Initial evaluation of the patient: vital signs and toxic syndromes

- **Vital signs (also known as vitals)** are a group of the four to six most important signs that indicate the status of the body's vital (life-sustaining) functions. These measurements are taken to help assess the general physical health of a person, give clues to possible diseases, and show progress toward recovery. The normal ranges for a person's vital signs vary with age, weight, gender, and overall health.
- There are four **primary vital signs**: body temperature, blood pressure, pulse (heart rate), and breathing rate (respiratory rate).

- In the practice of medical toxicology, **vital signs play an important role??**

1-Assessing and monitoring the overall status of a patient

2-They frequently provide valuable physiologic signs to the toxicologic etiology and severity of an illness.

3- A valuable parameter, which are used to assess and monitor a patient's response to supportive treatment and antidotal therapy.

This broad range of values considered normal should serve merely as a guide. Only a complete assessment of a patient can determine whether or not a particular vital sign is truly clinically normal

- The difficulty in defining what constitutes "**normal**" vital signs in an emergency setting has been inadequately addressed and may prove to be an impossible undertaking??

1-Published normal values may have **little significance to an acutely ill or anxious patient** in the emergency setting, yet that is precisely the environment in which we must define abnormal vital signs and address them accordingly.

2-Even in non-emergent situations, "normalcy" of vital signs ***depends on the clinical condition of the patient***. A sleeping or comatose patient may have physiologic bradycardia; a slow heart rate appropriate for his or her low energy requiring state

*Many xenobiotics affect the autonomic nervous system, which, in turn, affects the vital signs via the sympathetic pathway, the parasympathetic pathway, or both.

*Careful attention to both the initial and repeated determinations of vital signs is of extreme importance in identifying a pattern of changes suggesting a particular xenobiotic or group of xenobiotics

- The value of serial monitoring of the vital signs is demonstrated by the patient who presents with an anticholinergic overdose who is then given the antidote, physostigmine.
- In this situation, it is important to recognize (i.e., anticholinergic syndrome followed by physostigmine excess) when tachycardia becomes bradycardia.

- Similarly, consider the course of a patient who has ***opioid-induced bradypnea*** (a decreased rate of breathing) and then develops ***tachypnea*** (an increased rate of breathing) after the administration of the opioid antagonist ***naloxone***. The analysis becomes exceedingly complicated when that patient may have been exposed to two or more substances, such as an **opioid combined with cocaine**.??

- In this situation, the effects of cocaine may be "**unmasked**" by the naloxone used to counteract the opioid, and the clinician must then be forced to ***differentiate naloxone induced opioid withdrawal from cocaine toxicity***. The assessment starts by analyzing diverse information, including vital signs, history, and physical examination

- The signs that prove most clinically useful are those involving the central nervous system (CNS; mental status), ophthalmic system (pupil size), gastrointestinal system (peristalsis), dermatologic system (skin dryness versus diaphoresis), mucous membranes (moistness versus dryness), and genitourinary system (urinary retention versus incontinence).

In some instances, an unexpected combination of findings may be particularly helpful in identifying a xenobiotic or a combination of xenobiotics.

For example, a dissociation between such typically paired changes as an increase in pulse with a decrease in blood pressure (cyclic antidepressants or phenothiazines), or the presentation of a decrease in pulse with an increase in blood pressure (ergot alkaloids) may be extremely helpful in diagnosing a toxic etiology.

BLOOD PRESSURE

Xenobiotics cause ***hypotension*** by four major mechanisms:

1- decreased peripheral vascular resistance,

2-decreased myocardial contractility.

3-dysrhythmias,

4-depletion of intravascular volume.

Many xenobiotics can initially cause orthostatic hypotension without marked hypotension, and any xenobiotic that affects autonomic control of the heart or peripheral capacitance vessels may lead to orthostatic hypotension .

Hypertension from xenobiotics may be caused by

1-CNS sympathetic over activity,

2-Increased myocardial contractility

3-Increased peripheral vascular resistance,

4-A combination of these.

Blood pressure and pulse rate may vary significantly as a result of

a)-Changes in receptor responsiveness.

b)-Degree of physical fitness.

c)-Degree of atherosclerosis.

- Changing patterns of blood pressure often assist in the diagnostic evaluation: overdose with a monoamine oxidase inhibitor (MAOI) characteristically causes an initial normal blood pressure, to be followed by hypertension, which, in turn, may be followed abruptly by severe hypotension

TABLE Common Xenobiotics That Affect the Blood Pressure[a]

Hypotension	Hypertension
α_1-Adrenergic antagonists	α_1-Adrenergic agonists
α_2-Adrenergic agonists	α_2-Adrenergic antagonists
β-Adrenergic antagonists	Ergot alkaloids
Angiotensin-converting enzyme inhibitors and angiotensin receptor blockers	Lead (chronic)
Antidysrhythmics	Monoamine oxidase inhibitors (overdose early and drug–food interaction)
Calcium channel blockers	Nicotine (early)
Cyanide	Phencyclidine
Cyclic antidepressants	Sympathomimetics
Ethanol and other alcohols	
Iron	
Methylxanthines	
Nitrates and nitrites	
Nitroprusside	
Opioids	
Phenothiazines	

PULSE RATE

- Although the carotid artery is usually easily palpable, for reasons of both safety and reliability, the brachial artery is preferred in infants and in adults older than 60 years.
- The normal heart rate for adults was defined by consensus more than 50 years ago as a regular rate greater than 60 beats/min and less than 100 beats/min.
- More recent studies suggest that 95% of the population have bradycardia and tachycardia thresholds of 50 beats/min and 90 beats/min, respectively.

