(muscle filaments).*

Metabolic changes in muscle and their relation to muscle fatigue

Fatigue causes

- Blood closure
- Occlusion
- Co_2, lactate increase -> H^+ increase -> pH decrease
- Glycogen decrease
- Heat increase

MUSCLE FATIGUE:

= is the transient decrease in performance capacity of muscles, usually evidenced by a failure to maintain or develop a certain expected force or power.

manifested as weakness, pain on exertion, soreness to the touch, and cramping.

FATIGUED MUSCLES become weaker, slower and more efficient at using ATP to maintain tension

THIS IS PROBABLY DUE TO THE pH DECREASE, THUS CAUSING ENZYME DYSFUNCTION

Chronic Fatigue Syndrome

DIAGNOSIS IS BY EXCLUSION !!

= partly physical, partly psychological in nature.

DEFINITION: must satisfy 2 criteria

1. **Have severe chronic fatigue of six months or longer duration** with other known medical conditions excluded by clinical diagnosis, and

2. **Concurrently have four or more of the following symptoms**:
   - substantial impairment in short-term memory or concentration,
   - sore throat, tender lymph nodes,
   - muscle pain,
   - multi-joint pain without swelling or redness,
   - headaches of a new type, pattern or severity,
   - unrefreshing sleep,
   - post-exertional malaise lasting more than 24 hours.

CAUSES OF TIREDNESS

10% of all presentations in general practice
the cause is usually psychological or social (in 50-80% of cases)

Psychosocial factors

associated features of **depression:**
- insomnia,
- morning tiredness,
- loss of interest,
- poor self esteem)

of anxiety
- worry,
- apprehension,
- irritability).

Physical causes

= persistent tiredness as a major manifestation.=
weight loss (malignancy, chronic infection, diabetes, hyperthyroid),
polydipsia, polyuria (diabetes),
fever (infection, malignancy),
cold intolerance (hypothyroidism),
melena, menorrhagia (anaemia),
amenorrhoea, nausea, mastalgia (pregnancy),
hypertension, dyspnoea (congestive cardiac failure),
snoring, daytime somnolence (sleep apnoea).

Infections which commonly cause persistent tiredness include:
- infectious mononucleosis,
- tuberculosis,
- HIV,
- hepatitis
- subacute bacterial endocarditis (SBE).

drugs
- alcohol,
- B-blockers,
- anticonvulsants,
- anxiolytics
- NSAIDS

Chronic fatigue syndrome should be considered in patients with fatigue lasting longer than six months in whom other diseases have been excluded.

Chronic viral infections

Non-specific symptoms of CVI:
- **fatigue**, (may be the presenting symptom in patients with cumulative tissue damage, eg hepatitis C
- **muscle aches**
- **low grade fever**

