MCQs BASED Topics

Sunday, March 13, 2016

Dr Kamal Deep Fortis Delhi created the group «**MCQs BASED Topics**»

Dr Kamal Deep Fortis Delhi 5:29:26 PM

Current intensity	Effect
1 mA	Tingling sensation, almost not perceptible
3-5 mA	"Let-go" current for an average child
6-9 mA	"Let-go" current for an average adult
16 mA	Maximum current a person can grasp and "let go"
16-20 mA	Tetany of skeletal muscles
20-50 mA	Paralysis of respiratory muscles; respiratory arrest
50-100 mA	Threshold for ventricular fibrillation
>2 A	Asystole
15-30 A	Common household circuit breakers
240 A	Maximal intensity of household current (U.S.)

Dr Kamal Deep Fortis Delhi 5:50:00 PM

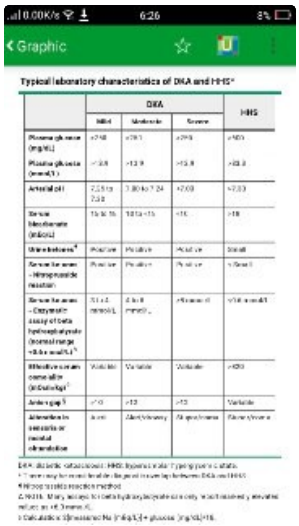
Electricity physics in UPTODATE

ven relatively low-intensity current (30 mA) at the frequency of household current (60 Hz) may induce an indefinite refractory state at the neuro-muscular junction, causing continuous tetanic contractions of involved muscles.

Dr Kamal Deep Fortis Delhi 6:21:59 PM

DKA:- Diagnostic Criteria

Dr Kamal Deep Fortis Delhi 6:37:31 PM



Dr Kamal Deep Fortis Delhi 8:06:31 PM

Acute pancreatitis:-
A CT scan is not required on the first day unless other diagnoses are being considered. It takes time for pancreatic necrosis to develop, and thus CT may be normal in the first 48 hours. As noted above, a guideline issued by the American Gastroenterological Association (AGA) suggests a CT be performed after 72 hours of illness in patients with predicted severe disease and those with evidence of organ failure during the initial 72 hours.

CLASSIFICATION — According to the Atlanta classification, acute pancreatitis can be divided into two broad categories [6]: 8:07:31 PM

●Interstitial edematous acute pancreatitis, which is characterized by acute inflammation of the pancreatic parenchyma and peripancreatic tissues, but without recognizable tissue necrosis.
●Necrotizing acute pancreatitis, which is characterized by inflammation associated with pancreatic parenchymal necrosis and/or peripancreatic necrosis.
According to the severity, acute pancreatitis is divided into the following:

●Mild acute pancreatitis, which is characterized by the absence of organ failure and local or systemic complications
●Moderately severe acute pancreatitis, which is characterized by no organ failure or transient organ failure (<48 hours) and/or local complications
●Severe acute pancreatitis, which is characterized by persistent organ failure (>48 hours) that may involve one or multiple organs

8:08:06 PM

Transfer to a monitored or intensive care unit may be considered in the following patients, although it is not uncommon for these patients to be treated on the floor in centers with significant expertise in acute pancreatitis [9]:

●APACHE II score >8 in the first 24 hours of admission (calculator 1)
●Persistent (>48 hours) SIRS (table 2)
●Elevated hematocrit (>44 percent), blood urea nitrogen (BUN) (>20 mg/dL), or creatinine (>1.8 mg/dL)
●Age >60 years
●Underlying cardiac or pulmonary disease, obesity

The American Gastroenterological Association (AGA) has issued guidelines for assessing the severity of pancreatitis [29]. The AGA recommends: 8:09:22 PM

●Prediction of severe disease be performed using the APACHE II system (using a cutoff of ≥8) (calculator 1).
●Those with actual or predicted severe disease and those with other severe comorbid conditions should be considered for triage to an intensive care or intermediate medical care unit.
●In patients with predicted severe disease (ie, APACHE II score of ≥8) and those with evidence of organ failure during the initial 72 hours, rapid-bolus computed tomography (CT) should be performed after 72 hours of illness to assess the degree of pancreatic necrosis. CT should be used selectively based upon clinical features in patients who do not meet these criteria.
●Laboratory tests can be used as an adjunct to clinical judgment and the APACHE II score. A serum C-reactive protein level of >150 mg/L at 48 hours is preferred

CT SEVERITY INDEX:- Balthazar 8:11:04 PM
ORGAN BASED:- MARSHALL ETC

Monday, March 14, 2016

Dr Kamal Deep Fortis Delhi 1:10:46 AM

1 mmol/l = 18 mg/dl glucose

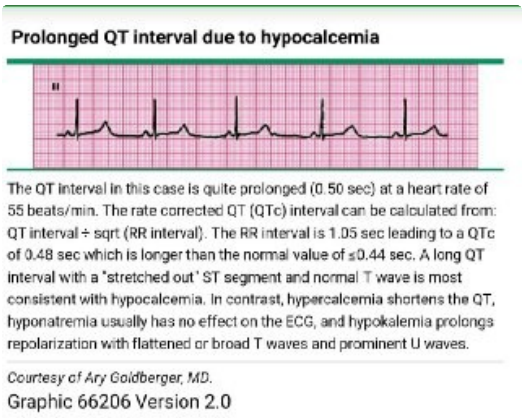
Dr Kamal Deep Fortis Delhi 4:04:44 PM

Hypertension — The following definitions were suggested in 2003 by the seventh report of the Joint National Committee (JNC 7) and are based upon the average of two or more properly measured (table 2) readings at each of two or more office visits after an initial screening [4,5]:

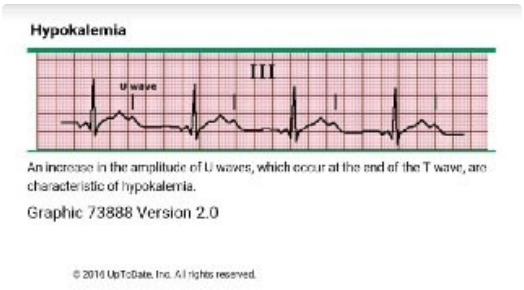
●Normal blood pressure: systolic <120 mmHg and diastolic <80 mmHg
●Prehypertension: systolic 120 to 139 mmHg or diastolic 80 to 89 mmHg (see "Prehypertension")
●Hypertension:
•Stage 1: systolic 140 to 159 mmHg or diastolic 90 to 99 mmHg
•Stage 2: systolic ≥160 mmHg or diastolic ≥100 mmHg

Resistant hypertension — Resistant hypertension is defined as: blood pressure that is not controlled despite adherence to an appropriate three-drug regimen (including a diuretic) in which all drugs are dosed at 50 percent or more of the maximum recommended antihypertensive dose; or blood pressure that requires at least four medications to achieve control. 4:06:55 PM

Dr Kamal Deep Fortis Delhi 4:37:53 PM



4:38:32 PM



Dr Kamal Deep Fortis Delhi 5:57:04 PM

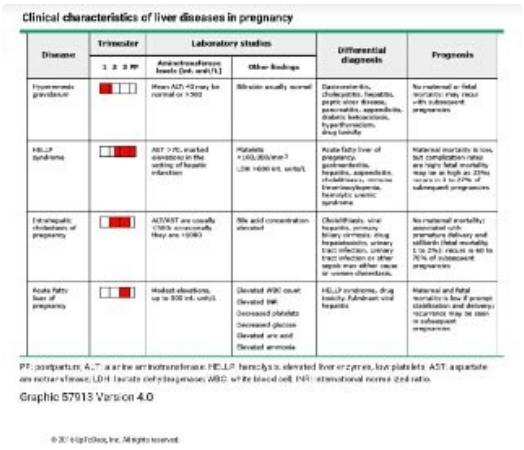
Listeria meningitis seen in elderly more than 60 years of age or immunocompromised.with diarrhea

Dr Kamal Deep Fortis Delhi 7:56:44 PM

Management of ARDS

MANAGEMENT OF SEPSIS...Always give more fluid first if fluid responsive before moving to OT for source control 7:57:25 PM

Dr Kamal Deep Fortis Delhi 11:44:54 PM



Clinical characteristics of liver diseases in pregnancy

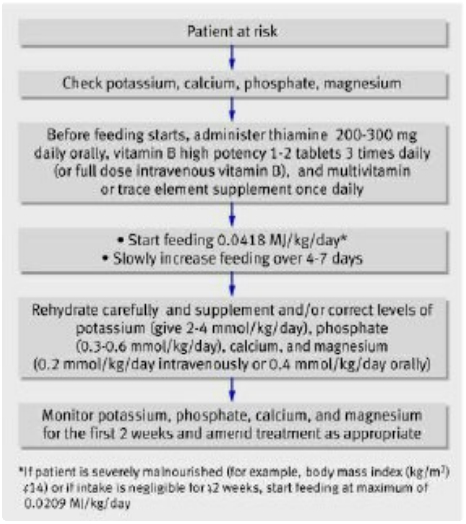
Disease	Trimester 1 2 3 PP	Laboratory studies: Aminotransferase levels (Int. unit/L)	Laboratory studies: Other findings	Differential diagnosis	Prognosis
Hyperemesis gravidarum		Mean AST <3 may be normal or >300	Bilirubin usually normal	Gastroenteritis, cholecystitis, hepatitis, peptic ulcer disease, pancreatitis, appendicitis, diabetic ketoacidosis, hyperthyroidism, drug toxicity	No maternal or fetal mortality; may recur with subsequent pregnancies
HELLP syndrome		AST >70, marked elevations in the setting of hepatic infection	Platelets <100,000/mm³ LDH >600 int. units/L	Acute fatty liver of pregnancy, gastroenteritis, hepatitis, appendicitis, cholelithiasis, immune thrombocytopenia, hemolytic uremic syndrome	Maternal mortality is low, but complication rates are high; fetal mortality may be as high as 23%; recurs in 4 to 27% of subsequent pregnancies
Intrahepatic cholestasis of pregnancy		AST/ALT are usually <300; occasionally they are >1000	Bile acid concentration elevated	Cholelithiasis, viral hepatitis, primary biliary cirrhosis, drug hepatotoxicity, urinary tract infection, urinary tract infection or other sepsis may either cause or worsen cholestasis	No maternal mortality; associated with premature delivery and stillbirth (fetal mortality 1 to 2%); HELLP is 40 to 70% of subsequent pregnancies
Acute fatty liver of pregnancy		Modest elevations, up to 500 int. units/L	Elevated WBC count; Elevated INR; Decreased platelets; Decreased glucose; Elevated uric acid; Elevated ammonia	HELLP syndrome, drug toxicity, fulminant viral hepatitis	Maternal and fetal mortality is low if prompt stabilization and delivery; recurrence may be seen in subsequent pregnancies

PP: postpartum; ALT: alanine aminotransferase; HELLP: hemolysis, elevated liver enzymes, low platelets; AST: aspartate aminotransferase; LDH: lactate dehydrogenase; WBC: white blood cell; INR: international normalized ratio.

Graphic 57913 Version 4.0

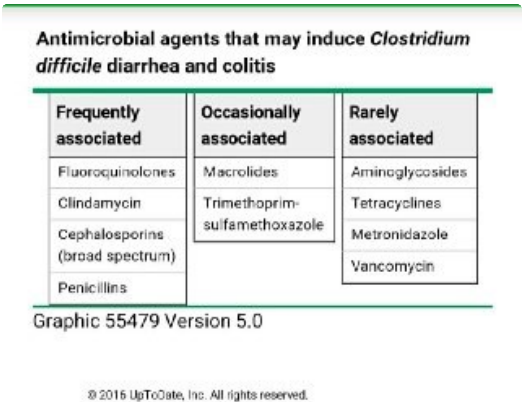
Tuesday, March 15, 2016

Dr Kamal Deep Fortis Delhi 2:32:48 AM



Refeeding Syndrome above 2:33:02 AM

Dr Kamal Deep Fortis Delhi 3:32:30 AM

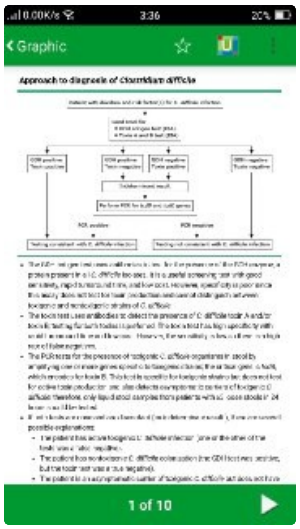


Antimicrobial agents that may induce *Clostridium difficile* diarrhea and colitis

Frequently associated	Occasionally associated	Rarely associated
Fluoroquinolones	Macrolides	Aminoglycosides
Clindamycin	Trimethoprim-sulfamethoxazole	Tetracyclines
Cephalosporins (broad spectrum)		Metronidazole
Penicillins		Vancomycin

Graphic 55479 Version 5.0

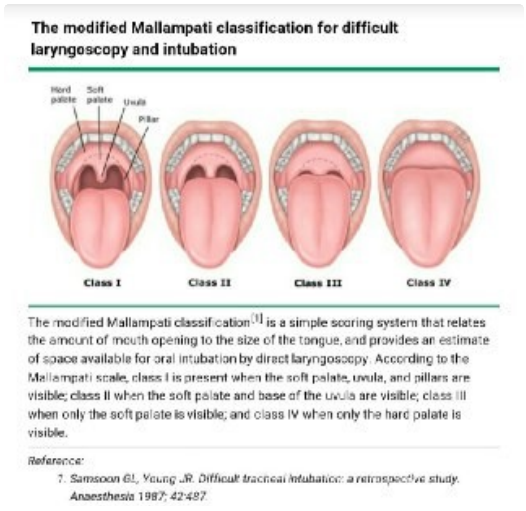
3:38:34 AM



Dr Kamal Deep Fortis Delhi 2:29:18 PM

Wednesday, March 16, 2016

Dr Kamal Deep Fortis Delhi 1:16:22 PM

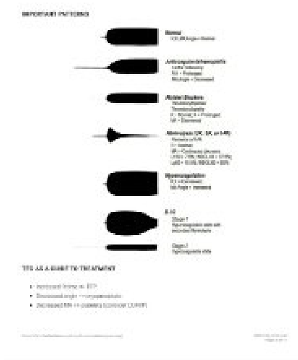


In Trauma patient ATLS..treating tension pneumothorax, hemothorax and cardiac tamponade is in primary survey as they are life threatening

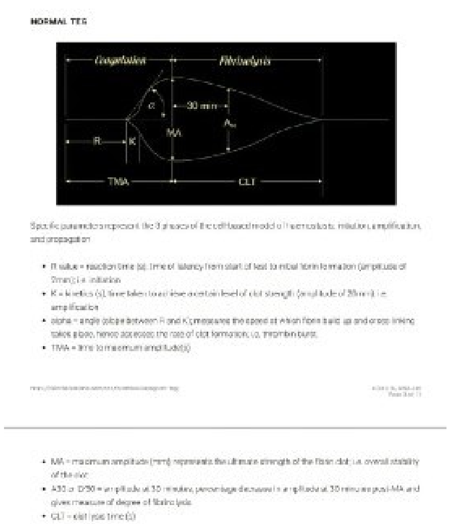
1:17:02 PM

Dr Kamal Deep Fortis Delhi

2:46:31 PM

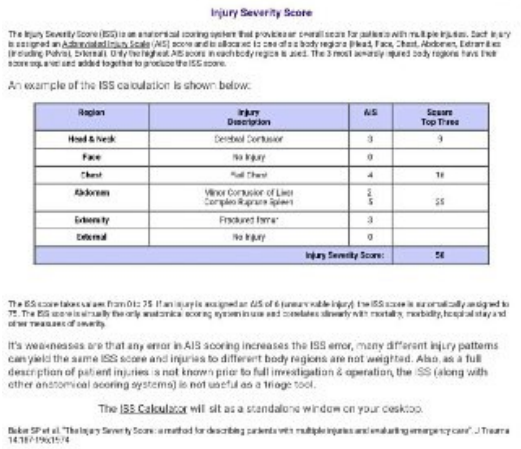


2:46:59 PM



Dr Kamal Deep Fortis Delhi

6:21:48 PM



Injury Severity Score

The Injury Severity Score (ISS) is an anatomical scoring system that provides an overall score for patients with multiple injuries. Each injury is assigned an Abbreviated Injury Scale (AIS) score and is allocated to one of six body regions (Head, Face, Chest, Abdomen, Extremities (including Pelvis), External). Only the highest AIS score in each body region is used. The 3 most severely injured body regions have their scores squared and added together to produce the ISS score.

An example of the ISS calculation is shown below:

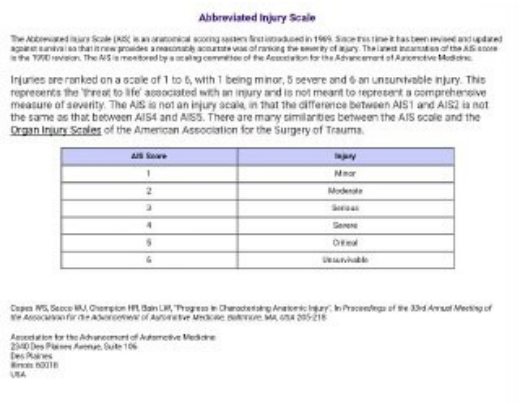
Region	Injury Description	AIS	Score Top Three
Head & Neck	Cerebral Contusion	3	9
Face	No Injury	0	
Chest	Flail Chest	4	16
Abdomen	Minor Contusion of Liver Complex Rupture Spleen	2 5	25
Extremity	Fractured Femur	3	
External	No Injury	0	
		Injury Severity Score:	50

The ISS score takes values from 0 to 75. If an injury is assigned an AIS of 6 (unsurvivable injury) the ISS score is automatically assigned to 75. The ISS score is virtually the only anatomical scoring system in use and correlates strongly with mortality, morbidity, hospital stay and other measures of severity.

It's weaknesses are that any error in AIS scoring increases the ISS error, many different injury patterns can yield the same ISS score and injuries to different body regions are not weighted. Also, as a full description of patient injuries is not known prior to full investigation & operation, the ISS (along with other anatomical scoring systems) is not useful as a triage tool.

The ISS Calculator will sit as a standalone window on your desktop.

Baker SP et al. "The Injury Severity Score: a method for describing patients with multiple injuries and evaluating emergency care". J Trauma 14:187-196(1974)



Abbreviated Injury Scale

The Abbreviated Injury Scale (AIS) is an anatomical scoring system first introduced in 1969. Since this time it has been revised and updated against survival so that it now provides a reasonably accurate way of ranking the severity of injury. The latest incarnation of the AIS score is the 1990 revision. The AIS is monitored by a scaling committee of the Association for the Advancement of Automotive Medicine.

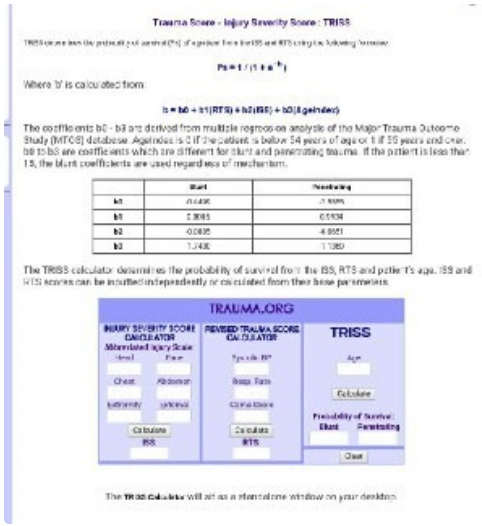
Injuries are ranked on a scale of 1 to 6, with 1 being minor, 5 severe and 6 an unsurvivable injury. This represents the 'threat to life' associated with an injury and is not meant to represent a comprehensive measure of severity. The AIS is not an injury scale, in that the difference between AIS1 and AIS2 is not the same as that between AIS4 and AIS5. There are many similarities between the AIS scale and the Organ Injury Scales of the American Association for the Surgery of Trauma.

AIS Score	Injury
1	Minor
2	Moderate
3	Serious
4	Severe
5	Critical
6	Unsurvivable

Copes WS, Sacco WJ, Champion HR, Bain LW, "Progress in Characterizing Anatomic Injury", In Proceedings of the 33rd Annual Meeting of the Association for the Advancement of Automotive Medicine, Baltimore, MA, USA 205-218

Association for the Advancement of Automotive Medicine
2340 Des Plaines Avenue, Suite 106
Des Plaines
Illinois 60018
USA

6:30:02 PM



Trauma Score - Injury Severity Score : TRISS

TRISS combines the probability of survival (Ps) of a patient from the ISS and RTS using the following formula:

$$Ps = 1 / (1 + e^{-b})$$

Where 'b' is calculated from:

$$b = b0 + b1(RTS) + b2(ISS) + b3(Age\ index)$$

The coefficients b0 - b3 are derived from multiple regression analysis of the Major Trauma Outcome Study (MTOS) database. Age index is 0 if the patient is below 54 years of age or 1 if 55 years and over. b0 to b3 are coefficients which are different for blunt and penetrating trauma. If the patient is less than 15, the blunt coefficients are used regardless of mechanism.

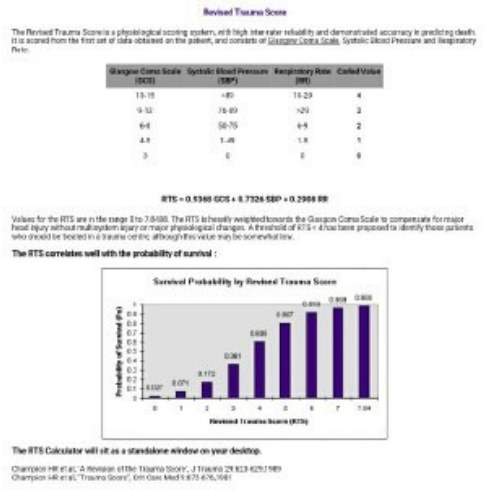
	Blunt	Penetrating
b0	-0.4499	-2.5355
b1	0.8085	0.9934
b2	-0.0835	-0.0651
b3	-1.7430	-1.1360

The TRISS calculator determines the probability of survival from the ISS, RTS and patient's age. ISS and RTS scores can be inputted independently or calculated from their base parameters.

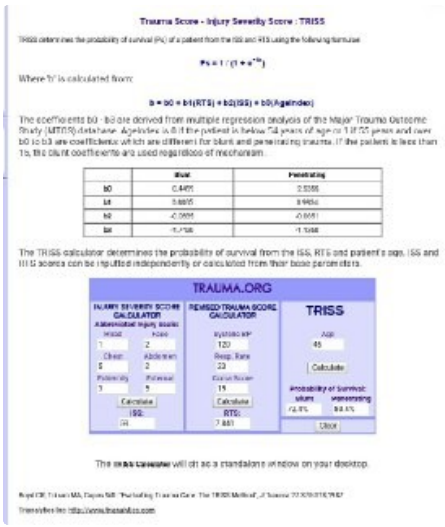
The TRISS Calculator will sit as a standalone window on your desktop.

6:36:52 PM

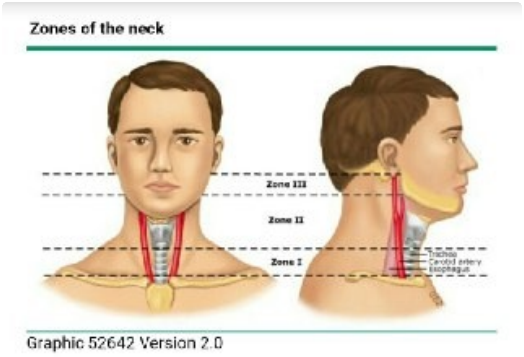
6:39:16 PM



6:43:34 PM



Dr Kamal Deep Fortis Delhi 8:03:48 PM



C-spine injuries are uncommon with penetrating neck trauma, particularly stab wounds, and immobilization can hinder management in some cases by impeding visualization of the airway or by obscuring other injuries. 8:13:30 PM

Dr Kamal Deep Fortis Delhi 11:40:06 PM

Penetrating neck injuries (PNIs) refer to neck injuries resulting from gunshot wounds, stab wounds, or penetrating debris, such as glass or shrapnel, that penetrate the "platysma".