Because pulse rate is the net result of a balance between sympathetic (adrenergic) and parasympathetic (muscarinic and nicotinic) tone, many xenobiotics that exert therapeutic or toxic effects or cause pain syndromes, hyperthermia, or volume depletion also affect the pulse rate.

With respect to temperature, there is a direct correlation between pulse rate and temperature in that **pulse rate increases approximately 8 beats/min for each (1°C)**

- In trying to differentiate between a **sympathomimetic and anticholinergic toxic syndrome**, it should be understood that although tachycardia commonly results from both sympathomimetic and anticholinergic xenobiotics, when tachycardia is accompanied by diaphoresis or increased bowel sounds, **adrenergic toxicity** is suggested, but when tachycardia is accompanied by decreased sweating, absent bowel sounds, and urinary retention, **anticholinergic toxicity** is likely

TABLE 2 Common Xenobiotics That Affect the Pulse[a]

Bradycardia	Tachycardia
α_2-Adrenergic agonists	Anticholinergics
β-Adrenergic antagonists	Antipsychotics
Baclofen	Cyclic antidepressants
Calcium channel blockers	Disulfiram/ethanol interaction
Cardioactive steroids	Ethanol and sedative–hypnotic withdrawal
Ciguatoxin	Iron
Ergot alkaloids	Methylxanthines
γ-Hydroxybutyric acid	Phencyclidine
Opioids	Sympathomimetics
Organic phosphorus	Thyroid hormone

Respirations

Establishment of an airway and evaluation of respiratory status are the initial priorities in patient stabilization.

Although respirations are typically assessed initially for

rate

depth

pattern

Is important essential for establishing the etiology of a systemic illness or toxicity

- ***Hyperventilation*** may result from the

1-Direct effect of a CNS stimulant, such as the direct effect of salicylates, on the brainstem. However, salicylate poisoning .characteristically produces hyperventilation

2-Pulmonary injury from any source, including aspiration of .gastric contents

- Bradypnea may occur when a CNS depressant acts on the brainstem.
- **A progression from fast to slow breathing may also occur in a patient exposed to increasing concentrations of cyanide or carbon monoxide.

TABLE 3 Common Xenobiotics That Affect Respiration[a]

Bradypnea	Tachypnea
α_2-Adrenergic agonists	Cyanide
Botulinum toxin	Dinitrophenol and congeners
Elapidae venom	Epinephrine
Ethanol and other alcohols	Ethylene glycol
γ-Hydroxybutyric acid	Hydrogen sulfide
Neuromuscular blockers	Methanol
Opioids	Methemoglobin producers
Organic phosphorus compounds	Methylxanthines
Sedative-hypnotics	Nicotine (early)
	Pulmonary irritants
	Salicylates

Temperature

temperature assessment can be done only if safe and reliable equipment is used.

The risks of inaccuracy are substantial when an oral temperature is taken in a tachypneic patient.

an **axillary temperature or a temporal artery temperature** is taken in any patient (especially those found outdoors),

*rectal temperatures using a non glass probe is essential for safe and accurate temperature determinations in agitated individuals and is considered the standard method of temperature determination.

The core temperature or deep internal temperature (T) is relatively stable (37° ±0.6°C) under normal physiologic circumstances.

Hypothermia (T <35°C) and hyperthermia (T >38°C) are common manifestations of toxicity.

Severe or significant hypothermia and hyperthermia may result in grave complications and inappropriate or inadequate resuscitative efforts.

Life-threatening hyperthermia (T >41.1°C) from any cause may lead to extensive rhabdomyolysis, myoglobinuric renal failure, and direct liver and brain injury and must therefore be identified and corrected immediately.

- Hyperthermia may result from a distinct neurologic response to a signal demanding thermal “up regulation.”

This signal can be from internal generation of heat beyond the capacity of the body to cool, such as occurs in association with

1- Agitation or mitochondrial uncoupling.

2-From an externally imposed physical or environmental factor, such as the environmental conditions causing heat stroke .

- **Hyperthermia** differs from **fever** in that the body's temperature set point remains unchanged. The opposite is **hypothermia**, which occurs when the temperature drops below that required to maintain normal metabolism

TABLE 4 Common Xenobiotics[a] That Affect Temperature[b]

Hyperthermia	Hypothermia
Anticholinergics	α_2-Adrenergic agonists
Chlorphenoxy herbicides	Carbon monoxide
Dinitrophenol and congeners	Ethanol
Malignant hyperthermia[a]	γ Hydroxybutyric Acid
Monoamine oxidase inhibitors	Hypoglycemics
Neuroleptic malignant syndrome[a]	Opioids
Phencyclidine	Sedative-hypnotics
Salicylates	Thiamine deficiency
Sedative-hypnotic or ethanol withdrawal	
Serotonin syndrome[a]	
Sympathomimetics	
Thyroid hormone	

Hypothermia is probably less of an immediate threat to life than hyperthermia, but it requires rapid appreciation, accurate diagnosis, and skilled management.

Hypothermia impairs the metabolism of many xenobiotics, leading to unpredictable delayed toxicologic effects when the patient is warmed.

Toxin	HR/BP	Resp	Temp	Eyes	Mental status
Sympathomimetic	↑↑	↑	↑	↑	agitated
Anticholinergic	H.W	H.W		H.W	H.W
Cholinergic	H.W	H.W	H.W	H.W	H.W
Sedative-hypnotic	H.W	H.W	H.W	H.W	H.W
Opioids	H.W	H.W	H.W	H.W	H.W

Thank you