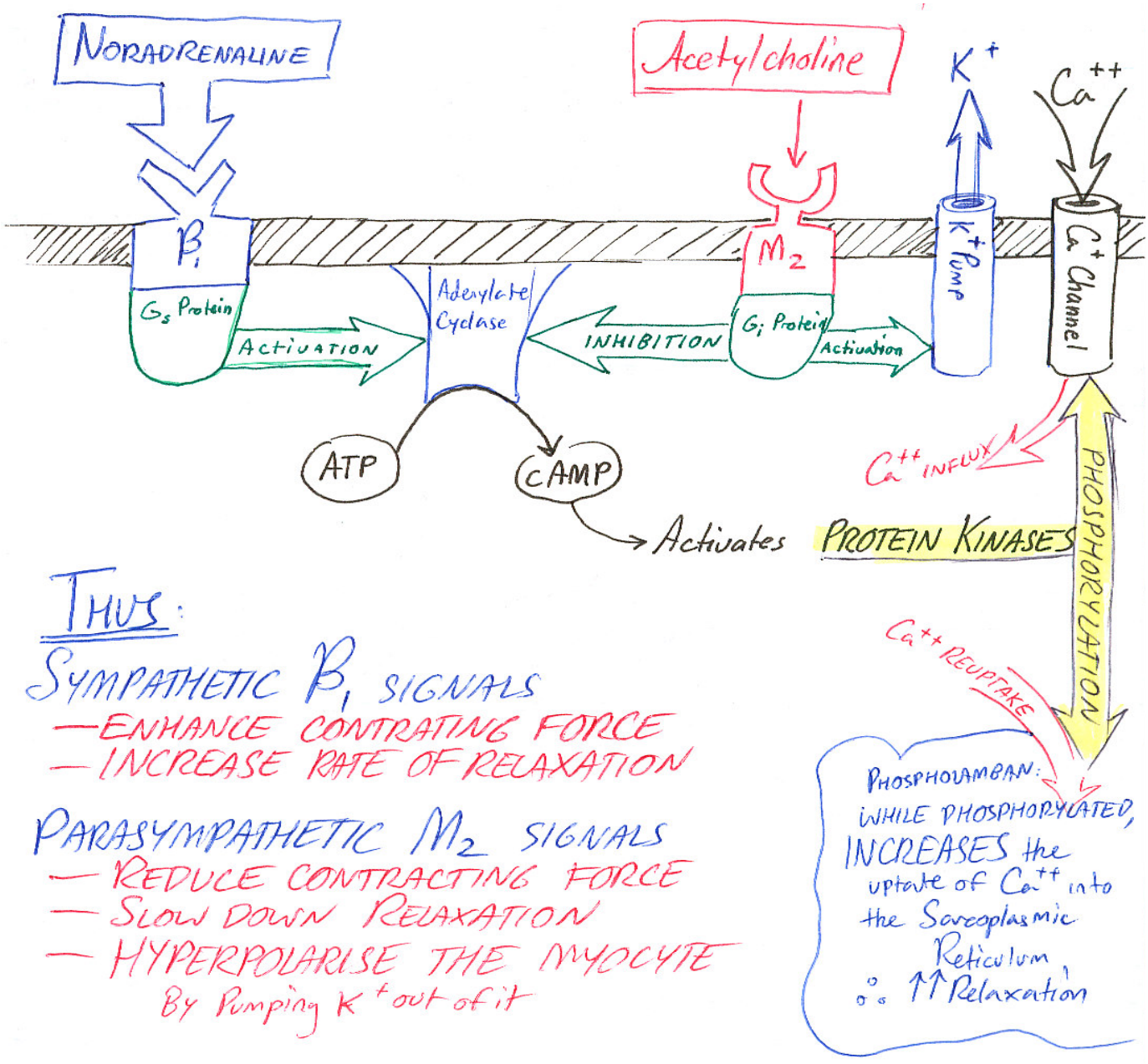
they may…

- **cause apparently acute illness**, by reactivation
- **lead to 'non-communicable' diseases** including malignancies (Burkitt's lymphoma and cancer of the cervix for example) and degenerative CNS syndromes (eg progressive multifocal leucoencephalopathy (PML) , tropical paraparesis)
- **produce continuing long-term damage** to their target organ, either directly or by inducing immunological destruction of infected cells

Chronic viral infections induce on-going production of lymphokines such as interferon, and lymphocyte proliferation may also be found.

The control of chronic viral infections is difficult because asymptomatic people are likely to remain infectious for many years and treatment regimes, if available, are very long term.

Autonomic nervous system physiology and biochemistry



ADRENALINE: affects ALL adrenoceptors

NORADRENALINE: affects all adrenoceptors EXCEPT beta-2

→ which is lucky, as veins have both alpha and beta-2 receptors and wil respond differently to each

Beta-2 relaxes veins
Alpha-1 constricts them

ALPHA-1 = activates phospholipase-C;
thus → increased inositol triphosphate and diacylglycerol (2ndary intracellular messengers)

ALPHA-2 = inhibits adenylyl cyclase, THUS → less cAMP,

BETA-1 and BETA-2 = activate the adenylyl cyclase, thus more cAMP

ALPHA-1 actions:

- smooth muscle contraction (vascular, gastrointestinal sphincters, bladder trigone, urethral internal sphincter, iris radial),
- relaxation of gastrointestinal non-sphincter smooth muscle,
- K^+-rich saliva secretion
- glycogenolysis.