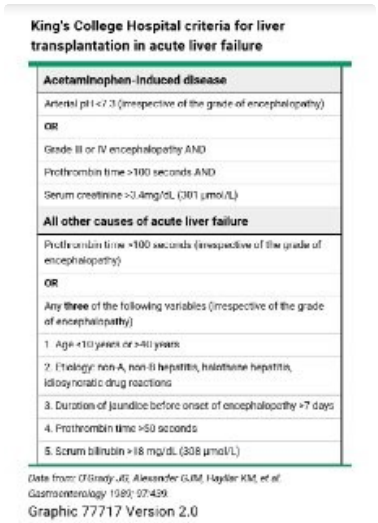
Thursday, March 17, 2016

Dr Kamal Deep Fortis Delhi 12:03:06 AM

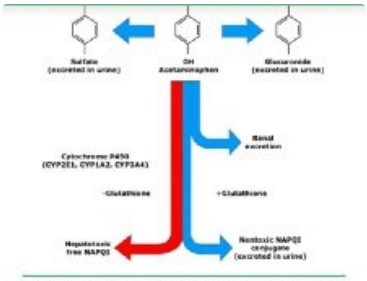
Practice guidelines from the Eastern Association for the Surgery of Trauma (EAST) which state that immobilization of the cervical spine (C-spine) is only necessary when a neurologic deficit is present or a proper physical examination cannot be performed (eg, unconscious patient) and the mechanism is suspicious for a possible spinal cord or column injury

12:05:28 AM

Dr Kamal Deep Fortis Delhi 9:28:15 AM



Dr Kamal Deep Fortis Delhi 10:07:57 AM



At therapeutic doses, 90 percent of acetaminophen is metabolized in the liver to sulfate and glucuronide conjugates that are then excreted in the urine. One-half of the remaining acetaminophen is excreted unchanged in the urine and one-half is metabolized via the hepatic cytochrome P450 (CYP2E1, CYP1A2, CYP3A4 subfamilies) mixed function oxidase pathway to N-acetyl-p-benzoquinoneimine (NAPQI), which is hepatotoxic. With normal doses (blue arrows), NAPQI is rapidly conjugated to hepatic glutathione, forming nontoxic cysteine and mercaptate compounds that are excreted in the urine. With toxic doses (red arrow), the sulfate and glucuronide pathways become saturated, resulting in an increased fraction of acetaminophen being metabolized by cytochrome P450 enzymes. Once glutathione stores are depleted, NAPQI begins to accumulate and hepatic injury ensues.

10:10:09 AM

Stages of acute acetaminophen intoxication

Stage	Hours after ingestion	Clinical features
I	0 to 24 hours	Nausea, vomiting, diaphoresis, pallor, lethargy, and malaise
		some patients remain asymptomatic
		Laboratory studies are typically normal
II	24 to 72 hours	Clinical symptoms resolve
		Hepatotoxicity and extrahepatocytic toxicity become evident
		Right upper quadrant pain, liver enlargement and tenderness
		Elevation of aminotransferase concentrations within 36 hours of ingestion in patients with hepatic injury
		Elevation of prothrombin time (PT) and international normalized ratio of PT
		Oliguria, abnormalities of renal function
		Elevation of serum amylase with or without clinical pancreatitis may occur
III	72 to 96 hours	Liver enzyme and function abnormalities peak (prothrombin time may be >100 and INR >10)
		Resolution of Stage I symptoms
		Jaundice, hepatic encephalopathy, hyperammonemia, bleeding diathesis, hypoglycemia, lactic acidosis
		Renal failure, the incidence of which is related to the severity of intoxication
		Death from multi-organ failure, occurs most commonly in this stage
IV	4 to 14 days	Recovery phase
		symptoms and laboratory values may not normalize for several weeks
		Histologic recovery lags behind clinical recovery and may take up to three months

Graphic 97032 Version 2.0

Dr Kamal Deep Fortis Delhi 2:23:08 PM

Acute liver failure refers to the development of severe acute liver injury with encephalopathy and impaired synthetic function (INR of ≥1.5) in a patient without cirrhosis or preexisting liver disease . While the time course that differentiates acute liver failure from chronic liver failure varies between reports, a commonly used cutoff is an illness duration of <26 weeks.

Acute liver failure is diagnosed by demonstrating all of the following: 2:29:14 PM

●Elevated aminotransferases (often with abnormal bilirubin and alkaline phosphatase levels) (see 'Laboratory test abnormalities' above)
●Hepatic encephalopathy (see 'Neurologic examination' above)
●Prolonged prothrombin time (INR ≥1.5) (see 'Laboratory test abnormalities' above)

Dr Kamal Deep Fortis Delhi 5:23:14 PM

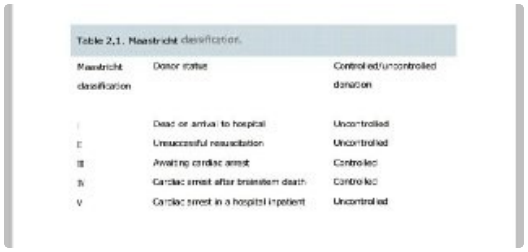


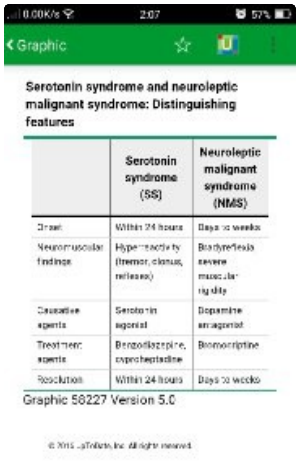
Table 2.1. Maastricht classification.

Maastricht classification	Donor status	Controlled/uncontrolled donation
I	Dead on arrival to hospital	Uncontrolled
II	Unsuccessful resuscitation	Uncontrolled
III	Awaiting cardiac arrest	Controlled
IV	Cardiac arrest after brainstem death	Controlled
V	Cardiac arrest in a hospital inpatient	Uncontrolled

The modified Maastricht classification is used to classify donation after cardiac death patients (DCD). There are five categories (I-V). Most DCD donors come from categories III (anticipated cardiac arrest — controlled) and IV (cardiac arrest in a brain-dead donor — controlled). Categories I (dead on arrival), II (unsuccessful resuscitation) and V (unexpected arrest in ICU patient) describe patients in whom retrieval follows unexpected and irreversible cardiac arrest (uncontrolled DCD). 5:32:10 PM

Sunday, March 20, 2016

Dr Kamal Deep Fortis Delhi 2:07:56 PM



Graphic

Serotonin syndrome and neuroleptic malignant syndrome: Distinguishing features

	Serotonin syndrome (SS)	Neuroleptic malignant syndrome (NMS)
Onset	Within 24 hours	Days to weeks
Neuromuscular findings	Hyperreactivity (tremor, clonus, reflexes)	Bradykinesia severe muscular rigidity
Causative agents	Serotonin agonist	Dopamine antagonist
Treatment agents	Benzodiazepine, cyproheptadine	Bromocriptine
Resolution	Within 24 hours	Days to weeks

Graphic 58227 Version 5.0

Dr Kamal Deep Fortis Delhi 4:09:12 PM

Parkinsonism caused by decrease in DA and increase in cholinergic activity leading to tremors bradykinesia.

Antipsychotic decrease DA in psychotics thus leading to EPS symptoms. Bromocriptine increase DA ,so useful in overdose.

TCA increase biogenic amines NA/Serotonin levels and indirectly facilitates DA Transmission with lot of anticholinergic activity.So bromocriptine will be harmful.

SSRI have no anticholinergic side effects as selective increase in 5ht levels only.. They also don't cause hypotension as they don't inhibit ALPHA adrenergic.as (http://adrenergic.as) DA/ CHOLINERGIC System is less affected side effects are less seen.In overdose bromocriptine will not be useful.Anticholinergic can be given though.

Dr Kamal Deep Fortis Delhi 4:24:54 PM

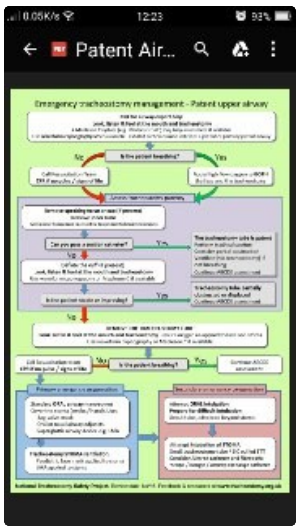
correction.:- anticholinergic not useful but anti serotonorgic agent like cyproheptadine is helpful..

Dr Kamal Deep Fortis Delhi 5:37:35 PM

ICU DELIRIUM & HYPONATREMIA

Monday, March 21, 2016

Dr Kamal Deep Fortis Delhi 12:28:32 AM



Dr Kamal Deep Fortis Delhi 4:55:30 PM

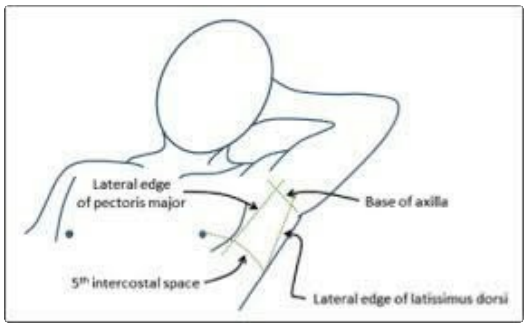
SDD

Pulmonary hypertension 4:55:36 PM

PAOP and normal values 4:55:46 PM

Sengstaken Blackmore tube 5:02:28 PM

Dr Kamal Deep Fortis Delhi 5:25:46 PM



Dr Kamal Deep Fortis Delhi 5:53:36 PM

REPERFUSION THERAPY IN STEMI

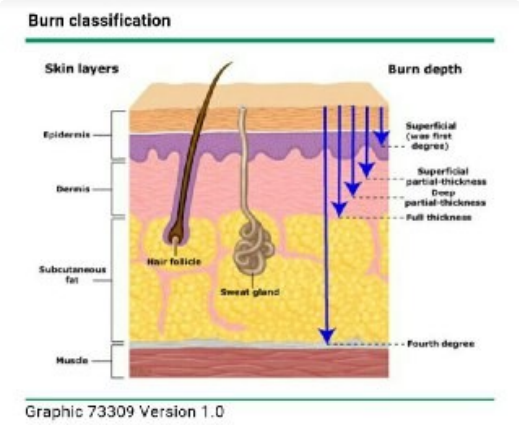
As a result, 2013 American College of Cardiology Foundation/American Heart Association guideline for the management of STEMI recommends use of primary PCI for any patient with an acute STEMI who can undergo the procedure in a timely manner by persons skilled in the procedure [4,5]. Timely is defined as an ideal first medical contact to PCI time of 90 minutes or less for patients transported to PCI-capable hospital or 120 minutes or less for patients who initially arrive at or are transported to a non-PCI capable hospital and are then taken to a PCI-capable hospital. 5:55:10 PM

The 2013 American College of Cardiology Foundation/American Heart Association guideline for the management of STEMI recommends the use of fibrinolytic therapy in patients with symptom onset within 12 hours who cannot receive primary percutaneous coronary intervention within 120 minutes of first medical contact (table 2A-B) [4,5]. The time interval from hospital arrival to initiation of fibrinolytic drug infusion should be less than 30 minute 5:57:57 PM

For patients presenting 12 to 24 hours after symptom onset, the performance of primary PCI is reasonable if the patient has severe heart failure, hemodynamic or electrical instability, or persistent ischemic symptoms. Randomized trials of routine late PCI have shown an improvement in left ventricular function but not in hard clinical end points. This approach is not recommended 6:03:26 PM

Fibrinolytic therapy has generally not improved outcomes in patients presenting at 12 hours or later and is therefore not indicated in those who are stable and asymptomatic. However, fibrinolysis can be considered up to 24 hours after symptom onset if the patient has ongoing or stuttering chest pain and PCI is not available. 6:04:00 PM

Dr Kamal Deep Fortis Delhi 6:29:04 PM



Dr Kamal Deep Fortis Delhi 11:18:44 PM

11:20:28 PM

Burn center referral criteria*

Partial-thickness burns greater than 10% of TBSA
Burns that involve the face, hands, feet, genitalia, perineum, or major joints
Third-degree burns in any age group
Electrical burns, including lightning injury
Chemical burns
Inhalation injury
Burn injury in patients with preexisting medical disorders that could complicate management, prolong recovery, or affect mortality
Any patient with burns and concurrent trauma (such as fractures) in which the burn injury poses the greatest risk for morbidity or mortality. In such cases, if the trauma poses the greater immediate risk, the patient may be stabilized initially in a trauma center before being transferred to a burn unit. Physician judgment will be necessary in such situations and should be in concert with the regional medical control plan and triage protocols.
Burned children in hospitals without qualified personnel or equipment for the care of children
Burn injury in patients who will require special social, emotional, or rehabilitative intervention

TBSA: total body surface area
* A burn center may treat adults, children, or both. Burn injuries that should be referred to a burn center include any of the criteria listed.

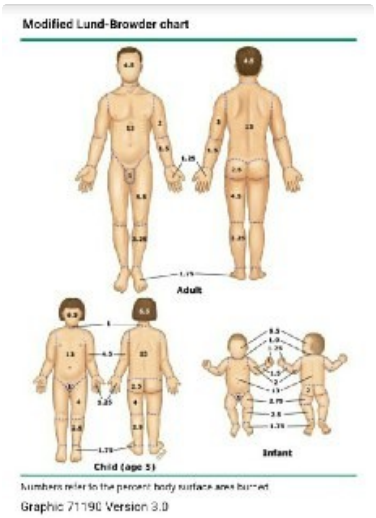
Dr Kamal Deep Fortis Delhi 11:37:57 PM

Lund-Browder — The Lund-Browder chart is the most accurate method for estimating TBSA for both adults and children. It

takes into account the relative percentage of body surface area affected by growth (figure 2) [4,11,12]. Children have proportionally larger heads and smaller lower extremities, so the percentage BSA is more accurately estimated using the Lund-Browder chart

Rule of Nines — For adult assessment, the most expeditious method to estimate TBSA in adults is the "Rule of Nines" [13,14]: 11:38:18 PM

●Each leg represents 18 percent TBSA
●Each arm represents 9 percent TBSA
●The anterior and posterior trunk each represent 18 percent TBSA
●The head represents 9 percent TBSA



11:39:54 PM

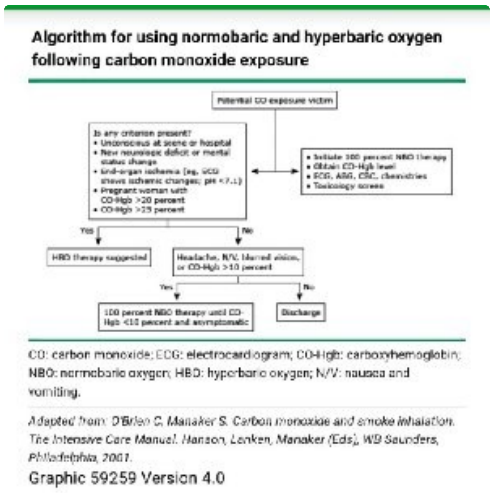
Superficial burns are not included in the TBSA burn assessment. The location of partial-thickness and full-thickness burned areas are recorded on a burn diagram 11:39:54 PM

Tuesday, March 22, 2016

Dr Kamal Deep Fortis Delhi 2:11:50 AM

Intoxicated trauma patients with head injuries have not been clearly shown to have worse outcomes than those who are not intoxicated at time of injury, once adjustments for injury severity are made. The biggest difference shown is that they are more likely to have unsuspected injuries. In contrast, burn patients who are intoxicated at time of injury have a sixfold increase in mortality compared with non-intoxicated patients with similar injury.

2:19:20 AM



CO: carbon monoxide; ECG: electrocardiogram; CO-Hgb: carboxyhemoglobin; NBO: normobaric oxygen; HBO: hyperbaric oxygen; N/V: nausea and vomiting.

Adapted from: O'Brien C, Manaker S. Carbon monoxide and smoke inhalation. The Intensive Care Manual. Hanson, Lenken, Manaker (Eds), WB Saunders, Philadelphia, 2001.

Graphic 59259 Version 4.0

Dr Kamal Deep Fortis Delhi 9:36:24 AM

According to the American Burn Association's practice guidelines, any patient with greater than 15 percent total body surface area (TBSA) nonsuperficial burns should receive formal fluid resuscitation

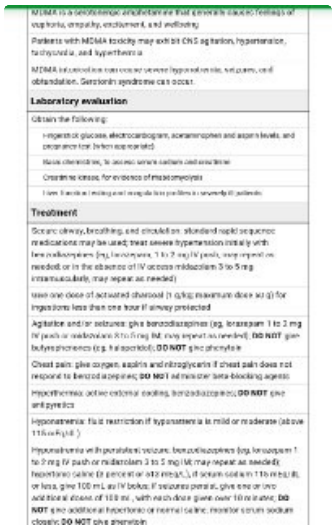
Dr Kamal Deep Fortis Delhi 2:36:20 PM

MDMA use can lead to hyponatremia due to a marked increase in fluid intake and in some patients, persistent secretion of antidiuretic hormone that slows the rate of water excretion.

Seizures that occur in the setting of drug intoxication typically result from an altered balance of excitatory and inhibitory neurotransmitters. This mechanism is distinct from that of patients with a known seizure disorder, who generally have electrical instability in a discrete area of brain tissue amenable to treatment with sodium channel blocking agents, such as phenytoin. 2:45:04 PM

The mechanism of MDMA-induced seizures makes phenytoin an illogical treatment choice 2:45:34 PM

2:46:16 PM



MDMA is a sympathomimetic amphetamine that produces classic feelings of euphoria, empathy, excitement, and wellbeing
Patients with MDMA toxicity may exhibit CNS agitation, hypertension, tachycardia, and hyperthermia
MDMA intoxication can cause severe hyponatremia, seizures, and obtundation. Serotonin syndrome can occur.
Laboratory evaluation
Obtain the following:
Fingerstick glucose, electrocardiogram, acetaminophen and aspirin levels, and pregnancy test (when age-indicated)
Basic chemistries, to assess serum sodium and creatinine
Creatine kinase, for evidence of rhabdomyolysis
Liver function testing and coagulation profile in severely ill patients
Treatment
Secure airway, breathing, and circulation; standard rapid sequence medications may be used; treat severe hypertension initially with benzodiazepines (eg, lorazepam 1 to 2 mg IV push, midazolam 5 to 10 mg intramuscularly, may repeat as needed)
Give one dose of activated charcoal (1 g/kg; maximum dose 50 g) for ingestions less than one hour if airway protected
Agitation and/or seizures: give benzodiazepines (eg, lorazepam 1 to 2 mg IV push or midazolam 5 to 5 mg IM; may repeat as needed); **DO NOT** give butyrophenones (eg, haloperidol); **DO NOT** give phenytoin
Chest pain: give oxygen, aspirin and nitroglycerin if chest pain does not respond to benzodiazepines; **DO NOT** administer beta-blocking agents
Hyperthermia: active external cooling, benzodiazepines; **DO NOT** give antipyretics
Hyponatremia: fluid restriction if hyponatremia is mild or moderate (above 115 mEq/L)
Hyponatremia with generalized seizure: benzodiazepines (eg, lorazepam 1 to 2 mg IV push or midazolam 5 to 5 mg IM; may repeat as needed); hypertonic saline (3 percent or 513 mEq/L), if serum sodium 115 mEq/L or less, give 100 mL as IV bolus; if seizures persist, give one or two additional doses of 100 mL, with each dose given over 10 minutes; **DO NOT** give additional hypertonic or normal saline; monitor serum sodium closely; **DO NOT** give phenytoin

The number needed to treat (NNT) is an epidemiological measure used in communicating the effectiveness of a health-care intervention, typically a treatment with medication. The NNT is the average number of patients who need to be treated to prevent one additional bad outcome (e.g. the number of patients that need to be treated for one to benefit compared with a control in a clinical trial). It is defined as the inverse of the absolute risk reduction. It was described in 1988.[1] The ideal NNT is 1, where everyone improves with treatment and no one improves with control. The higher the NNT, the less effective is the treatment

2:53:38 PM

NNT is the statistical inverse of the absolute risk reduction i.e. 1/absolute risk reduction

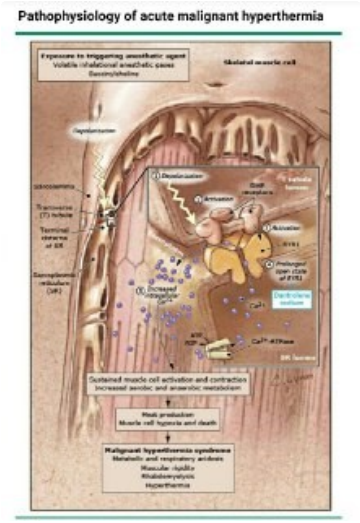
2:54:10 PM

Jehovah's Witnesses believe that the Bible prohibits ingesting blood and that Christians should not accept blood transfusions or donate or store their own blood for transfusion

3:01:36 PM

Dr Kamal Deep Fortis Delhi

3:16:54 PM



Malignant hyperthermia — The majority of MHS patients have mutations encoding for abnormal RYR1 or DHP receptors; exposure to triggering agents in these patients may lead to unregulated passage of calcium from the sarcoplasmic reticulum into the intracellular space, leading to an acute MH crisis (figure 1) [11-20]. The accumulation of myoplasmic calcium causes sustained muscle contraction. 3:18:22 PM

Accelerated levels of aerobic metabolism sustain the muscle for a time, but produce carbon dioxide and cellular acidosis, and deplete oxygen and adenosine triphosphate [21-23]. This causes the early signs of MH: hypercarbia and mixed respiratory/metabolic acidosis. A change to anaerobic metabolism worsens acidosis with the production of lactate. Once energy stores are depleted, rhabdomyolysis occurs and results in hyperkalemia and myoglobinuria. Hyperkalemia from rhabdomyolysis may occur early in muscular patients.

Over time, sustained contraction generates more heat than the body is able to dissipate. Marked hyperthermia occurs minutes to hours following the initial onset of symptoms. In some cases, core body temperature may rise 1ºC every few minutes. Severe hyperthermia (up to 45ºC [113ºF]) leads to a marked increase in carbon dioxide production, and increased oxygen consumption can cause widespread vital organ dysfunction. Severe hyperthermia is associated with the development of disseminated intravascular coagulation, a poor prognostic indicator and often terminal event [24].

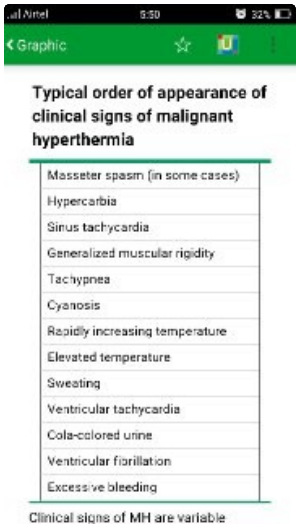
Dr Kamal Deep Fortis Delhi 5:13:54 PM

Zolpidem 10 mg is effective in treating insomnia when used intermittently no fewer than three and no more than five pills per week for a period of 12 weeks.[26] The 15-mg zolpidem dosage provided no clinical advantage over the 10-mg zolpidem dosage

Dr Kamal Deep Fortis Delhi 6:11:42 PM

The most common initial sign of an MH crisis is an unexpected rise in end-tidal carbon dioxide (ETCO2), which is difficult to decrease as minute ventilation is increased.

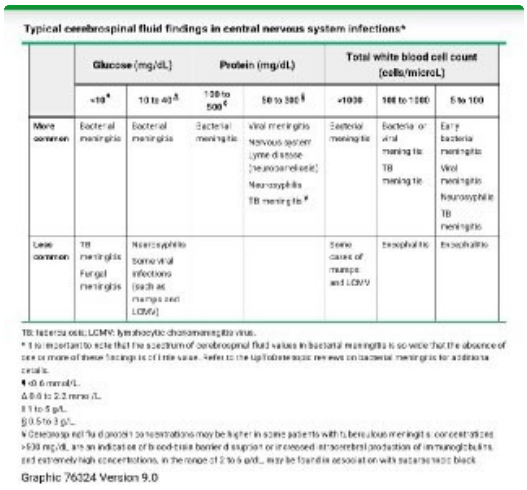
immediate administration of dantrolene (Grade 1A). The initial dose is 2.5 mg/kg intravenous (IV), to be given rapidly through a large-bore IV. The end-tidal carbon dioxide (ETCO2) typically normalizes within minutes; subsequent bolus doses of 1 mg/kg (up to 10 mg/kg) may be needed if signs of MH have not abated, most often in muscular males. 6:11:42 PM



Clinical signs of MH are variable

6:11:58 PM

Dr Kamal Deep Fortis Delhi 6:44:16 PM



Wednesday, March 23, 2016

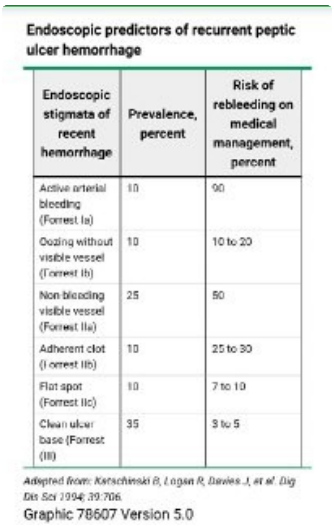
Dr Kamal Deep Fortis Delhi 1:14:24 AM

Stroke syndromes

Dr Kamal Deep Fortis Delhi 2:17:30 AM

().pdf 423 KB
Download ()

Dr Kamal Deep Fortis Delhi 6:14:43 PM



Endoscopic predictors of recurrent peptic ulcer hemorrhage

Endoscopic stigmata of recent hemorrhage	Prevalence, percent	Risk of rebleeding on medical management, percent
Active arterial bleeding (Forrest Ia)	10	90
Oozing without visible vessel (Forrest Ib)	10	10 to 20
Non-bleeding visible vessel (Forrest IIa)	25	50
Adherent clot (Forrest IIb)	10	25 to 30
Flat spot (Forrest IIc)	10	7 to 10
Clean ulcer base (Forrest III)	35	3 to 5

Adapted from: Katschinski B, Logan R, Davies J, et al. Dig Dis Sci 1994; 39:706.
Graphic 78607 Version 5.0

The Blatchford score (also known as the Glasgow Blatchford score), unlike the Rockall score, does not take endoscopic data into account and thus can be used when the patient first presents 6:29:26 PM

Thursday, March 24, 2016

Dr Kamal Deep Fortis Delhi 7:29:30 PM

We believe that a safe starting point for most critically ill patients is approximately 8 to 10 kcal/kg per day [62]. Attempting to achieve a goal of 25 to 30 kcal/kg per day after one week is reasonable for most stable patients. A goal of 35 kcal/kg per day is an acceptable goal if weight gain is desired in a relatively stable patient; weight gain should not be attempted until the patient is stable and in a lower inflammatory state. We keep the caloric goal at 25 kcal/kg per day or less if extubation is imminent.

Protein — Guidelines indicate that protein requirements increase as illness becomes more severe. This is based solely on nitrogen excretion and not on outcomes studies except in burns. Practice is to give patients with only mild to moderate illness 0.8 to 1.2 g/kg protein per day. Critically ill patients are generally prescribed 1.2 to 1.5 g/kg per day and patients with severe burns may benefit from as much as 2 g/kg per day. 7:33:12 PM

Standard enteral nutrition delivers 49 to 53 percent of calories as carbohydrate and 29 to 30 percent of calories as fat. In contrast, low carbohydrate/high fat enteral nutrition typically delivers 28 to 40 percent of calories as carbohydrate and 40 to 55 percent of calories as fat. High carbohydrate/low fat enteral nutrition delivers only 15 percent of calories as fat. 7:41:52 PM

Standard — The following characteristics are typical of standard enteral nutrition: 7:42:43 PM

●Isotonic to serum
●Caloric density of approximately 1 kcal/mL
●Lactose-free
●Intact (nonhydrolyzed) protein content of about 40 g/1000 mL (40 g/1000 kcal)
●Nonprotein calorie to nitrogen ratio of approximately 130
●Mixture of simple and complex carbohydrates
●Long-chain fatty acids (although some are now including medium-chain and omega-3 fatty acids)
●Essential vitamins, minerals, and micronutrients

The standard composition of concentrated enteral nutrition is similar to that of standard enteral nutrition, except that it is mildly hyperosmolar to serum and has a caloric density of 1.2, 1.5, or 2.0 kcal/mL 7:43:12 PM

Current adult formulations rarely exceed approximately 750 mOsm/L and are rarely the primary cause of diarrhea 7:43:26 PM

Predigested enteral nutrition usually has a caloric density of 1 or 1.5 kcal/mL 7:44:52 PM

Most guidelines suggest high-protein enteral nutrition (1.2 to 2 g per kg of ideal body weight per day) in patients with critical illness based upon observational studies that report improved mortality associated with this strategy 7:46:32 PM

Standard enteral nutrition delivers an intact (nonhydrolyzed) protein content of about 40 g/1000 mL, with a nonprotein calorie to nitrogen ratio of approximately 130. In contrast, low protein enteral nutrition delivers a protein content of 15 to 35 g/1000 mL, with a nonprotein calorie to nitrogen ratio of 173 to 391. Other features of low protein enteral nutrition include high caloric density (in order to reduce the volume delivered), fat content, and osmolarity, accompanied by a restricted amount of electrolytes (especially sodium, potassium, and phosphate). 7:48:24 PM

As a result, it seems reasonable to provide vitamins and trace elements to critically ill patients, regardless of the type of nutrition support that they are receiving [1]. The optimal mixture of vitamins and trace elements is yet to be determined, such that daily recommended allowances should not be routinely exceeded. 7:51:00 PM

In practice, it is generally considered acceptable that enteral feeding be initiated in critically ill patients at a rate of 10 to 30 mL/hr (for standard enteral formulations), so called "trophic" feeding, for six days and then incrementally increased to the target rate 7:53:14 PM

the low-volume feeding group had less vomiting, smaller gastric residual volumes, lower mean plasma glucose levels, and less constipation. The low-volume feeding group also required fewer prokinetic agents and less insulin 8:07:22 PM

Based on these findings, there is increasing consensus that the routine checking of gastric residual volumes is unnecessary in asymptomatic patients receiving tube feedings, and may inappropriately hamper calorie delivery. Gastric residuals should be measured if the patient exhibits a clinical change, such as abdominal pain, abdominal distension, or deterioration in hemodynamics or overall status. 8:08:49 PM

ESPEN endorsed recommendations: Nutritional therapy in major burnsq 8:14:58 PM

8:15:02 PM

All feeding products consist of only 70 to 80 percent water. As a result, they are unable to meet patients' normal water requirements alone (enteral nutrition providing 25 kcal/kg with a 1 kcal/mL formula provides an average of only 20 mL/kg of water). This may be beneficial for patients who require fluid restriction, but most patients require another source of water, and feeding tubes must be flushed regularly to avoid clogging. 8:18:52 PM

Motility agents may help achieve the desired rate of feeding in a subgroup of patients with poor gastric emptying. However, given 8:21:08 PM

the uncertainty of what constitutes an optimal feeding rate and the lack of evidence that motility agents improve clinical outcomes, we believe that use of motility agents as a preventive measure to prevent pneumonia is not warranted.

Friday, March 25, 2016

Dr Kamal Deep Fortis Delhi 10:30:06 AM

Diagnostic criteria for transfusion-related acute lung injury (TRALI) and possible TRALI

	TRALI	Possible TRALI
Acute lung injury (ALI)/acute respiratory distress syndrome (ARDS)	• Acute onset (during or within six hours of transfusion) • Hypoxemia* • Bilateral infiltrates on frontal chest radiograph • No evidence of circulatory overload/left atrial hypertension • No pre-existing ALI/ARDS before transfusion	Same as for TRALI
ALI/ARDS risk factor^ at time of transfusion	Must be absent	Must be present

The diagnostic criteria for TRALI and possible TRALI share the following features: acute onset of hypoxemia, bilateral infiltrates on frontal chest radiograph, and absence of circulatory overload as the primary etiology of respiratory insufficiency. For a diagnosis of TRALI to be made, all of these features must be present. In addition, there should be no preexisting ALI/ARDS risk factors at the time of transfusion. If ALI/ARDS risk factors are present, the diagnostic terminology "possible TRALI" is appropriate. 10:30:26 AM

Though TRALI has been associated with virtually all blood products, high-plasma-volume components such as plasma, apheresis platelet concentrates, and whole blood have been consistently shown to carry the greatest risk per component or per transfusion episode 10:33:31 AM

10:45:02 AM

Feature	TRALI	TACO
Body temperature	Fever may be present	Unchanged
Blood pressure	Hypotension may be present	Hypertension may be present
Respiratory symptoms	Acute dyspnea	Acute dyspnea
Neck veins	Unchanged	May be distended
Auscultation	Rales	Rales and S3 may be present
Chest radiograph	Diffuse bilateral infiltrates	Diffuse bilateral infiltrates
Ejection fraction	Normal	Decreased
PAOP	Most often 18 mmHg or less	Greater than 18 mmHg
Pulmonary edema fluid	Exudate	Transudate
Fluid balance	Neutral or negative	Positive
Response to diuretics	Inconsistent	Significant improvement
White cell count	Transient leukopenia may be present	Unchanged
BNP	<250 pg/mL	>1200 pg/mL

TRALI: transfusion related acute lung injury; TACO: transfusion associated circulatory overload; PAOP: pulmonary artery

Dr Kamal Deep Fortis Delhi 12:30:34 PM

Pneumonia and surgical-site infections were the most common, each accounting for 22 percent of all infections, followed by gastrointestinal, urinary tract, and primary bloodstream infections. Clostridium difficile and Staphylococcus aureus were the most common pathogens identified. Device-associated infections (ie, ventilator-associated pneumonia, catheter-related urinary tract infection, and catheter-related bloodstream infection) together accounted for 26 percent of all infections.

An important limitation of AHD is that it does not reduce the incidence of Clostridium difficile colitis [27]. This reflects the lack of activity of alcohol-based hand rubs against spore-forming bacteria. 12:36:36 PM

good handwashing after glove removal is essential. 12:40:41 PM

Masks should not be confused with particulate respirators that are used to prevent transmission by airborne droplet nuclei of infectious agents such as M. tuberculosis. 12:41:21 PM

Healthcare workers within six to ten feet of patients on droplet precautions should wear a facemask 12:51:32 PM

Airborne precautions should be used in the care of patients with suspected or confirmed tuberculosis (TB), measles, varicella, smallpox and SARS. TB is principally transmitted in hospitals by droplet nuclei 12:52:08 PM

Although SARS is transmitted predominantly by droplet spread and direct contact, airborne transmission can also occur. As a 12:52:28 PM

result, the CDC recommends contact and airborne precautions for this condition

Patients placed on airborne isolation precautions should be placed in an airborne infection isolation room (AIIR) [5]. The AIIR should be a private room with negative air pressure and a minimum of 6 to 12 air changes per hour. Doors to the isolation rooms must remain closed, and all persons entering must wear a respirator with a filtering capacity of 95 percent that allows a tight seal over the nose and mouth. 12:53:32 PM

Immunocompromised patients — Special protective environments are used for patients undergoing hematopoietic cell transplantation to minimize their exposure to invasive fungal infections such as aspergillosis. These include HEPA filtration of incoming air, positive room air pressure relative to corridors, directed room air flow, ventilation systems that provide at least 12 air changes per hour, dust control measures and the prohibition of flowers and potted plants in patient rooms. 12:54:28 PM

Dr Kamal Deep Fortis Delhi 1:49:38 PM

Dr Kamal Deep Fortis Delhi 7:40:46 PM

Despite this finding, an absent N20 was still 100 percent predictive of a dismal outcome, ie, no better than persistent coma [64].

The presence of the N20 responses, however, does not assure a good outcome

SSEPs are the best validated and most reliable of the ancillary tests currently available for clinical use. 7:41:26 PM

ANCILLARY TESTING — Several tests have been studied in the period after anoxic injury; these are often helpful at arriving at an earlier prognostic determination than would be possible with clinical testing alone. Of the available tests, bilaterally absent somatosensory evoked responses at 24 to 72 hours appears to be most useful to identify those with a poor prognosis. While very specific, these signs are not very sensitive for poor neurologic outcome. 7:45:43 PM

Two systematic reviews have concluded that two clinical criteria have been found to be 100 percent specific for poor outcome [36,47]: 7:47:52 PM

Absent or extensor motor response on day three
Absent pupillary or corneal reflexes on day three

Myoclonic status epilepticus — Persistent bilaterally synchronous myoclonus in the face, limbs, and axial musculature is usually associated with in-hospital death or poor outcome, even in patients with intact brainstem reflexes or some motor response 7:48:28 PM

EEG categories can be crudely classified into malignant and benign/types. The former includes complete or near complete suppression, burst-suppression, generalized periodic complexes, low voltage output pattern (≤10 μV), intermittent or continuous seizures, nonreactive to stimuli and the alpha-theta pattern [54,71]. In one series, these malignant EEG findings were associated with a higher mortality (91 versus 54 percent) compared with those who did not have these findings [71]. Of these findings, complete, generalized suppression (<20 microvolts) is the most specific for poor outcome; other patterns are less reliable for prognosis 7:50:48 PM

Further studies are needed before NSE or other biological markers can be used prognostically in cardiac arrest patients treated with hypothermia. 7:52:12 PM

Biomarkers, eg, neuronal specific enolase, appear to be promising indicators of poor outcome, but have uncertain predictive value at least for those patients treated with hypothermia. Further research is needed to better define their cut-off values and sensitivity 7:53:20 PM

7:54:39 PM

Clinical parameters associated with an unfavorable prognosis

Clinical parameters	Unfavorable prognosis
Duration of anoxia	>8-10 minutes
Duration of CPR	>30 minutes
Pupillary light reaction	Absent on day 3
Motor response to pain	Absent on day 3
Brainstem reflexes	Absent
Blood glucose on admission	>300 mg/dl
Glascow coma score on day 3	<5
GPCS on day 3	<22

Graphic 69852 Version 2.0

Dr Kamal Deep Fortis Delhi 9:11:58 PM

Patients with HF with preserved ejection fraction (HFpEF) presenting with hypotension should not receive inotropes and may require a vasopressor in addition to diuretic therapy. Patients who develop hypotension with dynamic left ventricular outflow obstruction are treated with beta blocker therapy and gentle hydration if pulmonary edema is not present.

PULMONARY EDEMA 9:17:22 PM

Saturday, March 26, 2016

Dr Kamal Deep Fortis Delhi 12:05:10 AM

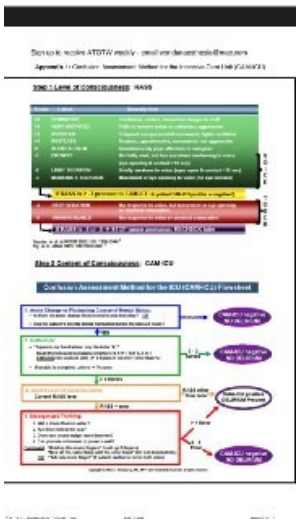
fluconazole-resistant organism, such as C. glabrata or C. krusei.

C. glabrata and C. krusei 12:06:08 AM

●An echinocandin is preferred over amphotericin B for treatment of candidemia due to C. glabrata and C. krusei. Voriconazole is approved for this indication, but there is likely to be cross-resistance between fluconazole and voriconazole among C. glabrata isolates. This cross-resistance does not occur with C. krusei.

Voriconazole is recommended as oral step-down therapy for patients with C. krusei, as this species is intrinsically resistant to fluconazole. For other Candida isolates, voriconazole does not offer a clear advantage compared with fluconazole. 12:07:56 AM

Dr Kamal Deep Fortis Delhi 1:26:36 AM



Dr Kamal Deep Fortis Delhi 8:15:34 AM

Antifungal Therapy

Dr Kamal Deep Fortis Delhi 8:37:20 AM

Peripheral methods of monitoring temperature (tympanic membrane, temporal artery, axillary, and oral thermometry) do not accurately reflect core body temperature as measured by central methods (pulmonary artery catheter, urinary bladder, esophageal, and rectal thermometry) and are less sensitive

Dr Kamal Deep Fortis Delhi 3:38:40 PM

For critically ill patients who have risk factors for stress ulceration, we recommend prophylaxis with agents that suppress gastric acid (eg, PPI, H2 blockers, antacid) rather than no prophylaxis (Grade 1B). Risk factors include the following: mechanical ventilation for more than 48 hours, coagulopathy, GI ulceration or bleeding within the past year, traumatic brain injury, traumatic spinal cord injury, severe burns >35 percent of the body surface area, or two or more minor risk factors

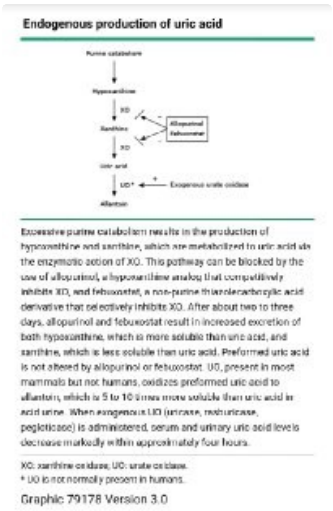
3:40:16 PM

●In critically ill patients, H2 blockers, PPIs, and antacids reduce the frequency of overt GI bleeding in critically ill patients compared to placebo or no prophylaxis. PPIs may be more effective at reducing the rate of GI bleeding but trials comparing individual agents are limited by methodologic flaws. (See 'Efficacy' above.)

●Prophylactic agents that increase gastric pH (PPI, H2 blockers, antacids) may increase the frequency of nosocomial pneumonia, compared to prophylactic agents that do not alter gastric pH (sucralfate). PPIs and H2 blockers may also be associated with an increased risk of clostridium difficile infection. This risk should not

preclude the administration of GI prophylaxis to critically ill patients, when it is indicated. (See 'Potential harms' above.)

Dr Kamal Deep Fortis Delhi 4:23:36 PM



Dr Kamal Deep Fortis Delhi 7:06:06 PM

Term pregnancy is 37 to 41 weeks, with early term being 37 and 38 weeks, full term 39 and 40 weeks, and late term 41 weeks. After 41 weeks, it is known as post term. Babies born before 37 weeks are preterm and are at higher risk of health problems such as cerebral palsy.[1] Delivery before 39 weeks by labor induction or caesarean section is not recommended unless required for other medical reasons

Dr Kamal Deep Fortis Delhi 8:07:36 PM

DIAGNOSIS — International guidelines generally agree that the diagnosis of preeclampsia should be made in a previously normotensive woman with new onset of hypertension and either proteinuria or end-organ dysfunction after 20 weeks of gestation [4,126-128]. Criteria for diagnosis are [4]:

•Systolic blood pressure ≥140 mmHg or diastolic blood pressure ≥90 mmHg, and
•Proteinuria ≥0.3 grams in a 24-hour urine specimen or protein:creatinine ratio ≥0.3, or
•Signs of end-organ dysfunction (platelet count <100,000/microliter, serum creatinine >1.1 mg/dL or doubling of the serum creatinine, elevated serum transaminases to twice normal concentration)
Severe hypertension and signs/symptoms of end-organ injury are considered the severe spectrum of the disease

If systolic blood pressure is ≥160 mmHg or diastolic blood pressure is ≥110 mmHg, confirmation within minutes is sufficient. 8:08:02 PM

Preeclampsia is generally classified as having severe features if any of the following are present in a woman with preeclampsia (table 2) [3,4,126-129]: 8:08:34 PM

●Severe hypertension (systolic blood pressure ≥160 mm Hg or diastolic blood pressure ≥110 mm Hg)
●Signs/symptoms of end-organ injury (thrombocytopenia, impaired liver function, progressive renal insufficiency, pulmonary edema, new onset cerebral or visual disturbances)

Observational data support the hypothesis that placental underperfusion, hypoxia, and/or ischemia may lead to release of circulating antiangiogenic factors (soluble fms–like tyrosine kinase [sFlt-1], soluble endoglin [sEng]) and other substances that can cause widespread maternal systemic endothelial dysfunction (increased vascular permeability, vasoconstriction, activation of coagulation system, microangiopathic hemolysis), resulting in hypertension, proteinuria, and the other clinical manifestations of preeclampsia [31]. The severity of the disease is influenced primarily by maternal and pregnancy-specific factors, but paternal and environmental factors may also play a role 8:12:58 PM

Dr Kamal Deep Fortis Delhi 9:49:36 PM

Acute liver failure refers to the development of severe acute liver injury with encephalopathy and impaired synthetic function (INR of ≥1.5) in a patient without cirrhosis or preexisting liver disease [2,3]. While the time course that differentiates acute liver failure from chronic liver failure varies between reports, a commonly used cutoff is an illness duration of <26 weeks

Dr Kamal Deep Fortis Delhi 10:23:51 PM

American College of Gastroenterology Guidelines: Liver disease in the pregnant patient

Use of gestational age of the pregnancy is the best guide to the differential diagnosis of liver disease in the pregnant woman
Hyperemesis gravidarum should be considered in the differential diagnosis of abnormal liver tests presenting in the first trimester
Cholestasis of pregnancy is common, and should be considered in the differential diagnosis of abnormal liver tests presenting initially in the second trimester. Affected pregnancies are at increased risk for prematurity and stillbirth, and early delivery should be considered when possible
HELLP (hemolysis, elevated liver tests, low platelets) syndrome and acute fatty liver of pregnancy should be considered in the differential diagnosis of abnormal liver tests in the second half of pregnancy, usually in the third trimester
Patients with acute fatty liver of pregnancy have true hepatic dysfunction, and may, or may not, have signs of pre-eclampsia and HELLP syndrome
Consider viral or drug-induced hepatitis, gallstone disease, or malignancy in the differential diagnosis of abnormal liver tests in any of the trimesters of pregnancy
Chronic hepatitis B or C poses a risk of transmission to the offspring

Dr Kamal Deep Fortis Delhi 11:19:16 PM

Child-Pugh classification of severity of cirrhosis

Parameter	Points assigned		
	1	2	3
Ascites	Absent	Slight	Moderate
Bilirubin	<2 mg/dL (<34.2 micromol/L)	2 to 3 mg/dL (34.2 to 51.3 micromol/L)	>3 mg/dL (>51.3 micromol/L)
Albumin	>3.5 g/dL (35 g/L)	2.8 to 3.5 g/dL (28 to 35 g/L)	<2.8 g/dL (<28 g/L)
Prothrombin time			
Seconds over control	<4	4 to 6	>6
INR	<1.7	1.7 to 2.3	>2.3
Encephalopathy	None	Grade 1 to 2	Grade 3 to 4

Modified Child-Pugh classification of the severity of liver disease according to the degree of ascites, the serum concentrations of bilirubin and albumin, the prothrombin time, and the degree of encephalopathy. A total Child-Turcotte-Pugh score of 5 to 6 is considered Child-Pugh class A (well-compensated disease); 7 to 9 is class B (significant functional compromise); and 10 to 15 is class C (decompensated disease). These classes correlate with one- and two-year patient survival: class A: 100 and 85 percent; class B: 80 and 60 percent; and class C: 45 and 35 percent.

INR: international normalized ratio

Dr Kamal Deep Fortis Delhi 11:35:16 PM

AFLP VS HELLP

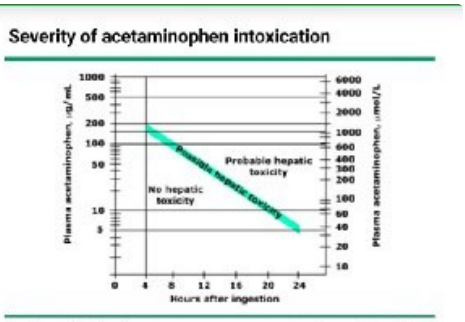
The major other condition that must be excluded is the HELLP syndrome, which is characterized by hemolysis, elevated liver enzymes, and a low platelet count. There is a large clinical overlap between acute fatty liver of pregnancy and HELLP syndrome, and it may be difficult, even impossible, to differentiate them. However, evidence of hepatic insufficiency such as hypoglycemia or encephalopathy and abnormalities in coagulation studies is more consistent with acute fatty liver of pregnancy. In one series of 46 women who developed liver disease during pregnancy severe enough to require admission to a liver failure unit, 70 percent had acute fatty liver and 15 percent had HELLP [10]. Most of the remaining patients had liver disease that was unrelated to pregnancy.

Acute alcohol ingestion — Acute alcohol ingestion is NOT a risk factor for hepatotoxicity and may even be protective by competing with acetaminophen for CYP2E1 and, thereby, decreasing the amount of NAPQI produced

11:48:47 PM

Sunday, March 27, 2016

Dr Kamal Deep Fortis Delhi 12:12:54 AM



The Rumeck-Matthews nomogram summarizes the relationship between plasma acetaminophen concentration (in µg/mL or µmol/L), the time after drug ingestion, and the risk of hepatic toxicity. The thick diagonal line of possible hepatic toxicity represents a 25 percent likelihood of disease. A relatively low level (such as 10 µg/mL) is safe soon after ingestion, but associated with appreciable risk at 24 hours since it reflects a high initial load which has now distributed into the tissues.

Adapted from Rumack BH, Matthews H. Pediatrics 1975; 55:873.