ALPHA-2 actions:

- prejunctional inhibition of noradrenaline release from sympathetic postganglionic nerve terminals (autoinhibition)
- platelet aggregation.
- ***NO REAL NEUROTRANSMISSION!***

BETA-1 actions:

- increases in heart rate and force of contraction,
- relaxation of gastrointestinal non-sphincter smooth muscle,
- amylase-rich saliva secretion
- lipolysis

BETA-2 actions:

- smooth muscle relaxation (vascular, bronchial, bladder detrusor, uterine),
- glycogenolysis
- inhibition of mast cell inflammatory mediator release

Acetylcholine affects all muscarinic receptor subtypes (M_1, M_2, M_3), PLUS both nicotinic receptor subtypes (neuronal, skeletal muscle)
M1, M3 receptors = activate phospholipase C, THUS → increased inositol triphosphate and diacylglycerol (just like alpha 1)
M2 receptors = inhibit adenylyl cyclase, enhance K+ conductance

M1 actions:
- gastric acid secretion
- acetylcholine-induced excitation in sympathetic ganglia

M2 actions:
- decrease in heart rate
- prejunctional inhibition of acetylcholine release from parasympathetic preganglionic nerve terminals (autoinhibition).

M3 actions:
- relaxation of vascular smooth muscle (via the release of endothelial nitric oxide),
- smooth muscle contraction (gastrointestinal, bronchial, bladder detrusor, iris and ciliary circular)
- exocrine gland secretion (K^+-rich salivary, sweat, gastrointestinal, bronchial).

NICOTINIC RECEPTOR ACTIVATION:
the opening of ligand-gated Na^+/K^+ channels;
THUS: **fast excitation (depolarization) of skeletal muscle and neuronal cells.**

Table 11.1 Characteristics of adrenoceptors

	α_1	α_2	β_1	β_2	β_3
Tissues and effects					
Smooth muscle:					
Blood vessels	Constrict	Constrict/dilate		Dilate	
Bronchi	Constrict			Dilate	
GI tract	Relax	Relax (presynaptic effect)		Relax	
GI sphincters	Contract				
Uterus	Contract			Relax	
Bladder detrusor				Relax	
Bladder sphincter	Contract				
Seminal tract	Contract			Relax	
Iris (radial muscle)	Contract				
Ciliary muscle				Relax	
Heart					
Rate			Increase		
Force of contraction			Increase		
Skeletal muscle				Tremor Increased muscle mass and speed of contraction Glycogenolysis	Thermogenesis
Liver	Glycogenolysis			Glycogenolysis	
Fat					Lipolysis Thermogenesis
Pancreatic islets		Decrease insulin secretion			
Nerve terminals					
Adrenergic		Decrease release		Increase release	
Cholinergic		Decrease release			
Salivary gland	K^+ release		Amylase secretion		
Platelets		Aggregation			
Mast cells				Inhibition of histamine release	
Brainstem		Inhibits sympathetic outflow			
Second messengers and effectors	PLC activation ↑ IP_3 ↑ DAG ↑ Ca^{2+}	↓ cAMP ↓ Calcium channels ↑ Potassium channels	↑ cAMP	↑ cAMP	↑ cAMP
Agonist potency order	NA ≥ A ≫ ISO	A > NA ≫ ISO	ISO > NA > A	ISO > A > NA	ISO > NA = A
Selective agonists	Phenylephrine, methoxamine	Clonidine, clenbuterol	Dobutamine, xamoterol	Salbutamol, terbutaline, salmeterol, formoterol	BRL 37344
Selective antagonists	Prazosin, doxazocin	Yohimbine, idazoxan	Atenolol, metoprolol	Butoxamine	

A, adrenaline; cAMP, cyclic 3′,5′-adenosine monophosphate; DAG, diacylglycerol; ISO, isoprenaline; IP_3, inositol trisphosphate; NA, noradrenaline; PLC, phospholipase C.

Table 9.1 The main effects of the autonomic nervous system

Organ	Sympathetic effect	Adrenergic receptor type	Parasympathetic effect	Cholinergic receptor type
Heart				
Sinoatrial node	Rate ↑	β_1	Rate ↓	M_2
Atrial muscle	Force ↑	β_1	Force ↓	M_2
Atrioventricular node	Automaticity ↑	β_1	Conduction velocity ↓	M_2
			Atrioventricluar block	M_2
Ventricular muscle	Automaticity ↑ Force ↑	β_1	No effect	
Blood vessels				
Arterioles				
Coronary	Constriction	α		
Muscle	Dilatation	β_2	No effect	
Viscera	Constriction	α	No effect	
Skin	Constriction	α	No effect	
Brain	Constriction	α	No effect	
Erectile tissue	Constriction	α	Dilatation	? M_3
Salivary gland	Constriction	α	Dilatation	? M_3
Veins	Constriction	α	No effect	
	Dilatation	β_2	No effect	
Viscera				
Bronchi				
Smooth muscle	No sympathetic innervation, but dilated by circulating adrenaline	β_2	Constriction	M_3
Glands	No effect		Secretion	M_3
Gastrointestinal tract				
Smooth muscle	Motility ↓	α_1, α_2, β_2	Motility ↑	M_3
Sphincters	Constriction	α_2, β_2	Dilatation	M_3
Glands	No effect		Secretion	M_3
			Gastric acid secretion	M_1
Uterus				
Pregnant	Contraction	α	Variable	
Non-pregnant	Relaxation	β_2		
Male sex organs	Ejaculation	α	Erection	? M_3
Eye				
Pupil	Dilatation	α	Constriction	M_3
Ciliary muscle	Relaxation (slight)	β	Contraction	M_3
Skin				
Sweat glands	Secretion (mainly cholinergic)	α	No effect	
Pilomotor	Piloerection	α	No effect	
Salivary glands	Secretion	α, β	Secretion	M_3
Lacrimal glands	No effect		Secretion	M_3
Kidney	Renin secretion	β_2	No effect	
Liver	Glycogenolysis Gluconeogenesis	α, β_2	No effect	

The adrenergic and cholinergic receptor types shown are described more fully in Chapters 10 and 11. Transmitters other than acetylcholine and noradrenaline contribute to many of these responses (see Table 9.2).

Behavioural science: coping with gay people

.**About 4 to 5% of the population are preferentially homosexual for their entire lives.**

Since 1973, the American Psychiatric Association has not considered homosexuality a disorder.

Like heterosexuality, homosexuality results from complex biologic and environmental factors leading to an almost inevitable preference in the selection of a sexual partner. **For most, it is not a matter of choice.** Nevertheless, many people, including physicians, regard homosexuality as immoral and sinful, and a physician's intense aversion to homosexuality (homophobia) may interfere with appropriate care of homosexuals.

Well-informed physicians can offer sensitive, disciplined advice on sexual matters and should not miss opportunities for helpful intervention, remembering that sexual practices differ by culture and that the strength of the sexual drive, individual needs, and the frequency of sexual contact vary greatly. Also the physician must consider time taken for family and friends to adjust to a new facet being exposed in the individual. Feelings of loss are common, and should be worked through and considered rationally in a group discussion.

Treating family and friends.:

It is not illegal, but it is discouraged.

Why do people consult doctors ?

- they have developed symptoms and/or signs which obviously threaten their autonomy and quality or duration of life (abdominal pain, swollen joints, sore throat, depression, for example).
- Others have a symptom/sign complex which does not cause any obvious ill health, but threatens to do so in the future (symptomless breast lump, symptomless hypertension).
- A third group have no evidence of disease, but have reason to fear that they will develop a more or less serious illness (**family history of colon cancer, hepatitis B exposure, relationship with HIV sufferer).**
- A fourth group will be in good health and wants to stay that way by ensuring health screening and health advice **(health maintenance plan, breast screening).**
- A fifth will have developed patterns of coping with life which involve using illness as a shelter (**abnormal illness behaviour, some chronic pain syndromes).**
- A sixth will not perceive that they are ill, but are sent by family or friends who do perceive a change (**occult cerebral tumour, alcoholism**).
- Finally, a seventh group present because they find themselves overwhelmed by anxiety or other symptoms of angst (**anxiety states, grieving, burnout).**

The nature of the clinical transaction

Doctor and patient work within an agency relationship, in which the patient entrusts the doctor to become his/her agent in order to secure something which s/he desires.

All such relationships are based on trust.

The transaction begins with the power markedly unbalanced in favour of the doctor and should end with a restoration of power to the patient.

The problems of treating family and friends.