Treat with N-acetylcysteine (NAC) if: 12:17:08 AM
Serum APAP concentration drawn at 4 hours or more after a single acute ingestion is above the "treatment" line of the treatment nomogram for APAP poisoning
Serum APAP concentration is unavailable or will not return within 8 hours of time of ingestion and APAP ingestion is suspected
Time of ingestion is unknown and serum APAP level is greater than 10 mcg/mL (66 µmol/L)
There is evidence of ANY hepatotoxicity with a history of APAP ingestion
Patient reports or clinician suspects repeated excessive APAP ingestions, patient has risk factors for APAP-induced hepatotoxicity, and the serum APAP concentration is greater than 10 mcg/mL (66 µmol/L)

Acute liver failure is diagnosed by demonstrating all of the following: 12:31:45 AM

●Elevated aminotransferases (often with abnormal bilirubin and alkaline phosphatase levels)
●Hepatic encephalopathy
●Prolonged prothrombin time (INR ≥1.5)

Dr Kamal Deep Fortis Delhi 1:01:04 AM

COLONY STIMULATING FACTORS — Colony stimulating factors (CSFs; also known as myeloid growth factors or hematopoietic growth factors), such as granulocyte and granulocyte-macrophage colony stimulating factors (G-CSF and GM-CSF), are not recommended for routine use in patients with established fever and neutropenia. The Infectious Diseases Society of America (IDSA) guidelines recommend against their use for all patients with established fever and neutropenia [1], whereas the American Society of Clinical Oncology guidelines state that their use can be "considered" for patients at high risk for infection-associated complications or who have prognostic factors that are predictive of a poor clinical outcome [80]. As the available data do not show a clear benefit, we agree with the IDSA guidelines and do not use CSFs in patients with established fever and neutropenia. These issues are discussed in greater detail separately

Dr Kamal Deep Fortis Delhi 2:15:00 AM

Short Bowel Syndrome

The proximal small intestine is primarily responsible for the absorption of proteins, carbohydrates, and fats. However, the ileum is able to compensate for absorption of these nutrients following loss of the proximal small intestine.

In contrast, resection of the ileum may be relatively more problematic than loss of the proximal small intestine since the jejunum is less able to compensate for the specialized absorptive functions of the ileum.

●The ileum is the selective site for absorption of vitamin B12 and bile salts. Resection of more than 60 cm of ileum is associated with malabsorption of intrinsic factor bound vitamin B12 [8]. (See "Etiology and clinical manifestations of vitamin B12 and folate deficiency".)
The liver is able to compensate for the loss of bile salts when less than 100 cm of ileum has been resected. In such cases, however, patients may develop diarrhea due to excessive presentation of bile salts in the colon, which can stimulate a secretory diarrhea (called cholerheic enteropathy). Resection of more than 100 cm of ileum is associated with insufficient bile salt concentrations in the duodenum for optimal fat absorption, leading to fat and fat soluble vitamin malabsorption.
●Unabsorbed lipids reaching the ileum cause delay in gastric emptying, which facilitates absorption of nutrients within the small intestine [9]. Ileal resection may be associated with loss of this compensatory mechanism, which may contribute to the diarrhea observed in short bowel syndrome

Dr Kamal Deep Fortis Delhi 2:29:32 PM

KoA, the quantitative measure of a particular dialyzer's efficiency of clearance, is defined by membrane porosity and thickness, solute size, and flow rate of blood and dialysate. The KoA for urea generally varies from 200 to 1100 among the various different dialyzers available [2]. The KoA for a particular dialyzer should be the measurement observed at a blood flow rate of 300 to 400 mL/min.

Dr Kamal Deep Fortis Delhi 4:09:57 PM

The propensity for impedance of any solute is described by the sieving coefficient, a numerical assessment of the potential for convective transport of a given solute. This coefficient equals the ratio of the solute concentration in the filtrate to that in the arterial plasma. A sieving coefficient of 1 denotes a solute which passes completely unimpeded, whereas a solute that is completely rejected has a coefficient of 0. Very large and small solutes therefore have sieving coefficients approximating 0 and 1, respectively. For small solutes, such as urea, glucose, and electrolytes, the solute concentration in the fluid removed by convective transport is similar to that in the plasma. Since the capacity for small-solute diffusion is generally much greater than that for large-solute diffusion (eg, vitamin B12), the relative contribution of convection may be most important in large-solute transport

Dr Kamal Deep Fortis Delhi 4:31:16 PM

— A standard dose of platelets for prophylactic therapy in adults is approximately one random donor unit per 10 kg of body weight, which translates to four to six units of pooled platelets or one apheresis unit, both providing approximately 3 to 4 x 1011 platelets [2,26]. A standard pediatric dose is 5 to 10 mL/kg. For prophylactic transfusion there is generally no reason to transfuse platelets more often than once a day. This platelet dosing is expected to raise the platelet count by approximately 30,000/microL within 10 minutes of the infusion.

Dr Kamal Deep Fortis Delhi 6:19:14 PM

King's College Hospital criteria for liver transplantation in acute liver failure

Acetaminophen-induced disease
Arterial pH <7.3 (irrespective of the grade of encephalopathy)
OR
Grade III or IV encephalopathy AND
Prothrombin time >100 seconds AND
Serum creatinine >3.4mg/dL (301 µmol/L)
All other causes of acute liver failure
Prothrombin time >100 seconds (irrespective of the grade of encephalopathy)
OR
Any **three** of the following variables (irrespective of the grade of encephalopathy)
1. Age <10 years or >40 years
2. Etiology: non-A, non-B hepatitis, halothane hepatitis, idiosyncratic drug reactions
3. Duration of jaundice before onset of encephalopathy >7 days
4. Prothrombin time >50 seconds
5. Serum bilirubin >18 mg/dL (308 µmol/L)

Dr Kamal Deep Fortis Delhi 7:00:26 PM

BMI <18.5:	Below normal weight
BMI ≥18.5 and <25:	Normal weight
BMI ≥25 and <30:	Overweight
BMI ≥30 and <35:	Class I Obesity
BMI ≥35 and <40:	Class II Obesity
BMI ≥40:	Class III Obesity

The refeeding syndrome is marked by: 7:02:24 PM

●Hypophosphatemia
●Hypokalemia
●Vitamin (eg, thiamine) deficiencies
●Congestive heart failure
●Peripheral edema

Hypophosphatemia is the hallmark of the syndrome and predominant cause of the refeeding syndrome 7:03:37 PM

The pathogenesis of hypophosphatemia begins when stores of phosphate are depleted during episodes of anorexia nervosa and starvation. When nutritional replenishment begins and patients are fed carbohydrates, glucose causes release of insulin, which triggers cellular uptake of phosphate (and potassium and magnesium). Insulin also causes cells to produce a variety of depleted molecules that require phosphate (eg, adenosine triphosphate (ATP) and 2,3-diphosphoglycerate), which further depletes the body's stores of phosphate [10]. The lack of 7:05:08 PM

phosphorylated intermediates causes tissue hypoxia and resultant myocardial dysfunction and respiratory failure due to an inability of the diaphragm to contract.

Vitamin and trace mineral deficiencies are due to starvation [10]. These deficiencies are exacerbated by the onset of anabolic processes that accompany refeeding the patient.

Volume overload begins with an increase in insulin secretion during the early stage of refeeding the patient [9]. This eventually increases renal sodium reabsorption and retention, and then fluid retention.

Monday, March 28, 2016

Dr Kamal Deep Fortis Delhi 11:05:45 PM

Early symptoms of acute aspirin toxicity include tinnitus, vertigo, nausea, vomiting, and diarrhea; subsequent symptoms portending a more severe intoxication include altered mental status (ranging from agitation to lethargy), hyperpyrexia, noncardiac pulmonary edema, and coma.

Hyperpnea is often observed in salicylate overdose and is an early clinical finding that helps establish the diagnosis. 11:10:28 PM

Dr Kamal Deep Fortis Delhi 11:34:38 PM

Treatment of salicylate intoxication is directed toward decreasing the fraction of uncharged (protonated) molecules, which is accomplished by increasing the systemic pH (ie, lowering the H+ ion concentration). This is referred to as “alkalinization” and is most easily accomplished by administering sodium bicarbonate. Increasing the systemic pH reduces the diffusion of salicylate anions into the central nervous system, as charged molecules do not easily diffuse across the blood-brain barrier. Alkalinization also "traps" salicylate anions within the renal tubule, preventing back-diffusion across the renal epithelium into the systemic circulation

Salicylates lower the CNS glucose concentration; therefore, neuroglycopenia may occur despite normal serum glucose levels 11:35:12 PM

Mortality from salicylate poisoning correlates closely with CNS salicylate concentration, and altered mental status due to salicylate toxicity is an absolute indication for hemodialysis 11:35:41 PM

Pulmonary edema — Salicylate-induced noncardiogenic pulmonary edema and acute lung injury (ALI) generally occur in 11:36:40 PM

older patients with chronic salicylate intoxication [21-24], but they should be considered in all patients presenting with salicylate toxicity. Salicylate-induced ALI and pulmonary edema can complicate volume resuscitation and the administration of sodium bicarbonate, two mainstays of treatment in this setting. Thus, the presence of salicylate-induced pulmonary edema is considered an absolute indication for hemodialysis.

Levels above 100 mg/dL (7.2 mmol/L) are associated with increased morbidity and mortality, and are considered an absolute indication for hemodialysis. 11:38:35 PM

Serum salicylate — Therapeutic serum salicylate concentrations fall between 10 to 30 mg/dL (0.7 to 2.2 mmol/L); values above 40 mg/dL (2.9 mmol/L) are associated with toxicity. Although toxicity does not correlate exactly with serum salicylate concentrations and symptoms, most patients exhibit signs of intoxication when the serum concentration exceeds 40 to 50 mg/dL (2.9 to 3.6 mmol/L) 11:39:10 PM

Some laboratories report salicylate concentrations as mg/L; such values are therefore 10-fold higher than those discussed in this topic review. Thus, a laboratory report of "an aspirin level of 110" in an asymptomatic individual should prompt the clinician to check the units of the result in order to avoid confusion. 11:39:52 PM

Creatinine — Aspirin is eliminated almost exclusively via the kidneys, so the serum creatinine concentration should be checked in all patients who present with known or suspected salicylate toxicity. Renal failure is an absolute indication for hemodialysis in the salicylate-poisoned patient; mild renal impairment is a relative indication for hemodialysis, and must be interpreted in light of the patient's overall clinical condition. 11:42:46 PM

Potassium — Hypokalemia, if present, must be treated aggressively. Hypokalemia promotes the absorption of potassium in the distal tubule; this absorption occurs via a K+/H (tg://bot_command?command=H)+ exchange pump (figure 1). The secretion of protons involved in this pump interferes with efforts at urinary alkalinization, which are a mainstay of therapy. 11:48:30 PM

MANAGEMENT — As with all poisoned patients, treatment consists of rapid assessment and stabilization of the airway, breathing, and circulation. This should be followed by gastrointestinal decontamination if indicated and the initiation of specific therapies designed to mitigate the effects of the toxin, which in the case of aspirin consists of alkalinization of the 11:52:51 PM

plasma and urine, and in some cases hemodialysis. A summary table to facilitate emergent management of aspirin intoxication is provided

Tracheal intubation of the aspirin-poisoned patient is dangerous [36], and should be reserved for cases of clear respiratory failure. Intubation of tachypneic patients to prevent physical exhaustion following an aspirin overdose has resulted in death [37]. 11:55:30 PM

Aspirin acts on the respiratory center of the medulla to increase the respiratory rate (RR) and tidal volume (Vt). Minute ventilation (MV) is determined by RR and Vt (RR x Vt = MV), so an increase in either can produce a dramatic increase in minute ventilation. As noted above, the resulting respiratory alkalosis "traps" salicylate anions in the blood, preventing them from continuing to cross into the CNS. (See 'Acid-base abnormalities' above.)

Clinicians should be aware of how airway management can exacerbate the condition of patients with salicylate poisoning. The brief period of apnea that occurs with the sedation and paralysis performed in preparation for intubation can cause an acute and substantial worsening of the patient's respiratory acidosis. During this period, salicylate anions become protonated to uncharged salicylic acid, diffuse across the blood-brain barrier, and increase toxicity. The high respiratory rate and minute ventilation observed in unintubated patients with salicylate poisoning is difficult to replicate with a ventilator. Therefore, orotracheal intubation often causes relative hypoventilation, resulting in a higher $PaCO_2$ than is maintained by spontaneous ventilation, and possibly respiratory acidosis, leading to increased toxicity. In addition, ventilator asynchrony may decrease a patient's ability to maintain appropriate acid-base homeostasis.

In general, intubation should be reserved for those patients with hypoventilation, as determined by clinical evaluation or blood gas analysis. When intubation becomes necessary due to primary respiratory failure, maintaining high tidal volume and a rapid respiratory rate becomes critically important. Ventilator settings should mimic the respiratory rate of the patient prior to intubation and tidal volumes will likely need to exceed the 6 to 8 mL/kg commonly used. Unless there is ventilator-patient asynchrony or a comparable problem making ventilation extremely difficult, long acting neuromuscular blockade and deep

sedation, which blunt the patient's ability to breath over the ventilator, should be avoided as much as possible

To appreciate how alkalinization acts, it is important to note that salicylic acid (HSal) is a weak acid. In the equilibrium reaction 11:58:48 PM

H+ + Sal- <—> HSal,

if the systemic pH is increased, the equation will move to the left. The ensuing fall in the plasma HSal concentration will allow HSal in the CNS and other tissues to diffuse into the extracellular fluid down a favorable concentration gradient where it will be trapped as Sal-. The decrease in the CNS salicylic acid concentration then causes the first equation to move to the right in the brain cell. This increase in the cellular salicylic acid concentration promotes further drug movement out of the CNS.

Tuesday, March 29, 2016

Alkalemia from a respiratory alkalosis is NOT a contraindication to sodium bicarbonate therapy. Aspirin-poisoned patients commonly present with an arterial pH between 7.50 and 7.55; these patients should be treated with sodium bicarbonate [43]. Blood gas analysis every two hours is indicated for monitoring to prevent severe alkalemia (arterial pH >7.60). A urine pH of 7.5 to 8 is desirable 12:02:52 AM

Acetazolamide is contraindicated in the standard management of salicylate poisoning. Acetazolamide is a carbonic anhydrase inhibitor that alkalinizes the urine by reducing bicarbonate reabsorption. While this does enhance salicylate excretion, the bicarbonate loss from plasma lowers the arterial pH, which promotes salicylate movement into the brain, potentially worsening salicylate neurotoxicity 12:06:04 AM

Hemodialysis — The efficiency of salicylate removal can be enhanced by hemodialysis [47]. Indications for hemodialysis include the following: 12:06:56 AM

●Altered mental status
●Pulmonary or cerebral edema
●Renal insufficiency that interferes with salicylate excretion
●Fluid overload that prevents the administration of sodium bicarbonate
●A serum salicylate concentration >100 mg/dL (7.2 mmol/L) in acute overdose

●Clinical deterioration despite aggressive and appropriate supportive care

Dr Kamal Deep Fortis Delhi 11:10:12 AM

Absorbed dose — The rad (radiation absorbed dose) is the traditional unit of absorbed dose and is defined as the transfer of 100 ergs per gram of tissue.

The rad has been superseded in the SI (Système International) by the Gray (Gy). One Gy, the unit most commonly used to measure radiation therapy dose, is equivalent to 100 rad (1 joule/kilogram), while one cGy is equivalent to 1 rad or 1000 mrad. One Gy is equivalent to 100 cGy or 1000 mGY.

The rem has been superseded in the SI by the Sievert (Sv). One Sv is equivalent to 100 rem. One Sv is equivalent to 1000 mSv or 1,000,000 microSv.

For most therapeutic radiation exposures (eg, x-rays, gamma rays) the Sievert and Gray are approximately equal. However, when there is exposure to highly ionizing particles (eg, neutrons, alpha particles) the radiation dose equivalent (in Sv) reflects resulting tissue damage better than the absorbed dose (in Gy). As an example, the quality factor for x-rays, gamma rays, and beta particles is 1, while that for alpha particles is 20, and the factor can range from 4 to 22 for neutrons, depending on neutron energy

A standard chest x-ray delivers a dose of 6 to 11 mrad (0.06 to 0.11 mGy). A two-view mammogram delivers a dose of 72 mrad (0.72 mGy). A full body computed tomography (CT) scan and standard chest CT deliver doses of 1000 mrad (10 mGy) and 700 mrad (7 mGy), respectively.

The lowest radiation dose resulting in an observable effect on bone marrow depression in man, with a resultant decrease in blood cell counts, is in the range of 100 to 200 rem (1000 to 2000 mSv, 1.0 to 2.0 Sv) or 100 to 200 rad (1.0 to 2.0 Gy; 1000 to 2000 mGy).
●Cataracts have occurred in atomic bomb survivors in a dose-response manner with ocular doses as low as 0.4 Gy (400 mGy) [17]. The hazard ratio for an ocular dose of 3 Gy (300 mGy) was 2.3 (95% CI 1.6-3.2). This represents an increase in incidence from 108 per 10,000 person years in the unexposed to 189 per 10,000 person years in individuals with ocular doses of ≥3 Gy.
●The lowest total body dose at which the first clinical signs and

symptoms of radiation sickness may be seen following exposure to ionizing radiation is in the range of 1.0 to 2.0 Gy (1000 to 2000 mGy; 1000 to 2000 mSv). In the absence of supportive care, 50 percent of people exposed to a dose of 3.5 to 4 Gy (3500 to 4000 mGy; 3500 to 4000 mSv) will be expected to die of radiation-induced injury. (See 'Lethal dose of radiation' below.)

●There is virtually no chance of survival following a total body exposure in excess of 10 to 12 Gy, regardless of therapy.

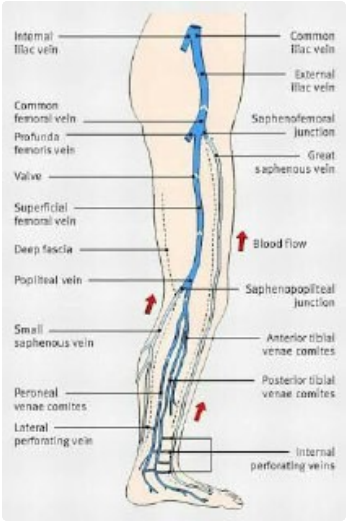
Based upon an analysis of all available data, the LD 50 at 60 days (LD 50/60) for humans has been estimated to be approximately 3.5 to 4.0 Gy in persons managed without supportive care, 4.5 to 7 Gy when antibiotics and transfusion support are provided, and potentially as high as 7 to 9 Gy in patients with rapid access to intensive care units, reverse isolation, and hematopoietic cell transplantation [22-25]. This differs from higher doses used for total body irradiation (TBI) in preparation for hematopoietic cell transplantation (eg, 12 to 16 Gy), because preparative TBI spares critical organs such as the heart, liver, and kidneys, allowing more selective ablation of hematopoietic cells.

The presence of chromosomal aberrations is currently the "gold standard" for biodosimetry. The frequency of dicentrics and rings can detect a radiation dose of as low as 1 Gy (100 mGy), and these changes persist for six months and possibly longer. 11:12:02 AM

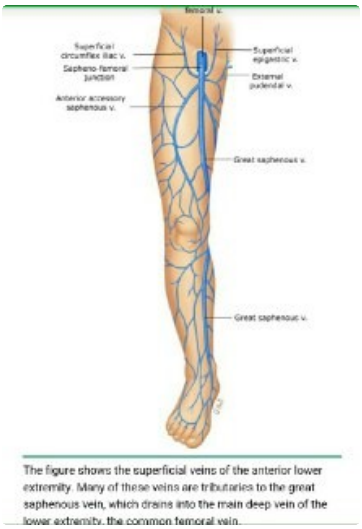
Dr Kamal Deep Fortis Delhi 11:39:28 AM

1 cGy= 1RAD
1 Gy = 100 RAD= 100cGy
1 RAD= 1000 MRAD
1 Gy= 1000 mGy
1 cGy = 10 mGy

Dr Kamal Deep Fortis Delhi 1:05:14 PM

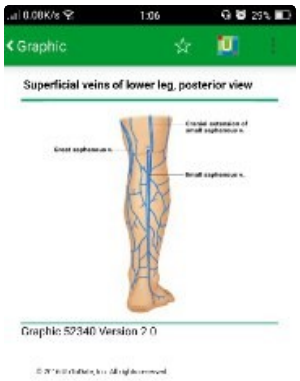


1:06:10 PM

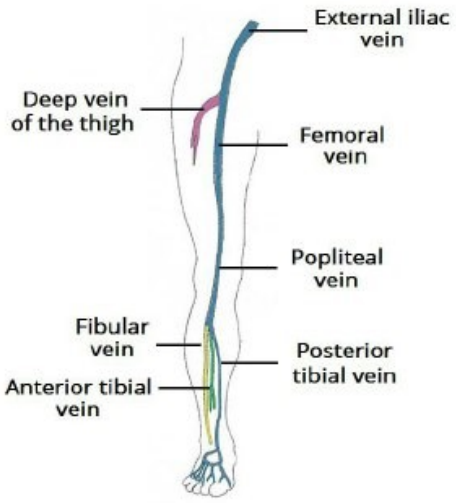


The figure shows the superficial veins of the anterior lower extremity. Many of these veins are tributaries to the great saphenous vein, which drains into the main deep vein of the lower extremity, the common femoral vein.

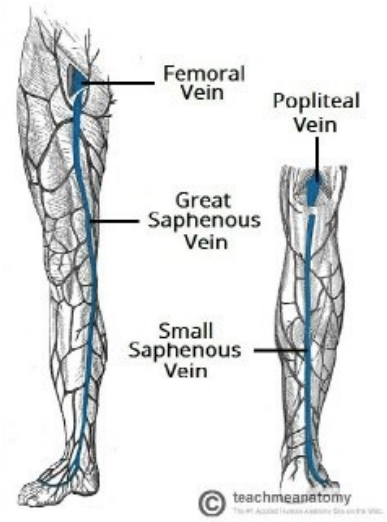
1:06:28 PM



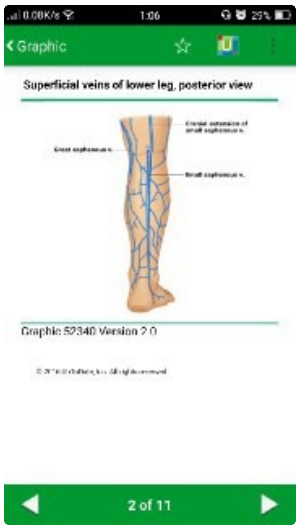
1:11:42 PM



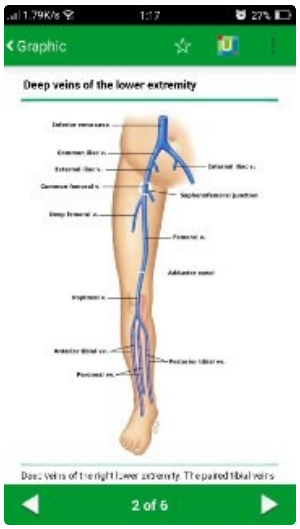
1:12:00 PM



Deep venous system and this one above is superficial venous system 1:12:26 PM



1:17:08 PM



1:17:34 PM

Wednesday, March 30, 2016

Dr Kamal Deep Fortis Delhi 11:56:58 AM

Anaphylaxis Rx:-The epinephrine dilution for intramuscular injection contains 1 mg per mL and may also be labeled as 1:1000.
For adults, the recommended dose of epinephrine (1 mg per mL) is 0.3 to 0.5 mg per single dose, injected intramuscularly into the mid-outer thigh (vastus lateralis muscle). Based on clinical experience and consensus opinion, this dose may be repeated at 5 to 15 minute intervals

Intramuscular injection is recommended over subcutaneous injection because it consistently provides a more rapid increase in the plasma and tissue concentrations of epinephrine 11:58:36 AM

Adverse cardiovascular events were significantly more likely with IV bolus compared with slow IV infusion or intramuscular injection (4 of 30 versus 0 of 4 and 3 of 245, respectively) 12:00:22 PM

For adults, the initial dose for intravenous epinephrine infusion is 2 to 10 micrograms per minute 12:01:08 PM

Over the past few decades, consensus has been reached that even mild systemic reactions are best treated immediately with epinephrine, as this appears to prevent progression to more severe symptoms more effectively than any other available therapies. As a result, successive guidelines for treatment of immunotherapy reactions have called for epinephrine to be given as soon as a systemic reaction of any severity is detected 12:03:20 PM

the heart is a target organ in anaphylaxis. In the healthy human heart, mast cells are present throughout the myocardium and in the intima of coronary arteries. In patients with coronary artery disease, mast cells are found in atherosclerotic lesions and contribute to atherogenesis. Anaphylaxis can unmask subclinical coronary artery disease, and myocardial infarction and/or arrhythmias can occur during anaphylaxis, even if epinephrine is not injected 12:04:12 PM

Key:- MI can occur in high risk Patient in anaphy 12:10:49 PM

Medication errors have occurred due to confusion with epinephrine products expressed as ratio strengths (eg, 1:1000 vs 1:10,000). 12:13:41 PM

Epinephrine 1:1000 = 1 mg/mL and is most commonly used IM

Epinephrine 1:10,000 = 0.1 mg/mL and is used IV

Medication errors have occurred when topical epinephrine 1 mg/mL (1:1000) has been inadvertently injected. Vials of injectable and topical epinephrine look very similar. Epinephrine should always be appropriately labeled with the intended administration.