Family and friends do not stand in an agency relationship with you as a doctor. They may trust you, but may also find it difficult to accept your authority when you give advice. It is likely that they will appeal to your special relationship and to make special judgements which may not accord with your own perceptions of right action. The language you commonly use with family and friends is not the language of medicine, and linguistic communication is easily distorted. It is particularly hard to offer probabilistic advice to family and friends who want to know what will happen to them as unique individuals. It is also hard to break bad news without offering false hopes. This all occurs because intersubjectivity is replaced by sympathy and identification with the other. **You are connected with the other by special social and emotional bonds.**

MEDICOLEGAL MUMBO-JUMBO: giving information

Autonomy

"The fundamental principle underlying consent is said to be a right of self-determination: the principle, or value choice, of autonomy of the person ... It is an ethical principle which is simply reflected in legal rules because our law has been developed by judges sensitive to the practical application of generally held community ethical principles (Kirby, M. (1983) "Informed consent: what does it mean?" Journal of Medical Ethics 9:69, at page 70).

Giving information

The giving of information to patients is an ethical obligation and a legal requirement, and relates to the concept of autonomy, that is, enabling the individual to control their own life.

The term "informed consent" clearly specifies that consent to a medical procedure needs to be based on adequate information.

Valid and Informed consent

Informed consent is not synonymous with valid consent, because the requirement that consent be informed is only one aspect of a valid consent. **The individual should**

- have the **legal capacity** to give consent,
- be in a situation where consent may be given freely, without any duress, force, fraud, deceit or coercion,
- have sufficient information to make an informed and enlightened decision.

three areas of law are relevant to informed consent –
(1) the crime of battery,
(2) the tort of trespass which includes assault and battery, and
(3) the tort of negligence.
Practically, the issue is raised usually within the arena of the civil law or torts.
Trespass is a general term for several causes of action, the most relevant here being assault and battery.

- **A battery is an intentional touching of another person without consent of that person and without lawful excuse**;
- **an assault is the intentional creation in another person of an apprehension of imminent, harmful or offensive contact**.
- **Negligence is relevant where it can be shown that there was an obligation on the part of the practitioner to provide certain information, and that if the information had been given to the patient that the patient would not have consented to the procedure being carried out**.

The nature of the information

The patient should be told

- The diagnosis,
- the alternative treatments,
- what the practitioner recommends and why.

The information given to the patient must

- encompass an adequate explanation of the proposed procedure,
- be couched in language that the patient can comprehend.

Giving a patient a pamphlet may well be insufficient, particularly if there is no follow-up to ensure the patient could read and understand the pamphlet, and had no further questions.

Medical jargon should be avoided.

Imprecise terminology such as, "rare", "often", and "not serious") should be **avoided**, in favour of precise description of risks and probabilities (where they are available).

the more invasive the procedure, or the graver the consequences, the greater should be the level of disclosure.

DISCLOSE THE FOLLOWING:

- risk of death,
- disability and other serious consequences
- possible benefit
- effect on work,
- time required for recuperation,

large risks of minor harm and small risks of major harm

A valid consent is not required in an emergency, because the courts have presumed that an unconscious patient in a life-threatening situation would have consented to treatment.

If a patient cannot give a valid consent owing to temporary or permanent disability, recourse should be made to obtaining third party consent, consistent with the provisions of the Guardianship Act 1987 (NSW) (see Learning Topic on Guardianship).

Consent forms

A valid consent form needs to contain details of the actual procedure to be performed.
The signed consent form does not provide proof that the patient consented, but is merely evidence that he or she signed the particular form.
The signature itself does not prove that the patient understood the information given, or the nature and effect of the procedure.
Consent must be operative at the time the procedure is performed, and it is important for the consent form to be signed as close as possible to the time the procedure is performed.
The patient's ability to give valid consent should not be compromised by the effects of pre-operative medication, for example.

DON'T LET THEM SAY "I don't care, yer the doctor, you know whats best"

Waiving the important and fundamental right to autonomy in decision making about one's own body is so significant that **it would be preferable for the waiver to be written, and for independent corroboration to be obtained that the patient prefers a third party to make the decision**.
If the patient persists in refusing to accept information or decision-making responsibility, recourse may have to be made to the Guardianship Tribunal. The **medical practitioner *cannot* assume the right to make decisions on the part of the patient, except in an emergency**.

THE SICK ROLE: responses to health and illness

Role theory is a sociological approach which conceives of all social situations as if individuals are playing parts.