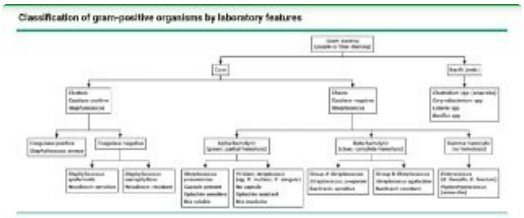
Hypersensitivity reaction (eg, anaphylaxis): Note: SubQ administration results in slower absorption and is less reliable. IM administration in the anterolateral aspect of the middle third of the thigh is preferred in the setting of anaphylaxis (AHA [Vanden Hoek 2010]; Kemp 2008). 12:16:22 PM

IM, SubQ: 0.2 to 0.5 mg (1:1000 [1 mg/mL] solution) every 5 to 15 minutes in the absence of clinical improvement (AHA [Vanden Hoek 2010]; Kemp 2008; Lieberman 2010). If clinician deems appropriate, the 5-minute interval between injections may be shortened to allow for more frequent administration (Lieberman 2010).

IV: 0.1 mg (1:10,000 [0.1 mg/mL] solution) over 5 minutes; may infuse at 1 to 4 mcg/minute to prevent the need to repeat injections frequently or may initiate with an infusion at 5 to 15 mcg/minute (with crystalloid administration) (AHA [Vanden Hoek 2010]; Brown 2004). In general, IV administration should only be done in patients who are profoundly hypotensive or are in cardiopulmonary arrest refractory to volume resuscitation and several epinephrine injections

0.5 mg IM epinephrine= 500 ug 12:26:20 PM

Dr Kamal Deep Fortis Delhi 12:42:06 PM



The finding of gram-positive diplococci (spheres in pairs) is pathognomonic for S. pneumoniae; this organism may also be arranged in short chains. 12:52:54 PM

Status epilepsy:- evidence supports and experts agree that benzodiazepines should be the agent of choice for emergent initial treatment. When skilled health care personnel are available, 1:03:48 PM

intravenous (IV) administration is preferred. However, benzodiazepines can be administered via intramuscular (IM), rectal, nasal, or buccal routes when IV therapy is not feasible. For IV therapy, lorazepam is the preferred agent; midazolam is preferred for IM therapy (and can also be given nasally or buccally); and diazepam is preferred for rectal administration

Urgent control therapy:- 1:05:46 PM
InpatientswithknownepilepsywhohavebeenonanAEDbeforeadmission,itisreasonabletoprovideanIVbolusofthisAED,ifavailable,priortoinitiatinganadditionalagent.Thismayincludeadditionalbolusesthatwillresultinhigherthannormaltargetconcentrationsoftheaedtoachievethedesiredtherapeuticresponse(i.e.,cessationofseizureactivity).

DEFINITION — The duration of continuous seizure activity used to define status epilepticus has varied over time. Historically, the International League Against Epilepsy (ILAE) and others defined status epilepticus as a single epileptic seizure of >30 minutes duration or a series of epileptic seizures during which function is not regained between ictal events in a 30 minute period [1]. 1:13:18 PM

Because of the clinical urgency in treating generalized convulsive status epilepticus (GCSE), however, a 30 minute definition is neither practical nor appropriate in clinical practice. Once seizures have continued for more than a few minutes, treatment should begin without further delay.

Considering the need for rapid evaluation and intervention in GCSE to avoid cardiovascular morbidity and refractory status, an accepted operational definition of GCSE consists of the following [2-4]:

●≥5 minutes of continuous seizures, or
●≥2 discrete seizures between which there is incomplete recovery of consciousness
In 2015, the ILAE published a revised conceptual definition of status epilepticus that incorporates two operational dimensions, t1 and t2 [5]:

●Status epilepticus is a condition resulting from either the failure of the mechanisms responsible for seizure termination or from the initiation of mechanisms that lead to abnormally prolonged seizures (after time point t1)
●Status epilepticus is a condition that can have long-term consequences (after time point t2), including neuronal death, neuronal injury, and alteration of neuronal networks, depending

on the type and duration of seizures
For GCSE, the ILAE proposal specifies that t1 and t2 are 5 and 30 minutes, respectively, based on the best available data from animal and clinical studies [5]. For other types of status epilepticus, the most appropriate time intervals for t1 and t2 have not been well defined and are far more speculative, particularly for nonconvulsive status. The ILAE suggests using a t1 and t2 of 10 and >60 minutes for focal status epilepticus with impaired consciousness (often diagnosed as complex partial status epilepticus) and a t1 of 10 to 15 minutes for absence status epilepticus.

Dr Kamal Deep Fortis Delhi 1:37:08 PM

Helium reduces the work of breathing by decreasing airway resistance and has no effect on relieving bronchoconstriction

In asthma 1:37:16 PM

Dr Kamal Deep Fortis Delhi 2:47:50 PM

Etiology of lactic acidosis

Increased lactate production

Increased pyruvate production
Enzymatic defects in glycogenolysis or gluconeogenesis (as with type 1 glycogen storage disease)
Respiratory alkalosis, including salicylate intoxication
Pheochromocytoma
Beta-agonists
Sepsis

Impaired pyruvate utilization
Decreased activity of pyruvate dehydrogenase or pyruvate carboxylase
• Congenital
• Possibly a role in diabetes mellitus, Reye's syndrome

Altered redox state favoring pyruvate conversion to lactate:-

Enhanced metabolic rate:-
• Grand mal seizure
• Severe exercise
• Hypothermic shivering
• Severe asthma

Decreased oxygen delivery:-
• Shock
• Cardiac arrest

• Acute pulmonary edema
• Carbon monoxide poisoning
• Severe hypoxemia (PO2 <25 to 30 mmHg)
• Pheochromocytoma

Reduced oxygen utilization:-
• Cyanide intoxication (decreased oxidative metabolism), which may result from cyanide poisoning or, during a fire, from smoke inhalation of vapors derived from the thermal decomposition of nitrogen-containing materials such as wool, silk, and polyurethane
• Drug-induced mitochondrial dysfunction due to zidovudine or stavudine
• Sepsis
D-lactic acidosis

Primary decrease in lactate utilization:-
Hypoperfusion and marked acidemia
Alcoholism
Liver disease

Mechanism uncertain:-
Malignancy
Diabetes mellitus, including metformin in the absence of tissue hypoxia
Acquired immune deficiency syndrome
Hypoglycemia
Idiopathic
Although this table has been divided into either increased production or decreased utilization of lactate, there is considerable overlap among listed causes.
Modified with permission from: Rose BD, Post TW. Clinical Physiology of Acid-Base and Electrolyte Disorders, 5th ed, McGraw-Hill, New York 2001. p.594. Copyright 2001 McGraw-Hill.
Graphic 78983 Version 6.0

Key point:- Beta agonists and diabetes mellitus can cause lactate high without normal oxygen delivery 2:49:44 PM

Dr Kamal Deep Fortis Delhi 3:40:32 PM

Hypothermia

CLINICAL PRESENTATION

General findings and progression — As the compensatory mechanisms preventing hypothermia are overwhelmed, the following changes typically occur (table 3) [9,17,18]:

●Patients with mild hypothermia demonstrate tachypnea, tachycardia, initial hyperventilation, ataxia, dysarthria, impaired judgment, shivering, and so-called "cold diuresis."
●Moderate hypothermia is characterized by proportionate reductions in pulse rate and cardiac output, hypoventilation, central nervous system depression, hyporeflexia, decreased renal blood flow, and loss of shivering. Paradoxical undressing may be observed. Atrial fibrillation, junctional bradycardia, and other arrhythmias can occur.
●Severe hypothermia can lead to pulmonary edema, oliguria, areflexia, coma, hypotension, bradycardia, ventricular arrhythmias (including ventricular fibrillation), and asystole

Especially in chronic hypothermia with dehydration, rewarming of the trunk should be undertaken BEFORE the extremities. These actions are performed in order to minimize core temperature afterdrop and hypotension and acidemia due to arterial vasodilation

3:49:24 PM

Core temperature afterdrop is a particular risk of active external rewarming. This complication occurs when the extremities and trunk are warmed simultaneously. Cold, acidemic blood that has pooled in the vasoconstricted extremities of the hypothermic patient returns to the core circulation, causing a drop in temperature and pH. At the same time, removal from the cold environment results in peripheral vasodilation, potentially contributing to precipitous hypotension, inadequate coronary perfusion, and ventricular fibrillation [9]. These phenomena may explain the fatal dysrhythmias that sometimes occur during rewarming [51,52]. Combining active core rewarming techniques with AER can minimize rewarming shock and afterdrop in patients with severe hypothermia

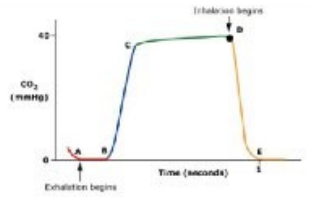
3:50:16 PM

3:57:54 PM

Dr Kamal Deep Fortis Delhi

5:38:20 PM

Normal CO2 waveform

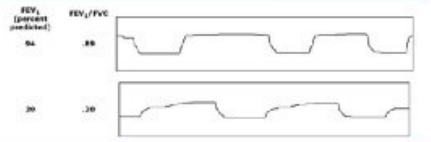


A - B: Dead space ventilation
B - C: Ascending expiratory phase
C - D: Alveolar Plateau
D: End-tidal CO_2
D - E: Descending inspiratory phase

Reproduced with permission from: Krauss B, Deykin A, Lam A, et al. Capnogram shape in obstructive lung disease. Anesth Analg 2005; 100:884. Copyright © 2005 Lippincott Williams & Wilkins.
Graphic 67700 Version 11.0

5:40:08 PM

CO_2 waveform in obstructive lung disease



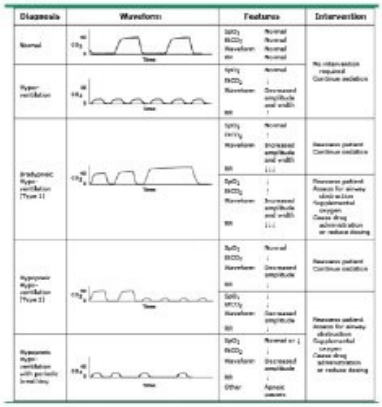
The top waveform is from a patient with normal lung function and has a characteristic rectangular appearance. The bottom waveform is from a patient with severe chronic obstructive pulmonary disease and has a characteristic curved appearance and up-sloping of the alveolar plateau.

FEV_1: forced expiratory volume in one second; FVC: forced vital capacity.

Reproduced with permission from: Krauss B, Deykin A, Lam A, et al. Capnogram shape in obstructive lung disease. Anesth Analg 2005; 100:884. Copyright © 2005 Lippincott Williams & Wilkins.
Graphic 64586 Version 10.0

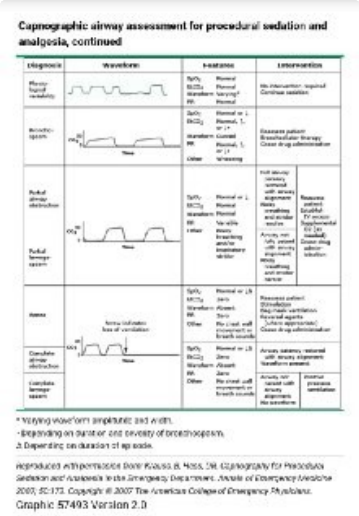
5:41:30 PM

Capnographic airway assessment for procedural sedation and analgesia



Reproduced with permission from: Krauss, B, Hess, DR. Capnography for Procedural Sedation and Analgesia in the Emergency Department. Annals of Emergency Medicine 2007; 50:172. Copyright © 2007 The American College of Emergency Physicians.
Graphic 78408 Version 2.0

5:41:54 PM



Capnographic airway assessment for procedural sedation and analgesia, continued

Diagnosis	Waveform	Features	Interventions
Normal			
Bronchospasm			
Partial airway obstruction			
Partial laryngospasm			
Apnea			
Complete airway obstruction			
Complete laryngospasm			

* Varying waveform amplitudes and width.
† Beginning on duration and severity of bronchospasm.
‡ Depending on duration of apnea.

Reproduced with permission from: Krauss B, Hess DR. Capnography for Procedural Sedation and Analgesia in the Emergency Department. Annals of Emergency Medicine 2007; 50:172. Copyright © 2007 The American College of Emergency Physicians.

Graphic 57493 Version 2.0

Prognosis in cardiac arrest — In several prospective, observational studies, EtCO2 levels of ≤10 mmHg measured 20 minutes after the initiation of advanced cardiac life support accurately predicted death in adult patients with cardiac arrest [42-45]. The prognostic value of measuring EtCO2 has been demonstrated in animal [46] and human studies [42-45,47]. 5:46:42 PM

Unlike pulse oximetry, capnography does not misinterpret motion artifact and provides reliable readings in low perfusion states. 5:47:27 PM

Dr Kamal Deep Fortis Delhi 10:18:40 PM

The electrical activity in each EEG channel can be described in terms of amplitude and frequency. The amplitude of typical EEG recordings ranges from 5 to 200 microvolts, but most awake background EEG recordings are in the range of 20 to 50 microvolts. Frequency of EEG activity is expressed according to the following terminology:

●Delta — 0 to 4 Hz
●Theta — 4 to 8 Hz
●Alpha — 8 to 13 Hz
●Beta —13-30 Hz
●Gamma – Greater than 30 Hz

Thursday, March 31, 2016

Dr Kamal Deep Fortis Delhi 2:19:51 PM

Virtually all of the drugs that produce LQTS act by blocking the IKr current mediated by the potassium channel encoded by the KCNH2 gene

2:23:43 PM

Hypokalemia, hypomagnesemia, and hypocalcemia — Hypokalemia and hypomagnesemia can predispose to TdP. These disorders can occur together since hypomagnesemia directly causes hypokalemia. Hypocalcemia, alone or induced by hypomagnesemia, is a less common cause

As noted above, virtually all of the drugs that produce LQTS act by blocking the IKr current mediated by the potassium channel encoded by the KCNH2 gene [12-25,28]. The increase in risk with hypokalemia may be related to enhanced drug blockade of IKr 2:24:35 PM

Acquired LQTS is commonly associated with bradycardia and pauses, in contrast to some forms of congenital LQTS (particularly types 1 and 2) (figure 1) in which arrhythmias often follow a sudden adrenergic surge 2:30:00 PM

Drugs that prolong the QT interval include antiarrhythmic drugs (classes IA and III), certain nonsedating antihistamines (eg, terfenadine and astemizole), macrolide antibiotics, certain psychotropic medications, and certain gastric motility agents (eg, cisapride) 2:30:42 PM

Prompt, nonsynchronized electric defibrillation is indicated in patients with hemodynamically unstable TdP. 2:31:26 PM
•In the conscious patients with TdP, we suggest a brief trial of medical therapy (see 'Acute therapy of TdP' above):
•Intravenous magnesium is first-line therapy, as it is highly effective for both treatment and prevention of recurrence of long QT-related ventricular ectopic beats or TdP. The benefit is seen even in patients with normal serum magnesium concentrations at baseline.
•Temporary transvenous overdrive pacing (atrial or ventricular) at about 100 beats per minute is generally reserved for patients who do not respond to intravenous magnesium.
•Isoproterenol (initial dose 0.05 to 0.1 mcg/kg per min in children and 2 mcg/min in adults, then titrated to achieve a heart rate of 100 beats per minute) can be used as a temporizing measure to achieve a heart rate of 100 beats per minute prior to pacing.

Antipsychotics 2:35:04 PM
Known risk: chlorpromazine, haloperidol, methotrimeprazine (levomepromazine)Δ, pimozide, sulpirideΔ, thioridazine

The membrane is most permeable to K+ (mainly due to leak channels) and relatively impermeable to other ions. The resting membrane potential is therefore dominated by the K+ equilibrium potential (-80mV) according to the K+ gradient across the cell 2:45:24 PM

membrane. The membrane potential can be calculated using the Goldman-Hodgkin-Katz voltage equation. The maintenance of this electrical gradient is due to various ion pumps and exchange mechanisms, which use ATP (energy) to pump ions against their electrochemical gradient, some of which include the Na+-K+ ion exchange pump, the Na+-Ca2+ exchanger current and the IK1 inwardly rectifying K+ current. I is the symbol for an electric current.

Dr Kamal Deep Fortis Delhi 3:19:16 PM

SMR The standardized mortality ratio is the ratio of observed deaths in the study group to expected deaths

If the SMR is quoted as a ratio and is equal to 1.0, then this means the number of observed deaths equals that of expected cases. If higher than 1.0, then there is a higher number of deaths than is expected. SMR constitutes an indirect form of standardization. It has an advantage over the direct method of standardization since age-adjustment is permitted in situations where age stratification may not be available for the cohort being studied or where strata-specific data are subject to excessive random variability. 3:20:04 PM

Dr Kamal Deep Fortis Delhi 4:02:20 PM

Patient Contact	Examples	Device Classification	Minimum Inactivation Level
Intact skin		Non-Critical	Low Level or Intermediate Level Disinfection
Mucous membranes or non-intact skin		Semi-Critical	High Level Disinfection
Sterile areas of the body, including blood contact		Critical	Sterilization

Spaulding classification 4:02:32 PM

Dr Kamal Deep Fortis Delhi 8:01:14 PM

World Federation of Neurological Surgeons subarachnoid hemorrhage grading scale

Grade	GCS score	Motor deficit
1	15	Absent
2	13 to 14	Absent
3	13 to 14	Present
4	7 to 12	Present or absent
5	3 to 6	Present or absent

GCS: Glasgow Coma Scale.

Based upon data from: Report of World Federation of Neurological Surgeons Committee on a Universal Subarachnoid

In the absence of ICP measurement, antihypertensive therapy is often withheld unless there is a severe elevation in blood pressure [43]. The patient's cognitive status may be a useful guide; if the patient is alert, then CPP is adequate, and lowering the blood pressure may decrease the risk of rerupture; we typically keep the systolic blood pressure in such patients below 140 mmHg. In contrast, antihypertensive therapy is generally withheld in those with a severely impaired level of consciousness since the impairment may be due to a reduced CPP. 8:03:28 PM

SAH

The mechanism of benefit of nimodipine in SAH is unknown. Putative mechanisms include neuroprotection via reduction of calcium-dependent excitotoxicity, diminished platelet aggregation, dilation of small arteries not visible on angiograms, inhibition of ischemia triggered by red blood cell products, or some combination of these actions. 8:10:43 PM

Nimodipine — Nimodipine 60 mg every four hours is administered to all patients with aneurysmal SAH, ideally within four days of SAH. The typical dose is 60 mg every four hours by mouth or nasogastric tube. 8:11:02 PM

Euvolemia is the goal of intravenous fluid maintenance, usually with normal saline, which should be monitored by documentation of fluid input and output. 8:12:20 PM

Aggressive therapy of vasospasm can only be pursued after the aneurysm has been treated with surgery or intraluminal therapy 8:15:20 PM

8:15:54 PM

More recently, the focus has shifted toward maintenance of euvolemia using crystalloid or colloid solution, and induced hypertension with vasopressor agents such as phenylephrine, norepinephrine, or dopamine [2,7,33]. Blood pressure augmentation should progress in a stepwise fashion with assessment of clinical status at each mean arterial pressure level

Patients treated with hemodynamic augmentation need to be monitored for complications such as pleural or cerebral edema and volume overload. 8:16:22 PM

This approach does not appear to be helpful as a prophylaxis against vasospasm.

Dr Kamal Deep Fortis Delhi 9:12:26 PM

In significantly malnourished patients, the initial stage of oral, enteral, or parenteral nutritional replenishment causes electrolyte and fluid shifts that may precipitate disabling or fatal medical complications [5-11]. The refeeding syndrome is marked by:

●Hypophosphatemia
●Hypokalemia
●Vitamin (eg, thiamine) deficiencies
●Congestive heart failure
●Peripheral edema
Hypophosphatemia is the hallmark of the syndrome and predominant cause of the refeeding syndrome

Patients in intensive care — Patients in ICUs account for the greatest number of episodes of candidemia in most hospitals. Surgical units, especially those caring for trauma and burn patients, and neonatal units have the highest rates of Candida infections. Besides the risks associated with the extremes of age and trauma or burns, other factors include [3,9]: 9:18:04 PM

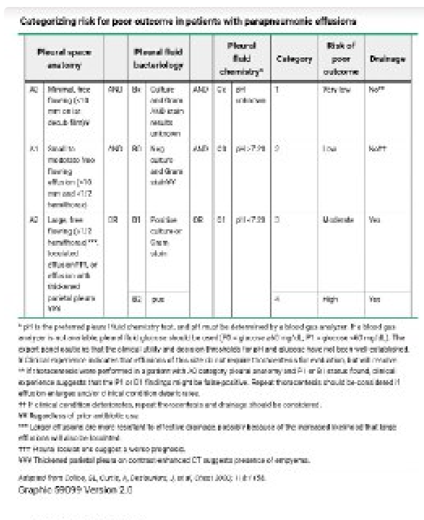
●Central venous catheters
●Total parenteral nutrition
●Broad-spectrum antibiotics
●High APACHE scores
●Acute renal failure, particularly if requiring hemodialysis
●Prior surgery, particularly abdominal surgery
●Gastrointestinal tract perforations and anastomotic leaks

Knowing the prevalence of the non-albicans Candida species is important because susceptibility to antifungal agents varies among the species. As an example, all isolates of C. krusei are fluconazole resistant, and an increasing proportion of C. glabrata 9:20:38 PM

are fluconazole resistant. Risk factors for candidemia caused by fluconazole-resistant isolates, mostly C. glabrata, include neutropenia, chronic renal disease, chronic lung disease, male gender, and previous fluconazole or other antifungal exposur

Penetration through the gastrointestinal tract mucosa is probably the most common mechanism for Candida species to enter the bloodstream in both immunocompromised patients (eg, those receiving chemotherapy who are neutropenic) and in ICU patients 9:25:22 PM

Dr Kamal Deep Fortis Delhi 10:19:47 PM



Categorizing risk for poor outcome in patients with parapneumonic effusions

Pleural space anatomy			Pleural fluid bacteriology			Pleural fluid chemistry*		Category	Risk of poor outcome	Drainage
A0	Minimal, free flowing (<10 mm on lat decub film)¶	AND	Bx	Culture and Gram stain results unknown	AND	Cx	pH unknown	1	Very low	No**
A1	Small to moderate free flowing effusion (>10 mm and <1/2 hemithorax)	AND	B0	Neg culture and Gram stain[illegible]	AND	C0	pH >7.20	2	Low	No¶¶
A2	Large, free flowing (≥1/2 hemithorax)***, loculated effusion[illegible], or effusion with thickened parietal pleura [illegible]	OR	B1	Positive culture or Gram stain	OR	C1	pH <7.20	3	Moderate	Yes
			B2	pus				4	High	Yes

* pH is the preferred pleural fluid chemistry test, and pH must be determined by a blood gas analyzer. If a blood gas analyzer is not available, pleural fluid glucose should be used [illegible]. The expert panel cautions that the clinical utility and decision thresholds for pH and glucose have not been well established.
[illegible]

Graphic 89099 Version 2.0

Dr Kamal Deep Fortis Delhi 10:44:26 PM

In general, all parapneumonic effusions, EXCEPT those that are free flowing and layer less than 10 mm on a lateral decubitus film, should be sampled by thoracentesis. If a small bore catheter is used for thoracentesis, all free-flowing pleural fluid is drained prior to catheter removal. (See 'Pleural fluid analysis' above.)

●In patients with a parapneumonic effusion or an empyema, an empiric, broad spectrum antibiotic that includes coverage for anaerobic organisms should be initiated promptly, as empyemas often harbor multiple species of bacteria, particularly anaerobes. (See 'Choice of agent' above.)

●In patients with an uncomplicated parapneumonic effusion that is small to moderate in size, free flowing, and has a pH of 7.20 or greater (eg, category 2) (table 1), we recommend observation rather than drainage of any remaining pleural fluid (Grade 1B). (See 'Management' above.)

●In patients with a large, loculated, or complicated parapneumonic effusion (eg, category 3) (table 1), we recommend prompt drainage of any remaining pleural fluid

rather than observation (Grade 1B). For further drainage of the pleural space, we suggest tube thoracostomy rather than a more invasive procedure (eg, thoracoscopy, decortication) (Grade 2C). CT or ultrasound-guided placement of multiple tubes may be needed when pleural loculations prevent adequate drainage by a single tube. (See 'Complicated parapneumonic effusions' above.)

●In patients with a thoracic empyema documented by the presence of pleural pus (eg, category 4) (table 1), we recommend prompt drainage of any remaining pleural fluid rather than observation (Grade 1B). Acceptable initial methods for pleural drainage include tube thoracostomy and video-assisted thoracoscopic surgery (VATS) with debridement. The latter may be preferred in patients with multiple loculations and a thick pleural peel. (See 'Empyema' above.)

●When tube thoracostomy is used for initial drainage of an empyema, a chest CT scan should be obtained within 24 hours after chest tube placement to document appropriate placement of the tube and assess drainage. For patients who do not have good drainage of empyema fluid from a well-placed chest tube, we suggest intrapleural administration of a combination of tissue plasminogen activator (TPA) 10 mg and deoxyribonuclease (DNase) 5 mg, twice daily for three days rather than no intrapleural therapy or either agent alone (Grade 2B). (See 'Fibrinolytic agents' above and 'Fibrinolytic and mucolytic agents' above.)

●Continued failure of adequate pleural drainage should prompt thoracoscopy or thoracotomy to lyse adhesions, fully drain the pleural space, and optimize chest tube placement. The choice between thoracoscopic debridement and decortication depends on several factors; those favoring decortication include more adhesions, greater visceral pleural thickness, and larger empyema cavity size.

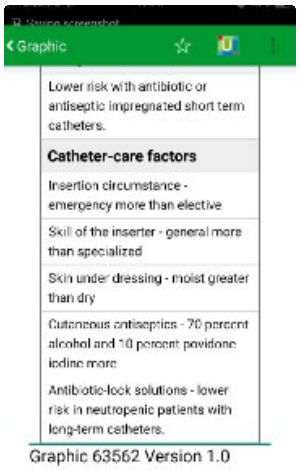
Friday, April 1, 2016

Dr Kamal Deep Fortis Delhi 12:18:46 AM

BSIs may be either primary or secondary. Secondary infections are related to infections at other sites, such as the urinary tract, lung, postoperative wounds, and skin. Most nosocomial BSIs are primary

While some primary BSIs have no identifiable source, most are associated with intravascular catheters, and central venous catheters (CVCs) in particular 12:19:06 AM

12:25:51 AM



Graphic 63562 Version 1.0

Central venous catheters
Internal jugular more than subclavian
Repeated catheterization
Presence of septic focus elsewhere
Nontunneled more than tunneled
Tunneled more than totally implantable device
Catheter insertion using submaximal barrier precautions
Lower risk with silver-chelated collagen cuff
Lower risk with antibiotic or antiseptic impregnated short term catheters.

12:26:36 AM

Skin colonization — The most common source of CVC-related infections is colonization of the intracutaneous and intravascular portions of the catheter by microorganisms from the patient's skin and occasionally the hands of healthcare workers (on insertion or as a result of manipulation) 12:28:28 AM

CVC-related infection due to anaerobic bacteria is exceedingly rare. 12:34:38 AM

Ventilator-Associated Pneumonia (VAP) Rate per 1,000 Ventilator Days 12:41:34 AM
Definition
Ventilator-associated pneumonia (VAP) is defined as pneumonia in a patient intubated and ventilated at the time of or within 48 hours before the onset of the event. NOTE: There is no minimum period of time that the ventilator must be in place in order for the pneumonia to be considered ventilator-associated.

The ventilator-associated pneumonia (VAP) rate is defined as the number of ventilator-associated pneumonias per 1,000 ventilator days. In this case, for a particular time period, we are interested in the total number of cases of ventilator-associated pneumonia in the ICU. For example, if in February there were 12 cases of VAP, the number of cases would be 12 for that month.

We want to be able to understand that number as a proportion of the total number of days that patients were on ventilators. Thus, if 25 patients were ventilated during the month and, for purposes of example, each was on mechanical ventilation for 3 days, the number of ventilator days would be 25 x 3 = 75 ventilator days for February. The Ventilator-Associated Pneumonia Rate per 1,000 Ventilator Days then would be 12/75 x 1,000 = 160.

Goal
Decrease the VAP rate per 1,000 ventilator days by 50 percent within 12 months.

Data Collection Plan
Report the monthly VAP rate for the last several months. This will serve as your baseline. Continue to track the measure monthly. If possible, track the rate in an annotated run chart, with notes reflecting any interventions you made to improve.

If your organization's infection control practitioner reports data quarterly for other review or accreditation agencies, this data should be disaggregated and reported monthly.

Definition:- 12:53:18 AM
CRBSI DIAG
Catheter related Bacteremia or fungemia in a patient who has an intravascular device and >1 positive blood culture result obtained from the peripheral vein, clinical manifestations of infection (eg, fever, chills, and/or hypotension), and no apparent source for bloodstream infection (with the exception of the catheter). One of the following should be present: a positive result of semiquantitative (>15 CFU per catheter segment) or quantitative (>102 CFU per catheter segment) catheter culture, whereby the same organism (species) is isolated from a catheter segment and a peripheral blood culture; simultaneous quantitative cultures of blood with a ratio of >3:1 CFU/mL of blood (catheter versus peripheral blood); differential time to positivity (growth in a culture of blood obtained through a catheter hub is detected by an automated blood culture system at least 2 hours earlier than a culture of simultaneously drawn peripheral blood of equal volume). Note that this definition

differs from the definition of central line-associated bloodstream infection used for infection control surveillance activities.

Chlorhexidine-based solutions appear to be superior to both aqueous and alcohol-based povidone-iodine in reducing the risk for catheter colonization and CRBSIs and is the preferred antiseptic of choice 12:58:12 AM

Dr Kamal Deep Fortis Delhi 1:28:11 AM

Removal — Following diagnosis of catheter-related infection, catheter removal is warranted in the following circumstances [1,7-11]:

●Severe sepsis
●Hemodynamic instability
●Endocarditis or evidence of metastatic infection
●Erythema or exudate due to suppurative thrombophlebitis
●Persistent bacteremia after 72 hours of antimicrobial therapy to which the organism is susceptible

In general, systemic antibiotic therapy is NOT required in the following circumstances (see 'Indications for treatment' above): 1:32:04 AM
•Positive catheter tip culture in the absence of clinical signs of infection
•Positive blood cultures obtained through a catheter with negative cultures through a peripheral vein
•Phlebitis in the absence of infection

Dr Kamal Deep Fortis Delhi 10:09:19 AM

catheter removal is not necessary for hemodynamically stable patients with unexplained fever in the absence of documented bloodstream infection and without endovascular prosthetic material (such as a prosthetic valve, pacemaker, or vascular graft)

The nature of the pathogen is also important for guiding decisions regarding catheter removal. Short-term catheters (indwelling <14 days) should be removed in the setting of catheter-related bloodstream infection (CRBSI) due to Staphylococcus aureus, enterococci, gram-negative bacilli, fungi, and mycobacteria. Long-term catheters (indwelling ≥14 days) should be removed in the setting of CRBSI due to S. aureus, Pseudomonas aeruginosa, fungi, or mycobacteria. Removal of long-term catheters can be a management challenge, particularly in the setting of surgically implantable intravascular devices. Therefore, it is important to establish true CRBSI. 10:11:10 AM

Salvage — Following diagnosis of catheter-related infection, catheter salvage may be attempted in the setting of uncomplicated CRBSI involving long-term catheters due to pathogens other than S. aureus, P. aeruginosa, fungi, or mycobacteria [1]. Salvage is also difficult in the setting of CRBSI due to organisms of relatively low virulence that are difficult to eradicate (eg, Bacillus spp, Micrococcus spp, or Propionibacteria). Catheter salvage in the setting of coagulase-negative staphylococcal infection does not influence resolution of bacteremia but may be a risk factor for recurrence (relative risk 6.6 in a retrospective series of 175 cases) [16].

If salvage is attempted, both systemic and antimicrobial lock therapy may be administered through the colonized catheter for the duration of therapy, depending upon the microorganism. The efficacy of antibiotic lock therapy remains uncertain and concerns have been raised about the emergence of antimicrobial resistance and fungal superinfection. The optimal antimicrobial dosing for lock therapy is also uncertain. Antibiotic lock therapy is not warranted for management of catheter infection for devices in place for <2 weeks; these are usually extraluminal infections [17].

Two sets of blood cultures should be obtained after 72 hours of appropriate antimicrobial therapy (for neonates, one set is acceptable); positive cultures should prompt catheter removal.

Catheter removal is not necessary for hemodynamically stable patients with unexplained fever in the absence of documented bloodstream infection and without endovascular prosthetic material (such as a prosthetic valve, pacemaker, or vascular graft)

Catheter removal is always necessary in the setting of fungemia. Treatment of catheter-associated fungemia without catheter removal has a low success rate and is associated with high mortality 10:12:53 AM

Coagulase-negative staphylococci are the most common cause of catheter-related infection. Interpreting blood cultures positive for coagulase-negative Staphylococcus can be challenging, since this organism is both the most common blood culture contaminant as well the most common cause of CRBSI. A high proportion of positive blood cultures performed on samples drawn from multiple sites (both peripherally and through the suspected catheter) is the best indicator for true CRBSI due to this organism 10:16:51 AM

S. aureus — In general, management of CRBSI due to S. aureus consists of catheter removal and systemic antibiotic therapy [1]. 10:19:19 AM

Following catheter removal, a new catheter may be placed if additional blood cultures demonstrate no growth at 72 hours.

Therefore, in general, transesophageal echocardiogram (TEE) should be pursued in the setting of S. aureus bacteremia to rule out IE; transthoracic echocardiography (TTE) is not sufficient for ruling out IE due to S. aureus. Possible exceptions are patients whose fever and bacteremia resolve within 72 hours following catheter removal who have no underlying cardiac predisposing conditions or clinical signs of endocarditis. Valvular vegetations and/or regurgitation may progress within the first week after onset of bacteremia; therefore, most favor performing TEE five to seven days after the onset of bacteremia 10:19:55 AM

Patients with hematogenous complications of S. aureus bacteremia should receive four to six weeks of antimicrobial therapy. Positive blood cultures 72 hours following initiation of appropriate antibiotic therapy and catheter removal are a useful predictor for hematogenous complications [9,53,57-59]. In the absence of hematogenous complications or risk factors, a shorter duration of therapy (≥14 days) is appropriate 10:21:34 AM

Patients with catheter-drawn blood culture positive for S. aureus in the setting of negative peripheral blood culture should also be observed closely. Additional blood cultures should be obtained both peripherally and through the catheter. If both repeat cultures are positive, treatment for CRBSI is warranted. If only the catheter culture is positive, the optimal approach is uncertain. The device should best be removed if feasible; otherwise, some favor 14 days of systemic antibiotic therapy with repeat blood cultures, while others favor close clinical observation with repeat blood cultures. Clinical signs of infection should prompt catheter removal. 10:22:19 AM

Salvage of CRBSI due to S. aureus using four weeks of antibiotic lock therapy combined with systemic therapy has a low success rate 10:22:36 AM

Catheter salvage should not be attempted in the setting of insertion site or pocket infection, suppurative thrombophlebitis, sepsis, endocarditis, persistent bacteremia, or metastatic infection. 10:28:16 AM

In patients with gram-negative bacillary CRBSI involving a long-term catheter and persistent bacteremia or severe sepsis despite systemic and antibiotic lock therapy, the device should be 10:29:32 AM

removed and an evaluation for metastatic infection should be pursued. Routine evaluation for IE is generally not necessary in patients with CRBSI due to gram-negative rods.

EPIC GUIDELINES:- 10:37:04 AM

Selection of catheter type IVAD6 IVAD7 IVAD8 IVAD9 Use a catheter with the minimum number of ports or lumens essential for management of the patient. Class A Preferably use a designated singlelumen catheter to administer lipidcontaining parenteral nutrition or other lipid-based solutions. Class D/GPP Use a tunnelled or implanted central venous access device with a subcutaneous port for patients in whom long-term vascular access is required. Class A Use a peripherally inserted central catheter for patients in whom mediumterm intermittent access is required. New recommendation Class D/GPP IVAD10 Use an antimicrobial-impregnated central venous access device for adult patients whose central venous catheter is expected to remain in place for >5 days if catheter-related bloodstream infection rates remain above the locally agreed benchmark, despite the implementation of a comprehensive strategy to reduce catheter-related bloodstream infe

Antimicrobial-impregnated catheters — Use of antimicrobial- or antiseptic-impregnated CVCs has been shown to reduce the incidence of catheter-related infections in adults and children 10:39:14 AM

IVAD16 Do not apply antimicrobial ointment routinely to the catheter placement site prior to insertion to prevent catheter-related bloodstream infection. Class D/GPP 10:40:29 AM

Antimicrobial lock solutions should not be used routinely to prevent catheterrelated bloodstream infections. 10:42:10 AM

Administration sets in continuous use do not need to be replaced more frequently than every 96 h, unless device-speci c recommendations from the manufacturer indicate otherwise, they become disconnected or the intravascular access device is replaced. Class A IVAD38 Administration sets for blood and blood components should be changed when the transfusion episode is complete or every 12 h (whichever is sooner). Class D/GPP IVAD39 Administration sets used for lipidcontaining parenteral nutrition should be changed every 24 h. Class D/GPP 10:43:47 AM

Dr Kamal Deep Fortis Delhi 11:56:55 AM

Patients with C. difficile carriage are a reservoir for environmental contamination in the presence or absence of clinical infection. C difficile is highly transmissible via the fecal-oral route by ingestion of spores. The organism can be cultured readily from the hospital environment, including items in patient rooms as well as the hands, clothing, and stethoscopes of healthcare workers

The antibiotics most frequently implicated in predisposition to CDAD include fluoroquinolones, clindamycin, and broad-spectrum penicillins and cephalosporins (table 1) [3,5,56-59]. However, any antibiotic can predispose to colonization by C. difficile, including metronidazole and vancomycin, which are the major antibiotics used to treat C. difficile infection [60]. The use of broad-spectrum antimicrobials, use of multiple antibiotic agents, and increased duration of antibiotic therapy all contribute to the incidence of CDAD

12:00:32 PM

Gastric acid suppression — Proton pump inhibitors (PPIs) and histamine 2 receptor antagonists have been associated with an increased risk of CDAD

12:01:21 PM

Toxins — C. difficile releases two potent exotoxins that mediate colitis and diarrhea: toxin A ("enterotoxin") and toxin B ("cytotoxin"). The organism is not invasive, and non-toxigenic strains do not cause disease.

12:02:16 PM

Once intracellular, toxins A and B inactivate regulatory pathways mediated by Rho family proteins that are involved in cytoskeleton structure and signal transduction via guanosine triphosphate. This disruption leads to cell retraction and apoptosis (grossly visible as cell rounding in tissue culture assays and as shallow ulceration on the intestine mucosal surface) [80,81]. Both toxins also disrupt intercellular tight junctions

12:03:06 PM

Healthcare personnel should wash hands with soap and water when caring for CDI patients; this is particularly important in the setting of a C. difficile outbreak. Alcohol-based hand rub (ABHR) does not eradicate C. difficile spores. Hand washing with soap and water involves vigorous mechanical scrubbing and rinsing, so it is more effective than ABHR for physical removal of bacterial spores. However, given that physical removal of C. difficile spores via hand washing with soap and water is not as effective as ABHR for eradication of vegetative (ie, non-sporulating) bacteria, glove use is critically important for preventing transmission.

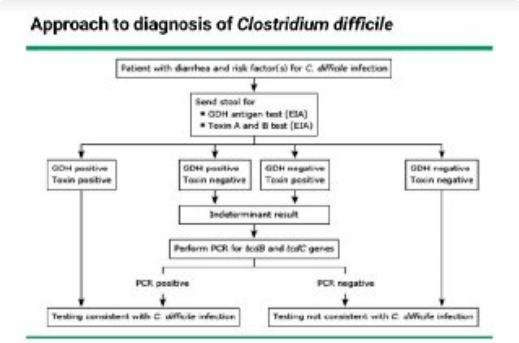
12:06:16 PM

12:07:22 PM

C. difficile spores can survive on dry surfaces for up to several months and resist killing by standard disinfectants. A disinfectant with a C. difficile sporicidal label that has been registered with the Environmental Protection Agency should be used for disinfection of patient rooms and bathrooms

Gastrointestinal colonization by nontoxigenic C. difficile strains has been shown to prevent CDI with exposure to a toxigenic strain

12:11:42 PM



12:14:24 PM

Findings of pseudomembranous colitis (severe inflammation of the inner lining of the bowel) on radiographic or endoscopic examination are highly suggestive of C. difficile infection and should prompt laboratory testing if not already performed

12:15:46 PM

However, Lower gastrointestinal endoscopy is not warranted in patients with classic clinical manifestations of C. difficile infection, a positive laboratory test, and/or clinical response to empiric treatment. In general, endoscopy may be pursued for circumstances in which an alternative diagnosis is suspected

12:16:08 PM

●For initial treatment of nonsevere CDI, we suggest oral metronidazole (Grade 2B). (See 'Initial therapy' above.)
●For treatment of severe CDI, we recommend vancomycin 125 mg four times daily for 10 to 14 days (Grade 1B). For patients with severe disease who do not demonstrate clinical improvement, we suggest treatment with oral vancomycin 500 mg four times daily (Grade 2C); fidaxomicin may be considered in patients who cannot tolerate vancomycin, although more data are needed. In critically ill patients with fulminant or refractory disease, we suggest oral vancomycin 500 mg four times daily and intravenous metronidazole 500 mg every eight hours (Grade 2C); fidaxomicin may be considered in patients who cannot tolerate vancomycin, although more data are needed. (See 'Treatment' above.)
●For treatment of severe disease in patients with profound ileus, we suggest addition of intracolonic vancomycin (Grade 2C), but

12:19:42 PM

there is risk of colonic perforation. Therefore, use of intracolonic vancomycin should be restricted to patients who are not responsive to oral therapy, and the procedure should be performed by personnel with expertise in administering enemas. (See 'Treatment' above.)
●For treatment of a nonsevere initial recurrence of CDI, we suggest oral metronidazole (Grade 2A). Alternatives include oral vancomycin or fidaxomicin. (See 'Management of initial recurrence' above.)
●For treatment of a second recurrence of CDI, we suggest intermittent and tapering vancomycin therapy or fidaxomicin (table 1) (Grade 2B). For treatment of subsequent recurrences of CDI, we suggest administering either fidaxomicin or vancomycin followed by rifaximin (Grade 2C). (See 'Management of subsequent recurrences' above.)
●We recommend urgent surgical evaluation for patients with a white blood cell count ≥20,000 cells/microL and/or a plasma lactate between 2.2 and 4.9 mEq/L (Grade 1B). In addition, surgical intervention should be strongly considered in the setting of peritoneal signs, severe ileus, or toxic megacolon. (See 'Surgery' above.)
●Potential alternative therapies requiring further investigation prior to routine use include new antibiotic agents, binding resins, intravenous immune globulin, and fecal bacteriotherapy

Treatment of C. difficile is not indicated in patients who have a positive toxin assay but are asymptomatic 12:23:55 PM

Intravenous vancomycin has no effect on C. difficile colitis since the antibiotic is not excreted appreciably into the colon. 12:25:41 PM

intravenous metronidazole at a dose of 500 mg every eight hours may also be used for treatment of CDI in patients in whom oral therapy is not feasible. Fecal concentrations in the therapeutic range are achievable with this regimen because of the drug's biliary excretion and increased exudation across the intestinal mucosa during CDI 12:26:04 PM

Antibiotic resistance does not appear to be a factor in recurrence. However, treatment with metronidazole or vancomycin for an initial episode of CDI may alter the colonic microenvironment (with regard to flora or other factors), potentially increasing susceptibility to reinfection and recurrence. 12:27:49 PM

The mechanism of CDI recurrence following initial infection is not fully understood. It may be due to persistent spores from the initial infection. C. difficile spores in colonic diverticula, for example, may escape mechanical clearance by peristalsis 12:28:04 PM

12:29:12 PM

Nonsevere initial recurrence following therapy for CDI can be treated with metronidazole. The decision to administer vancomycin as treatment for a first recurrence should be based upon the presence of markers of severe disease at the time of first recurrence rather than on previous drug exposure.

There are no rigorous studies of management for multiple recurrences of CDI. Patients with multiple recurrences may benefit from vancomycin (administered in a pulse tapered fashion), fidaxomicin, or rifaximin, with or without the use of probiotics 12:30:00 PM

Administration of vancomycin in a pulse tapered fashion may be effective for management of recurrent C. difficile infection (table 1). The use of intermittent antibiotic therapy is based upon a theory that relapse may be due to the presence of persistent spores that survive antibiotic therapy. Intermittent therapy may allow the spores to germinate on the days when no antibiotics are administered. Once the spores have converted to the fully functional vegetative, toxin-producing forms, they are susceptible to killing when the antibiotics are readministered. 12:30:36 PM

Studies of probiotics are inconclusive regarding treatment benefit. 12:31:24 PM

Antibiotics — We are in agreement with guidelines issued after 2010 that recommend oral vancomycin as first-line therapy for severe CDI [1,10,12]. This is in contrast with previous guidelines published in the 1990s that recommended oral metronidazole as initial therapy for both mild and severe C. difficile–associated diarrhea (CDAD) 12:32:40 PM

This change reflects reports of frequent metronidazole failures in CDI and studies showing the superiority of oral vancomycin in severe disease 12:32:58 PM

Oral antibiotics — Oral vancomycin (125 mg four times daily) should be initiated promptly for severely ill patients. Some data suggest that levels achieved with higher dosing of vancomycin (500 mg four times daily) may be equivalent to levels with standard dosing [31]. Nevertheless, many clinicians favor higher dosing for severe disease, although there is no supportive evidence 12:34:16 PM

Fecal bacteriotherapy may be useful for treatment of patients with recurrent CDI 12:35:45 PM

Dr Kamal Deep Fortis Delhi 2:07:54 PM

The most common type of burn in children is from a scald injury; in adults, the most common burn occurs from a flame.

The depth of the burn injury is related to contact temperature, duration of contact of the external heat source, and the thickness of the skin. Because the thermal conductivity of skin is low, most thermal burns involve the epidermis and part of the dermis [2]. The most common thermal burns are associated with flames, hot liquids, hot solid objects, and steam. The depth of the burn largely determines the healing potential and the need for surgical grafting

2:08:40 PM

Contact with acid produces tissue coagulation, while alkaline burns generate colliquation necrosis

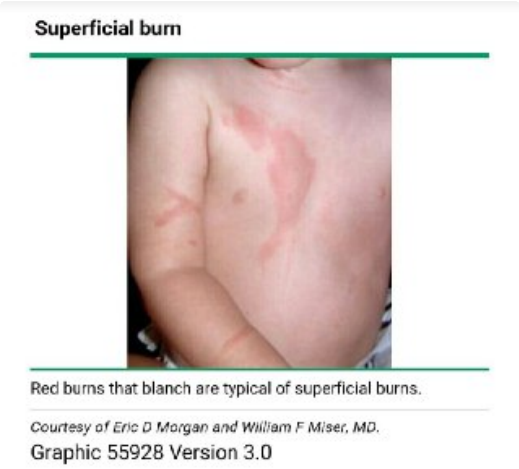
2:09:42 PM

Electrical energy is transformed into thermal injury as the current passes through poorly conducting body tissues. Electroporation (injury to cell membranes) disrupts membrane potential and function. The magnitude of the injury depends on the pathway of the current, the resistance to the current flow through the tissues, and the strength and duration of the current flow.

2:10:21 PM

Dr Kamal Deep Fortis Delhi

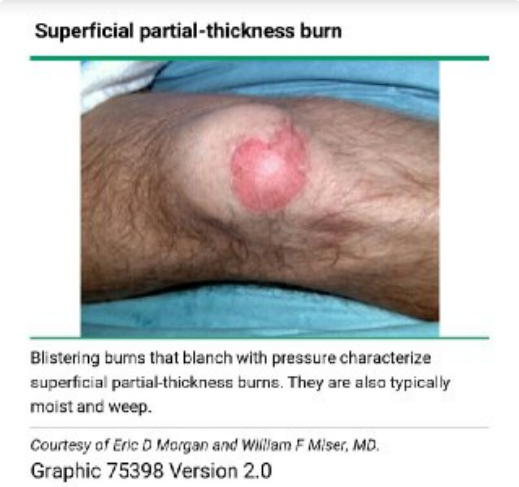
2:42:38 PM



Superficial or epidermal burns involve only the epidermal layer of skin. They do not blister but are painful, dry, red, and blanch with pressure (picture 1). Over the next two to three days the pain and erythema subside, and by about day four, the injured epithelium peels away from the newly healed epidermis. Such injuries are generally healed in six days without scarring. This process is commonly seen with sunburns.