The Sick Role

being sick is associated with a set of obligations and responsibilities which were first described by sociologist Talcott Parsons (1951).
The four main expectations for the sick role are:
(a) a right to exemption from normal social role responsibilities, eg work;
(b) a right not to be held accountable for the illness;
(c) a duty to experience illness as undesirable and not to resign themselves to the illness;
(d) a duty to seek out expert assistance and to cooperate with recommended treatment in attempting to get well.
One implication is that some illnesses are viewed as legitimate, and others which might be interpreted as being the 'fault' of the patient (such as substance abuse related illnesses), are viewed as illegitimate.
Conversely, **some illness sufferers push to have their illness recognised as a medical condition**, this medicalisation supposedly changing society's view of the sufferer from 'bad' to 'ill'

Parson's sick role has been criticised as:

(1) **more appropriate to acute disorders**, not chronic illness where the patient may in fact be encouraged to continue normal social role obligations; (2) **medicalising some disorders and removing patient's responsibilities for behavioural choices**;
(3) it is culture- and class-bound.

Labelling

Friedson (1970) comments on the unequal nature of the doctor-patient relationship, an**d the power of the doctor to label illness**.
The process of labelling is two-pronged; on one hand it may assist the patient by allowing understanding of their condition and relief from social role obligations, whereas on the other hand **it may cause the patient to enter the sick role unnecessarily (eg male patients who defined themselves as sick, when it was found that they had asymptomatic high blood pressure**), and may stigmatise the patient (as with mental illness).

Illness Behaviour

Mechanic introduced the term 'illness behaviour' to describe activities undertaken by a person who has symptoms in order to define the state of their health and discover a suitable remedy. Associated with this is the premise that **the experience of illness may be used to achieve social and personal goals unrelated to alterations in biological systems** or the pathogenesis of disease, that is, **secondary gain**.
Particular groups of individuals have been found to be **under-utilisers of medical services** and to be more likely to tolerate symptoms of disease, have a different definition of symptoms from the medical profession, be likely to consult friends or family or alternative healers, be frightened or wary about going to the doctor, feel that little could be done about their condition and be unable to 'make time' to go to the doctor. These intervening variables mediating between the presence of symptoms and the act of consulting a doctor are often patterned according to social and cultural factors, including the ability to tolerate pain, the social and cultural meaning of symptoms, and the social network of the individual.

Abnormal Illness Behaviour

In Mechanic's formulation of illness behaviour, the term 'abnormal illness behaviour' is non-evaluative and describes no more than statistical variation from a norm. Pilowsky, however, has developed an extension of this usage to cover *a range of disease-associated behaviours often labelled hypochondriacal, hysterical, malingering*, and so on. He defines abnormal illness behaviour as ***"the persistence of an inappropriate or maladaptive mode of perceiving, evaluating and acting in relation to one's own state of health, despite the fact that a doctor (or other appropriate social agent) has offered a reasonably lucid explanation of the nature of the illness and the appropriate course of management to be followed".***

DEPRESSION AND SOMATISATION

50% of cardiology referrals have a non-cardiac cause for their chest pain

SOMATISATION:the tendency to conceptualise, express and experience mental states and emotions as physiological symptoms or altered bodily functions

SOMATIC SYMPTOMS OF DEPRESSION:

- **disturbed sleep patterns**
- **appetite change**
- **weight change**
- **decrease in sexual drive**
- **loss of energy and fatigue**
- **menstrual changes**

Why eat so much when depressed?

WELL! Craving for carbohydrates may serve a function, as their consumption is SEROTONERGIC and will raise levels of serotonin, thus → antidepressant effect

(partial speculation, results may not be reproducible)

ETHICAL DECISION-MAKING FOR DUMMIES:

The bioethical principles that are widely recognised as fundamental to medical practice include

- beneficence,
- non-maleficence,
- autonomy
- distributive justice.

Just follow these simple steps and you'll never suffer the crab-like pinches of your guilty conscience.

- First identify the values in conflict (eg the responsibility to tell "the truth" versus respect for alternative views)
- Identify the options (what choices do I have in this situation?)
- Identify the key facts - medical, technical, psychosocial and legal
- Who has a problem - is it mine, the patient's, the family's, society's?
- What are the consequences of the choices that I could make?
- How should we weigh the competing values?
- How will I evaluate the results of my decisions?

"Nor is it always in the most distinguished achievements that men's virtues and vices may be best discerned, but very often an action of small note, a short saying, or a jest, shall distinguish a person's real character more than the greatest sieges, or the most important battles."

Plutarch