2:43:00 PM

2:43:23 PM



Blistering burns that blanch with pressure characterize superficial partial-thickness burns. They are also typically moist and weep.

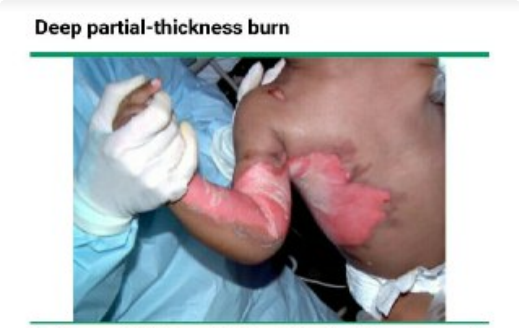
Courtesy of Eric D Morgan and William F Miser, MD.

Graphic 75398 Version 2.0

Superficial Partial Thickness burns Superficial - These burns characteristically form blisters within 24 hours between the epidermis and dermis. They are painful, red, and weeping, and blanch with pressure (picture 2). Burns that initially appear to be only epidermal in depth may be determined to be partial-thickness 12 to 24 hours later. These burns generally heal in 7 to 21 days; scarring is unusual, although pigment changes may occur 2:44:04 PM

These burns typically heal without functional impairment or hypertrophic scarring. 2:44:36 PM

2:45:04 PM



Easily unroofed blisters that do not blanch with pressure and have a waxy appearance typify deep partial-thickness burns.

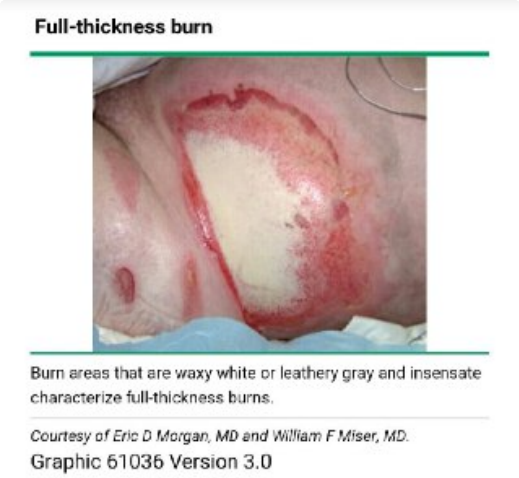
Courtesy of Eric D Morgan and William F Miser, MD.

Graphic 57851 Version 3.0

Deep - These burns extend into the deeper dermis and are characteristically different from superficial partial-thickness burns. Deep burns damage hair follicles and glandular tissue. They are painful to pressure only, almost always blister (easily unroofed), are wet or waxy dry, and have variable mottled colorization from patchy cheesy white to red (picture 3). They do not blanch with pressure. 2:45:37 PM

If infection is prevented and wounds are allowed to heal spontaneously without grafting, they will heal in three to nine weeks. These burns invariably cause hypertrophic scarring. If they involve a joint, joint dysfunction is expected even with aggressive physical therapy. A deep partial-thickness burn that fails to heal in three weeks is functionally and cosmetically equivalent to a full thickness burn. Differentiation from full-thickness burns is often difficult.

2:46:20 PM



Burn areas that are waxy white or leathery gray and insensate characterize full-thickness burns.

Courtesy of Eric D Morgan, MD and William F Miser, MD.

Graphic 61036 Version 3.0

Full-thickness — These burns extend through and destroy all layers of the dermis and often injure the underlying subcutaneous tissue. Burn eschar, the dead and denatured dermis, is usually intact. The eschar can compromise the viability of a limb or torso if circumferential. 2:46:44 PM

Full thickness burns are usually anesthetic or hypoesthetic. Skin appearance can vary from waxy white to leathery gray to charred and black. The skin is dry and inelastic and does not blanch with pressure (picture 4). Hairs can easily be pulled from hair follicles. Vesicles and blisters do not develop.

Pale full-thickness burns may simulate normal skin except that the skin does not blanch with pressure. Features that differentiate partial-thickness from full-thickness burns may take some time to develop.

The eschar eventually separates from the underlying tissue and reveals an unhealed bed of granulation tissue. Without surgery, these wounds heal by wound contracture with epithelialization around the wound edges. Scarring is severe with contractures; complete spontaneous healing is not possible.

Superficial burns are not included in the TBSA burn assessment. 2:47:58 PM

Cutaneous burns are classified according to the depth of tissue injury: superficial or epidermal (first-degree), partial-thickness (second degree), or full thickness (third degree). Burns extending beneath the subcutaneous tissues and involving fascia, muscle and/or bone are considered fourth degree

2:56:54 PM

The use of prophylactic antibiotics is controversial. A systematic review found no benefit for prophylactic antibiotics in reducing the incidence of burn wound infection

2:59:50 PM

Measures for attenuating hypermetabolism in severe burn patients include (see 'Burn wound management' below):

3:01:46 PM

●Early excision and coverage of burn wounds
●Increased ambient temperature to decrease resting energy expenditure
●Ensure adequate ventilation
●Effective pain management
●For children with severe burn injury, we suggest treatment with propranolol.
●For children and adults with severe burns, we recommend oxandrolone, initiated upon admission and sustained during the acute burn hospitalization.
●Physical therapy, which should be initiated upon admission
●Early initiation of enteral nutrition
●Prevention of infection and/or early initiation of appropriate antimicrobial coverage

Dr Kamal Deep Fortis Delhi

3:37:22 PM

Airway – Patients with upper airway burns should be intubated early, before airway anatomy becomes distorted by edema. Soot in the mouth, facial burns, and body burns may be more useful predictors of inhalation injury than symptoms of stridor, hoarseness, drooling, and dysphagi

3:39:08 PM

Chemical burns require copious irrigation. Life-threatening hypocalcemia can develop as the result of exposure to concentrated or anhydrous hydrofluoric acid 3:39:28 PM

Escharotomy — Circumferential partial and full thickness burns may lead to functional impairment (such as restriction of chest wall movement for chest burns or development of a compartment syndrome for extremity burns) as edema increases over the first 24 hours following a burn injury. An emergency escharotomy (which involves making an incision completely through the depth of the burn eschar) may be required to relieve restriction (as with chest burns) or reduce pressure (as with a compartment syndrome) 3:49:44 PM

Common signs of significant smoke inhalation injury and the potential need for intubation include: 3:52:26 PM

●Persistent cough, stridor, or wheezing
●Hoarseness
●Deep facial or circumferential neck burns
●Nares with inflammation or singed hair
●Carbonaceous sputum or burnt matter in the mouth or nose
●Blistering or edema of the oropharynx
●Depressed mental status, including evidence of drug or alcohol use
●Respiratory distress
●Hypoxia or hypercapnia
●Elevated carbon monoxide and/or cyanide levels

Injury from the inhalation of hot gases generally occurs above the vocal cords and can cause significant edema 3:53:32 PM

According to the American Burn Association's practice guidelines, any patient with greater than 15 percent total body surface area (TBSA) nonsuperficial burns should receive formal fluid resuscitation

3:55:30 PM

Antibiotics — Topical antibiotics are applied to all nonsuperficial burns. If the patient is immediately transferred to a burn center, burns are covered with clean, dry dressings and antibiotics are applied at the burn center.

4:00:40 PM

4:02:22 PM

Dr Kamal Deep Fortis Delhi

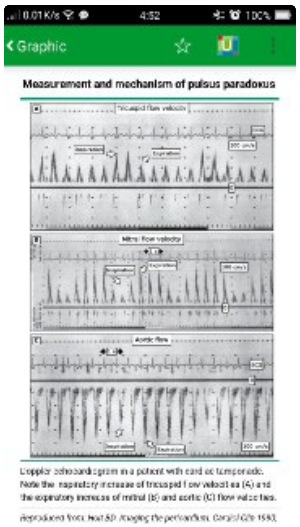
4:40:35 PM

An important pathophysiologic feature of both cardiac tamponade and constrictive pericarditis is greatly enhanced ventricular interaction or interdependence, in which the hemodynamics of the left and right heart chambers are directly influenced by each other to a much greater degree than normal.

Elevated jugular venous pressure — The jugular venous pressure is almost always elevated in cardiac tamponade and may be associated with venous distension in the forehead and scalp. The x descent is preserved, while the y descent is attenuated or absent because of the limited or absent late diastolic filling of the ventricle

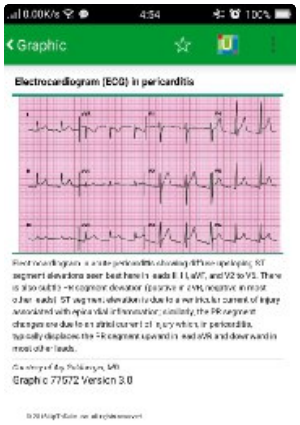
4:48:27 PM

4:52:34 PM



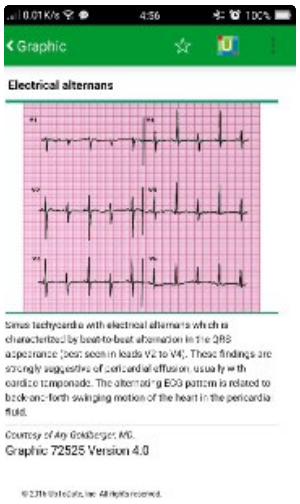
Pulsus paradoxus — Pulsus paradoxus, defined as an abnormally large decrease in systolic blood pressure (>10 mmHg) on inspiration, is a common finding in moderate to severe cardiac tamponade and is the direct consequence of ventricular interdependence. The limitation on outward expansion of the right ventricle as blood flows in during inspiration, along with relative underfilling of the left ventricle during inspiration, results in bulging of the interventricular septum into the left ventricle. Both the bulging of the interventricular septum and the reduction in left ventricular filling contribute to a large decrease in stroke volume

4:53:02 PM



4:55:02 PM

4:56:56 PM



Electrical alternans is relatively specific but not very sensitive for cardiac tamponade; rarely, this phenomenon is seen with very large pericardial effusions alone

4:57:12 PM

Chest radiograph — A chest radiograph showing an enlarged cardiac silhouette with clear lung fields may be seen in slowly developing cardiac tamponade

4:57:34 PM

Dr Kamal Deep Fortis Delhi 5:13:10 PM

Cardiomegaly is not usually seen in acute cardiac tamponade since at least 200 mL of pericardial fluid must accumulate before the cardiac silhouette enlarges

Dr Kamal Deep Fortis Delhi 9:51:48 PM

Resuscitation of the patient with acute paraquat poisoning follows standard guidelines except that oxygen therapy should not be administered unless there is confirmed hypoxia, as it may exacerbate the oxygen-mediated cellular damage.

Dr Kamal Deep Fortis Delhi 10:39:54 PM

The major diagnostic features of CIM are (table 1):

●Sensory nerve amplitudes >80 percent of the lower limit of normal in two or more nerves on NCS
●Needle EMG with short-duration, low-amplitude MUPs with early or normal full recruitment, with or without fibrillation potentials
●Absence of a decremental response on repetitive nerve stimulation
●Muscle histopathologic findings of myopathy with myosin loss
Supportive diagnostic features of CIM are:

●Motor amplitudes <80 percent of the lower limit of normal in two or more nerves without conduction block on NCS
●Elevated serum CK (best assessed in the first week of illness)
●Muscle inexcitability on direct muscle stimulation

Critical illness myopathy can be distinguished from critical illness polyneuropathy (CIP) if preservation of sensory function (indicative of the former) can be demonstrated. However, sensory evaluation can be difficult in critically ill patients, particularly when obtunded, comatose, or intubated, and it may be difficult to distinguish CIM from CIP on the basis of clinical features and neurologic examination findings alone 10:40:44 PM

The diagnosis of CIM is suspected in patients who have the clinical features cited above, particularly flaccid muscle weakness and ventilatory failure, in the setting of critical illness. Exposure to intravenous glucocorticoids is an important clue. 10:41:04 PM

Clinical features of CIP — Critical illness polyneuropathy usually occurs in patients who are in the ICU for one or especially two weeks or more. The clinical features can overlap with those of critical illness myopathy. In many cases, patients with CIP require prolonged weaning from mechanical ventilation [46,48]. 10:43:36 PM

Affected patients manifest a sensorimotor polyneuropathy characterized clinically by [35]:

●Limb muscle weakness and atrophy
●Reduced or absent deep tendon reflexes
●Loss of peripheral sensation to light touch and pin prick
●Relative preservation of cranial nerve function

n patients with CIP, direct needle stimulation of muscle elicits a relatively higher amplitude response compared with the response recorded from muscle after nerve stimulation. In contrast, in critical illness myopathy, there are proportionally low direct muscle- and nerve-evoked responses. 10:44:51 PM

Saturday, April 2, 2016

Dr Kamal Deep Fortis Delhi 1:01:52 PM

Neuromuscular toxicity is the most consistently observed complication of hypermagnesemia. Increased magnesium decreases impulse transmission across the neuromuscular junction, producing a curare-like effect

Magnesium infusion — Parenteral magnesium is commonly used to decrease neuromuscular excitability in pregnant women with severe preeclampsia or eclampsia. The usual plasma concentration achieved is 5 to 7 meq/L (6 to 8.4 mg/dL or 2.5 to 3.5 mmol/L), but much higher levels can occur 1:07:24 PM

Severe renal impairment — Dialysis is often required in patients with severe or symptomatic hypermagnesemia who have advanced CKD (patients with an eGFR less than 15 mL/min/1.73 m2 or who are on chronic dialysis), and in patients who have moderate to severe AKI. 1:10:40 PM

Moderate renal impairment — Renal elimination of magnesium may be limited in patients with moderate renal impairment, such as patients with chronic kidney disease (CKD) who have an estimated glomerular filtration rate (eGFR) between 15 and 45 mL/min/1.73 m2, and patients with mild acute kidney injury (AKI), especially if their serum creatinine concentration is increasing progressively. In most such cases, initial treatment consists of cessation of magnesium-containing medications and therapy with intravenous isotonic fluids (eg, normal saline) plus a loop diuretic (eg, furosemide). Higher diuretic doses may be required in these patients since they have reduced glomerular filtration rate (GFR) 1:11:16 PM

ECG changes are usually seen at concentrations of 5 to 10 meq/L (6 to 12 mg/dL or 2.5 to 5 mmol/L). These changes include prolongation of the P-R interval, an increase in QRS duration, and an increase in Q-T interval. Complete heart block and cardiac arrest may occur at a plasma magnesium concentration above 15 meq/L (18 mg/dL or 7.5 mmol/L). 1:16:38 PM

The kidney is crucial in maintaining the normal plasma magnesium concentration in the narrow range of 0.7 to 1.1 mmol/L. 1:21:28 PM

Magnesium 1.8-3.6 mg/dL; 1.5-3.0 mEq/L 1:21:54 PM

Above are normal levels 1:22:04 PM

Dr Kamal Deep Fortis Delhi 3:11:52 PM

Six units of whole blood-derived or one unit of apheresis-derived platelets will raise the platelet count by approximately 30,000/microL in an average sized adult.

Acute colonic pseudo-obstruction (Ogilvie's syndrome) is a disorder characterized by acute dilatation of the colon in the 3:18:02 PM

absence of an anatomic lesion that obstructs the flow of intestinal contents. Acute colonic pseudo-obstruction usually involves the cecum and right hemicolon, although occasionally colonic dilation extends to the rectum

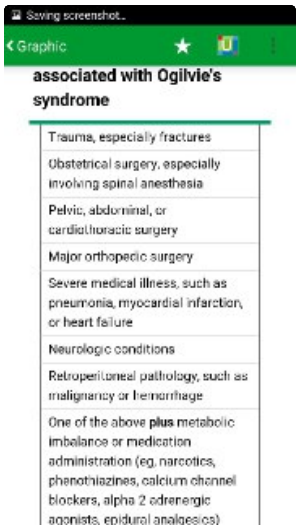
The diagnosis of acute intestinal pseudo-obstruction is established by abdominal imaging (eg, abdominal computed tomography [CT] scan or contrast enema) that helps to exclude a mechanical obstruction. Colonoscopy should not be used to make the diagnosis of acute intestinal pseudo-obstruction, as insufflation of air may increase the colonic dilatation 3:19:19 PM

Given the risk of colonic ischemia and perforation, patients with acute colonic pseudo-obstruction should be carefully monitored with serial physical examinations and plain abdominal radiographs every 12 to 24 hours 3:19:49 PM

3:20:20 PM

. A "cut-off sign" (lack of gas in the distal colon or rectum) or small bowel air-fluid levels on abdominal radiographs can also be seen in patients with acute colonic pseudo-obstruction. However, a mechanical obstruction can be differentiated from acute colonic pseudo-obstruction by visualization of an obstructing lesion on abdominal CT or contrast enema 3:26:00 PM

3:34:00 PM



Dr Kamal Deep Fortis Delhi 5:28:28 PM

PATHOGENESIS — Tetanus occurs when spores of Clostridium tetani, an obligate anaerobe normally present in the gut of mammals and widely found in soil, gains access to damaged human tissue. After inoculation, C. tetani transforms into a vegetative rod-shaped bacterium and produce the metalloprotease tetanospasmin (also known as tetanus toxin).

After reaching the spinal cord and brainstem via retrograde axonal transport [13,14] and binding tightly and irreversibly to receptors at these sites [13,15], tetanus toxin blocks neurotransmission by its cleaving action on membrane proteins involved in neuroexocytosis [16,17]. The net effect is disinhibition of neurons that modulate excitatory impulses from the motor cortex. Disinhibition of anterior horn cells and autonomic neurons results in increased muscle tone, painful spasms, and widespread autonomic instability.

Lack of neural control of adrenal release of catecholamines induced by tetanus toxin produces a hypersympathetic state that manifests as sweating, tachycardia, and hypertension 5:29:16 PM

Tetanus toxin-induced effects on anterior horns cells, the brainstem, and autonomic neurons are long lasting because recovery requires the growth of new axonal nerve terminals. 5:29:36 PM

Incubation period — The incubation period of tetanus can be as short as 2 days or as long as 38 days, with most cases occurring a mean of 7 to 10 days following exposure 5:32:40 PM

5:33:02 PM

The most common and severe clinical form of tetanus is generalized tetanus. The presenting symptom in more than half of such patients is trismus (lockjaw),

Since patients with tetanus have no impairment of consciousness or awareness, both the tonic contractions and spasms are intensely painful. T 5:33:50 PM

Tetanic spasms may be triggered by loud noises or other sensory stimuli such as physical contact or light 5:34:10 PM

onset of illness is typically more rapid in neonatal tetanus than in older individuals and may progress over hours rather than days, probably because axonal length is proportionately shorter in infants 5:36:24 PM

Duration of illness — Tetanus toxin-induced effects are long lasting because recovery requires the growth of new axonal nerve terminals. The usual duration of clinical tetanus is four to six weeks. 5:37:30 PM

administration of an anticholinergic agent such as benztropine mesylate will usually immediately reverse the spasms seen in drug-induced dystonias. Such therapy has no effect on patients with tetanus. 5:38:26 PM

Dr Kamal Deep Fortis Delhi 5:54:12 PM

Although antibiotics probably play a relatively minor role in the management of tetanus, they are universally recommended. However, it is important to emphasize that appropriate antimicrobial therapy may fail to eradicate C. tetani unless adequate wound debridement is performed.

The use of passive immunization to neutralize unbound toxin is associated with improved survival, and it is considered to be standard treatment. 5:55:30 PM

Active immunization — Since tetanus is one of the few bacterial diseases that does not confer immunity following recovery from acute illness, all patients with tetanus should receive active immunization with a total of three doses of tetanus and diphtheria toxoid spaced at least two weeks apart, commencing immediately upon diagnosis. Tetanus toxoid should be administered at a different site than tetanus immune globulin. It should be assumed that anyone who is not adequately vaccinated or protected against tetanus is also inadequately protected against diphtheria 5:57:18 PM

Muscle spasms are controlled with sedation (usually benzodiazepines) or neuromuscular blockade. (See 'Control of muscle spasms' above.)
●Autonomic hyperactivity can be treated with labetalol or morphine sulfate. Beta blockade without concomitant alpha blockade should be avoided. The use of magnesium sulfate for both autonomic dysfunction and additional control of muscle spasms has generated considerable interest. This drug is readily available and is used worldwide for the treatment of eclampsia. 6:08:12 PM

Dr Kamal Deep Fortis Delhi 6:51:12 PM

In acetaminophen-induced acute liver failure, there are two broad criteria for referral for orthotopic liver transplantation:

●An arterial pH of less than 7.30, irrespective of grade of encephalopathy or
●Grade III or IV encephalopathy with both a prothrombin time (PT) greater than 100 seconds and a serum creatinine concentration greater than 3.4 mg/dL (301 micromol/L)
For other causes of acute liver failure, referral for orthotopic liver transplantation is indicated for:

●A PT greater than 100 seconds, irrespective of the grade of encephalopathy or
●Any three of the following:
•Age less than 10 or greater than 40 years
•Unfavorable disease etiology, such as non-A, non-B viral hepatitis, idiosyncratic drug reactions, Wilson disease
•Duration of jaundice before development of encephalopathy greater than seven days
•PT greater than 50 seconds
•Serum bilirubin greater than 18 mg/dL (308 micromol/L)

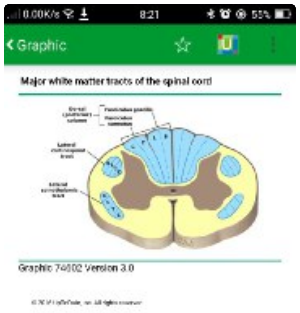
Dr Kamal Deep Fortis Delhi 8:18:37 PM

The peripheral nerves are made up of large myelinated fibers that transmit proprioceptive, vibratory, pressure, and touch stimuli, and small, poorly myelinated fibers that transmit pain, temperature, and touch stimuli. (Note that touch is transmitted by both large and small fibers.) Projections from the cell bodies receiving pain, temperature, and touch stimuli enter the spinal cord via the dorsal nerve root. These fibers terminate in the dorsal horns, fanning out over several segments [2]. They synapse with the second order neurons in the dorsal horns. These neurons then cross the midline of the cord in the anterior commissure in front of the central canal, and these second order

neurons ascend (now on the opposite side of the spinal cord to the peripheral nerve) in either the anterior spinothalamic tract (touch) or the lateral spinothalamic tract (pain and temperature) to the ventral posterolateral nucleus of the thalamus. Here, they synapse with neurons which ascend to the primary sensory cortex in the parietal lobe (figure 1).

Projections from the dorsal root ganglia that carry proprioceptive, vibratory, pressure, and touch stimuli directly enter the dorsal columns from the dorsal roots [1]. The gracile column is medial and carries stimuli from the lumbar and thoracic region; the cuneate column begins laterally in the cervical region, which it subserves. Thus, the dorsal columns consist of first order neurons traveling ipsilateral to the peripheral nerve from which they originate. These neurons synapse with second order neurons in the cuneate and gracile nuclei of the medulla. These second order neurons cross in the dorsal midline of the medulla and ascend through the brainstem as the medial lemniscus to the ventral posterolateral nucleus of the thalamus where they synapse with third order neurons which project through the internal capsule and the centrum semiovale to the primary sensory cortex in the parietal lobe

Dr Kamal Deep Fortis Delhi 10:19:20 PM



Complete cord injury — In a complete cord injury (ASIA grade A), there will be a rostral zone of spared sensory levels (eg, the C5 and higher dermatomes spared in a C5-6 fracture-dislocation), reduced sensation in the next caudal level, and no sensation in levels below, including none in the sacral segments, S4-S5. Similarly, there will be reduced muscle power in the level immediately below the injury, followed by complete paralysis in more caudal myotomes. In the acute stage, reflexes are absent, there is no response to plantar stimulation, and muscle tone is flaccid. A male with a complete TSCI may have priapism. The bulbocavernosus reflex is usually absent. Urinary retention and bladder distension occur. (See "Anatomy and localization of 10:20:14 PM

spinal cord disorders", section on 'Segmental syndrome'.)

Incomplete injury — In incomplete injuries (ASIA grades B through D), there are various degrees of motor function in muscles controlled by levels of the spinal cord caudal to the injury. Sensation is also partially preserved in dermatomes below the area of injury. Usually sensation is preserved to a greater extent than motor function because the sensory tracts are located in more peripheral, less vulnerable areas of the cord. The bulbocavernosus reflex and anal sensation are often present.

The relative incidence of incomplete versus complete spinal cord injury has increased over the last half century [1]. This trend has been attributed to improved initial care and retrieval systems that emphasize the importance of immobilization after injury (see 'Initial evaluation and treatment' below).

Central cord syndrome — An acute central cord syndrome, characterized by disproportionately greater motor impairment in upper compared with lower extremities, bladder dysfunction, and a variable degree of sensory loss below the level of injury, is described after relatively mild trauma in the setting of preexisting cervical spondylosis

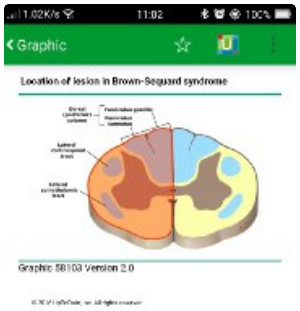
10:23:08 PM

Anterior cord syndrome — Lesions affecting the anterior or ventral two-thirds of the spinal cord, sparing the dorsal columns, usually reflect injury to the anterior spinal artery. When this occurs in TSCI, it is believed that this more often represents a direct injury to the anterior spinal cord by retropulsed disc or bone fragments rather than primary disruption of the anterior spinal artery.

10:24:04 PM

Dr Kamal Deep Fortis Delhi

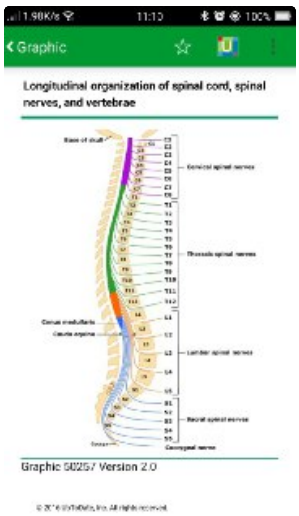
11:02:36 PM



11:02:58 PM

Brown-Sequard (hemi-cord) syndrome — A lateral hemisection syndrome, also known as the Brown–Sequard syndrome, involves the dorsal column, corticospinal tract, and spinothalamic tract unilaterally (figure 11). This produces weakness, loss of vibration, and proprioception ipsilateral to the lesion and loss of pain and temperature on the opposite side. The unilateral involvement of descending autonomic fibers does not produce bladder symptoms. While there are many causes of this syndrome, knife or bullet injuries and demyelination are the most common. Rarer causes include spinal cord tumors, disc herniation, infarction, and infections

11:10:18 PM



11:19:46 PM

Autonomic bladder control is primarily parasympathetic, and is unaffected by isolated injury to the sympathetic fibers. Voluntary bladder control is under somatomotor control, mediated by motor fibers originating from the anterior horn cells at levels S2-S4. A spinal cord lesion that interrupts descending motor and autonomic tracts above the S2 level produces an "automatic bladder" that cannot be emptied voluntarily, but empties reflexly when expanded to a certain degree, the so-called neurogenic bladder [7-10]. Loss of descending inhibition of segmental reflex control leads to urinary urgency and incontinence. Injury to S2-S4 spinal levels interrupts the bladder reflex circuit; the bladder becomes flaccid, and fills beyond capacity with overflow incontinence.

Sunday, April 3, 2016

Dr Kamal Deep Fortis Delhi 12:38:55 AM

degree of hyperthyroidism (elevation of T4 and/or T3 and suppression of TSH) in patients with thyroid storm is, in general, comparable to that in patients with uncomplicated overt

hyperthyroidism. Thus, the degree of hyperthyroidism is not a criterion for diagnosing thyroid storm

The principles of treatment outlined below are based upon clinical experience and case studies, since there are no prospective studies. They are frequently also applied to patients with severe hyperthyroidism who do not fully meet the criteria for thyroid storm. The therapeutic regimen typically consists of multiple medications, each of which has a different mechanism of action [7,9,10]: 12:48:04 AM

●A beta blocker to control the symptoms and signs induced by increased adrenergic tone
●A thionamide to block new hormone synthesis
●An iodine solution to block the release of thyroid hormone
●An iodinated radiocontrast agent (if available) to inhibit the peripheral conversion of thyroxine (T4) to triiodothyronine (T3)
●Glucocorticoids to reduce T4-to-T3 conversion, promote vasomotor stability, and possibly treat an associated relative adrenal insufficiency
●Bile acid sequestrants to decrease enterohepatic recycling of thyroid hormones

Dr Kamal Deep Fortis Delhi 4:22:22 PM

When used to treat acute infections, HBO should be implemented early, with two to three daily 90 minute HBO sessions at 3 atm. These high pressures maintain tissue oxygen tension above 300 mmHg (sufficient to inhibit clostridial spore and exotoxin production)

Dr Kamal Deep Fortis Delhi 10:50:06 PM

Other disadvantages of epinephrine include dysrhythmias (due to beta-1 adrenergic receptor stimulation) and splanchnic vasoconstriction. The degree of splanchnic vasoconstriction appears to be greater with epinephrine than with equipotent doses of norepinephrine or dopamine in patients with severe shock [18], although the clinical importance of this is unclear

At doses >10 mcg/kg per minute, the predominant effect of dopamine is to stimulate alpha-adrenergic receptors and produce vasoconstriction with an increased SVR [24,25]. However, the overall alpha-adrenergic receptor effect of dopamine is weaker than that of norepinephrine, and the beta-1 adrenergic receptor stimulation of dopamine at doses >2 mcg/kg per minute can result in dose-limiting dysrhythmias. 10:54:00 PM

Isoproterenol — Isoproterenol (Isuprel) also is primarily an inotropic and chronotropic agent rather than a vasopressor. It acts upon beta-1 adrenergic receptors and, unlike dobutamine, has a prominent chronotropic effect. 10:59:36 PM

Rebound hypotension appears to be common following withdrawal of vasopressin. To avoid rebound hypotension, the dose is slowly tapered by 0.01 units/min every 30 minutes. 11:02:38 PM

PDE inhibitors — Phosphodiesterase (PDE) inhibitors, such as inamrinone (formerly known as amrinone) and milrinone, are nonadrenergic drugs with inotropic and vasodilatory actions. In many ways, their effects are similar to those of dobutamine but with a lower incidence of dysrhythmias. 11:03:46 PM

Monday, April 4, 2016

Dr Kamal Deep Fortis Delhi 11:50:30 AM

Concerning findings associated with the use of mannitol include serum sodium >150 meq, serum osmolality >320 mOsm, or evidence of evolving acute tubular necrosis (ATN).

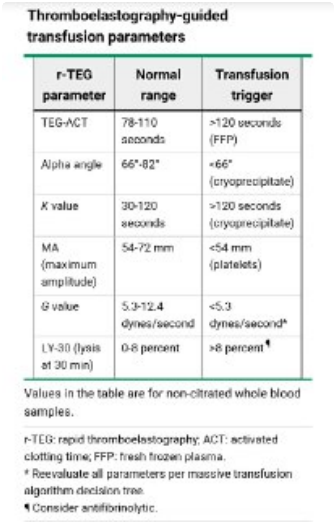
Hyperventilation should be minimized in patients with traumatic brain injury or acute stroke. In these settings, vasoconstriction may cause a critical decrease in local cerebral perfusion and worsen neurologic injury, particularly in the first 24 to 48 hours 11:54:06 AM

Indications for ICP monitoring in TBI are a GCS score ≤8 and an abnormal CT scan showing evidence of mass effect from lesions such as hematomas, contusions, or swelling [75]. ICP monitoring in severe TBI patients with a normal CT scan may be indicated if two of the following features are present: age >40 years; motor posturing; systolic BP <90 mmHg 11:58:02 AM

Dr Kamal Deep Fortis Delhi 12:32:04 PM

Because the R value on the TEG represents the time it takes for clot formation to start, it is a reflection of coagulation factor activity. Coagulation factors are essentially enzymes that drive clot formation. Thus, a prolonged R time should be treated with plasma. The alpha angle represents the thrombin burst and conversion of fibrinogen to fibrin. Thus, a depressed alpha angle should be treated with either cryoprecipitate or large volumes of plasma. 80% of the MA is derived from platelet function whereas the remaining 20% is derived from fibrin. Thus, a depressed MA is better treated with platelet transfusion or medications that improve platelet function, such as DDAVP. An elevated EPL or

LY30 suggests fibrinolysis and may be treated with an antifibrinolytic, such as tranexamic acid or aminocaproic acid, in the appropriate clinical setting.



Thromboelastography-guided transfusion parameters

r-TEG parameter	Normal range	Transfusion trigger
TEG-ACT	78-110 seconds	>120 seconds (FFP)
Alpha angle	66°-82°	<66° (cryoprecipitate)
K value	30-120 seconds	>120 seconds (cryoprecipitate)
MA (maximum amplitude)	54-72 mm	<54 mm (platelets)
G value	5.3-12.4 dynes/second	<5.3 dynes/second*
LY-30 (lysis at 30 min)	0-8 percent	>8 percent¶

Values in the table are for non-citrated whole blood samples.

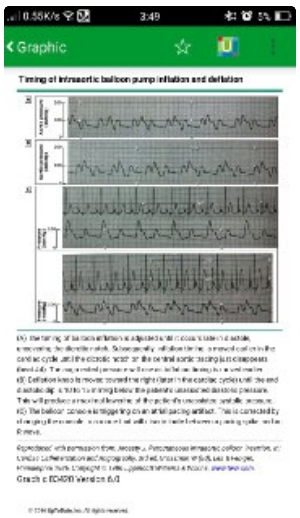
r-TEG: rapid thromboelastography; ACT: activated clotting time; FFP: fresh frozen plasma.
* Reevaluate all parameters per massive transfusion algorithm decision tree.
¶ Consider antifibrinolytic.

12:33:36 PM

Dr Kamal Deep Fortis Delhi 3:52:36 PM

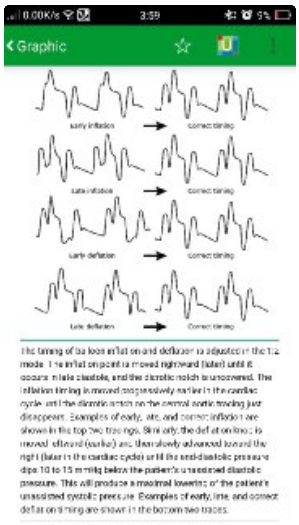
Inflation and deflation of the balloon has two major consequences:

●Blood is displaced to the proximal aorta by inflation during diastole.
●Aortic volume (and thus afterload) is reduced during systole through a vacuum effect created by rapid balloon deflation.



3:52:58 PM

4:00:06 PM



Increased blood flow is most likely to occur in coronary vascular beds maximally dilated by ischemia, a setting in which autoregulation is at a maximal level and flow becomes pressure-dependent

4:04:56 PM

Expected changes in the hemodynamic profile in the majority of patients with cardiogenic shock include: a decrease in systolic pressure; an increase in diastolic pressure, which may raise coronary blood flow to territory perfused by a vessel with a critical stenosis; a reduction of the heart rate; a decrease in the mean pulmonary capillary wedge pressure; and an elevation in the cardiac output

4:13:50 PM

The 2004 ACC/AHA guidelines on ST elevation MI made the following recommendations, which were not changed in the 2009 focused update, for the use of IABP in the following settings [17,29]:

4:15:48 PM

●Cardiogenic shock that is not quickly reversed with pharmacologic therapy. (See "Prognosis and treatment of cardiogenic shock complicating acute myocardial infarction".)
●Acute mitral regurgitation, particularly due to papillary muscle rupture, or ventricular septal rupture. (See "Mechanical complications of acute myocardial infarction".)
●Recurrent ischemic chest discomfort with signs of hemodynamic instability, poor left ventricular function, or a large area of myocardium at risk.
In the last three settings, the use of an IABP was considered to be beneficial both for hemodynamic support and as a stabilizing measure for angiography and prompt revascularization

Dr Kamal Deep Fortis Delhi

10:04:08 PM

Table 3. The modified clinical pulmonary infection score.

CPIS Points	0	1	2
Tracheal secretions	Rare	Abundant	Abundant + purulent
Chest X-ray infiltrates	No infiltrate	Diffused	Localized
Temperature, °C	36.5 and 38.4	38.5 and 38.9	39 or 36
Leukocytes count, per mm3	4,000 and 11,000	< 4,000 or > 11,000	< 4,000 or > 11,000 + band forms 500
PAO2/FIO2, mm Hg	> 240 or ARDS		240 and no evidence of ARDS
microbiology	negative	-	positive

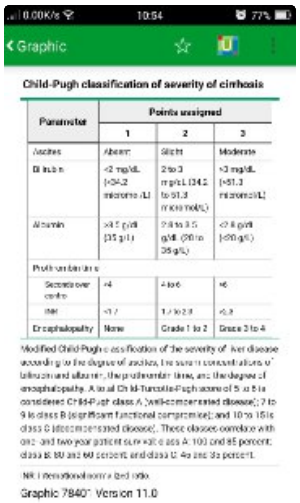
Pugin et al., introduced CPIS and found that threshold score of ≥ 6 was a fairly accurate indicator of VAP [9]. CPIS have been modified by excluding tracheal aspirate specimen culture (modified CPIS Table 3) and increasing the score to 7 [8,21,22,13,23]. Various studies have reported CPIS to have sensitivity and specificity between 77% to 93% and 17% to 100% respectively [18,21]. Fabregas et al., found that CPIS with BAL fluid >104
cfu/mL had a sensitivity of 77% and
specificity of 58% when compared with post mortem lung biopsy histology [24,25]. Similar to other studies in which culture of lower respiratory tract specimen of patients with VAP showed 60% (50%-80%) positive for microorganism [8,9]. In our study 29 VAP episodes were diagnosed using CPIS ≥ 6, out of which only 18 (66.7%) were positive for microorganism. In our study the high incidence of VAP (using CPIS score-36.7 and microbiological criteria-22.87) in neurological patients could be attributed to poor neurological condition and small sample size.
There is significant (p-0.005) correlation between the CPIS score with Pa
O2
/FiO2 (tg://bot_command?command=FiO2)
, TLC and chest X-ray in the first seven
days of intubation and MV. The parameter that significantly correlated after 7 days of MV was Pa
O2
/FiO2 (tg://bot_command?command=FiO2) and poor correlation
of TLC and chest X-ray after 7 days of MV could be due to confounding factors like presence of atelectasis, multiple invasive lines, prolonged immobilization and colonization or subclinical infection during prolonged ICU stay

10:06:54 PM

Dr Kamal Deep Fortis Delhi

10:35:20 PM

Dr Kamal Deep Fortis Delhi 11:12:12 PM



Wednesday, April 6, 2016

Dr Kamal Deep Fortis Delhi 12:07:36 AM

Effects of non reabsorbable ion in hypokalemia:-

Nonreabsorbable anions — The lumen-negative electrical gradient created by sodium reabsorption in the cortical collecting tubule is partially attenuated by chloride reabsorption. There are, however, a number of clinical settings in which sodium is presented to the distal nephron with relatively large quantities of a nonreabsorbable anion, including bicarbonate with vomiting or proximal renal tubular acidosis, beta-hydroxybutyrate in diabetic ketoacidosis, hippurate following toluene use (glue sniffing), or a penicillin derivative in patients receiving high-dose penicillin therapy. In these settings, more of the delivered sodium will be reabsorbed in exchange for potassium, leading to a potentially

marked increase in potassium excretion [59,60]. As an example, the serum potassium concentration is below 2 meq/L in approximately one-fourth of patients with toluene-induced metabolic acidosis [61]. (See "The Δanion gap/ΔHCO3 ratio in patients with a high anion gap metabolic acidosis", section on 'D-lactic acidosis and toluene inhalation'.)

The effect of nonreabsorbable anions is likely to be most prominent when there is concurrent volume depletion. In this setting, the decrease in distal chloride delivery (thereby limiting the ability of chloride reabsorption to dissipate the lumen-negative gradient) and the enhanced secretion of aldosterone both promote potassium secretion [60]. In diabetic ketoacidosis, for example, increased distal sodium and water delivery (due to the glucose osmotic diuresis), hypovolemia-induced hyperaldosteronism, and beta-hydroxybutyrate acting as a nonreabsorbable anion all can contribute to potassium wasting

Thursday, April 14, 2016

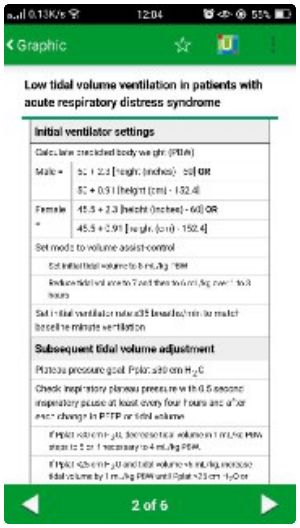
Dr Kamal Deep Fortis Delhi 9:09:26 AM

If neither ketoacidosis nor lactic acidosis is present, an elevated serum osmolal gap (greater than 10 mosmol/kg) in a patient with a high anion gap metabolic acidosis is consistent with but not diagnostic of methanol or ethylene glycol intoxication

Dr Kamal Deep Fortis Delhi 9:40:02 AM

Tilting the head back by extending the atlanto-occipital joint is helpful, but extension of the cervical spine below the atlanto-occipital joint can compromise the glottic view

Dr Kamal Deep Fortis Delhi 1:45:42 PM



Friday, April 15, 2016

1:46:24 PM

Dr Kamal Deep Fortis Delhi

The CAP treatment guidelines in the United States (summarized in Table 257-4) represent joint statements from the IDSA and the ATS; the Canadian guidelines come from the Canadian Infectious Disease Society and the Canadian Thoracic Society. In these guidelines, coverage is always provided for the pneumococcus and the atypical pathogens. In contrast, guidelines from some European countries do not always include atypical coverage based on local epidemiologic data. The U.S.–Canadian approach is supported by retrospective data from several studies of administrative databases including thousands of patients. Atypical pathogen coverage provided by the addition of a macrolide to a cephalosporin or by the use of a fluoroquinolone alone has been consistently associated with a significant reduction in mortality rates compared with those for -lactam coverage alone.

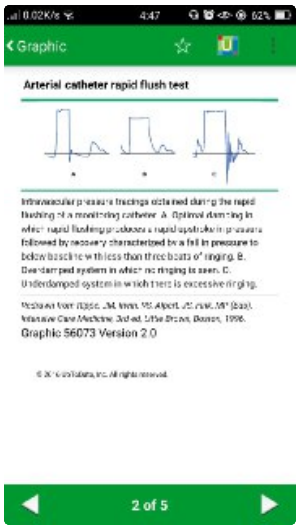
Dr Kamal Deep Fortis Delhi 4:47:22 PM

Sources of error — Despite the superiority of direct blood pressure measurement, technical problems may introduce errors [58].

Dynamic response — Dynamic response is determined by two factors, the resonant frequency and the damping coefficient:

●The resonant or natural frequency of the system is the frequency at which it oscillates when stimulated. Physiologic peripheral arterial waveforms have a fundamental frequency of 3 to 5 Hz, although some components may range up to 20 Hz [59]. Thus, the resonant frequency of the system used to monitor arterial pressure must be greater than 20 Hz to avoid ringing and systolic overshoot [60]
●The damping coefficient is a measure of how quickly an oscillating system comes to rest [60]. A system with a high damping coefficient (eg, compliant tubing) absorbs mechanical energy well and causes a diminution in the transmitted waveform. The damping coefficient and resonant frequency of a monitoring system can be assessed at the bedside by the rapid-flush test (figure 2). This test is performed by briefly opening and closing the valve in the continuous flush device, which produces a square wave on the monitor. The square wave is followed by ringing (ie, rapid variation around the baseline), and a return to baseline. No ringing is seen with an over-damped system and excessive ringing is seen with an under-damped system. Common causes of underdamping include connecting tubing with stopcocks, excessive tubing length, and patient factors (eg, tachycardia, high output states). A common cause of overdamping is air bubbles in the connecting tubing.

4:48:26 PM



Dr Kamal Deep Fortis Delhi 10:51:06 PM

HEMODYNAMIC EFFECTS — Inflation and deflation of the balloon has two major consequences:

●Blood is displaced to the proximal aorta by inflation during diastole.
●Aortic volume (and thus afterload) is reduced during systole through a vacuum effect created by rapid balloon deflation.
These effects may be quite variable, and they depend upon the volume of the balloon, its position in the aorta, heart rate, rhythm, the compliance of the aorta, and systemic resistance [7]. The higher the arterial elastance, which is determined in part by compliance, the greater the hemodynamic improvement from intraaortic balloon pump counterpulsation (IABP) [8].
Despite this variability, expected changes in the hemodynamic profile in the majority of patients with cardiogenic shock include (figure 2) [9]:

●A decrease in systolic pressure by 20 percent
●An increase in aortic diastolic pressure by 30 percent, which may raise coronary blood flow to territory perfused by a vessel with a critical stenosis
●An increase in mean arterial pressure especially in patients with shock due to an acute mechanical abnormality such as mitral regurgitation (MR) or ventricular septal defect (VSD).
●A reduction of the heart rate by less than 20 percent
●A decrease in the mean pulmonary capillary wedge pressure by 20 percent
●An elevation in the cardiac output by 20 percent, especially in patients with MR, VSD, or a large territory of medically refractory ischemia that is improved by the addition of counterpulsation

In addition, intraaortic balloon pumping reduces mean systemic impedance and developed systolic pressure, and causes a 14 percent decline in calculated peak left ventricular wall stress [10]. The reductions in afterload and wall stress lead to a fall in myocardial oxygen consumption, which is one of the goals of treatment of patients with myocardial ischemia

Saturday, April 16, 2016

Dr Kamal Deep Fortis Delhi 7:20:00 PM

Early intravenous vasodilator therapy is suggested in selected patients with ADHF who require a decrease in systemic vascular resistance and left ventricular afterload (eg, those with severe hypertension, acute mitral regurgitation, or acute aortic regurgitation). The aggressiveness of diuretic and vasodilator therapy depends on the patient's hemodynamic and volume status. Patients with flash pulmonary edema due to hypertension, for instance, require aggressive vasodilatory therapy. Patients with normotension and volume overload may be treated with diuretic therapy with or without vasodilator therapy.

In addition, furosemide and possibly other loop diuretics also have an initial morphine-like effect in acute pulmonary edema, causing venodilation that can decrease pulmonary congestion prior to the onset of diuresis [10]. This effect appears to be mediated by enhanced release of prostaglandins. 7:29:32 PM

Patients with HF with preserved left ventricular ejection fraction or restrictive physiology may be more sensitive to diuresis-induced reductions in preload. 7:31:28 PM

Changes in cardiac output and the consequent changes in renal perfusion are not the only determinant of changes in GFR in patients with HF. Among patients with an elevated central venous pressure, the associated increase in renal venous pressure can reduce the GFR, while lowering venous pressure with diuretics and other therapies might therefore increase the GFR. 7:33:14 PM

If increases in serum creatinine appear to reflect intravascular volume depletion, then reduction in or temporary discontinuation of diuretic and/or ACE inhibitor/ARB therapy should be considered. Adjunctive inotropic therapy may be required. 7:35:26 PM

Dr Kamal Deep Fortis Delhi 8:08:06 PM

If the systolic blood pressure is <85 mmHg or there is evidence of shock (eg, cool extremities, narrow pulse pressure, low urine output, confusion), addition of an inotrope is suggested [16]. In

patients with persistent shock, a vasopressor may be added as a temporizing measure to support perfusion to vital organs, though this is at the expense of increased left ventricular afterload. For selected patients with severe HFrEF (generally with left ventricular ejection fraction <25 percent) with acute, severe hemodynamic compromise, nondurable mechanical support (eg, intraaortic balloon pump [IABP], extracorporeal circulatory membrane oxygenator [ECMO], or extracorporeal ventricular assist devices) is an option as a "bridge to decision" or "bridge to recovery"

Vasopressor use should be limited to patients with persistent hypotension with symptoms or evidence of consequent end-organ hypoperfusion despite optimization of filling pressures and use of inotropic agents as appropriate. In this setting, invasive monitoring can be helpful to assess filling pressures and systemic vascular resistance. (See "Pulmonary artery catheterization: Indications, contraindications, and complications in adults", section on 'Indications'.) 8:14:16 PM

Vasopressors used in this setting include norepinephrine, high-dose dopamine (>5 micrograms/kg/min), and vasopressin,

Sunday, April 17, 2016

Dr Kamal Deep Fortis Delhi 1:18:53 PM

Norepinephrine 0.1 to 0.15 mcg/kg/minute
Epinephrine 0.014 mcg/kg/minute
Phenylephrine 100 to 180 mcg/minute until stabilized (alternatively, 0.5 to 2 mcg/kg/minute)
Dopamine 2 to 5 mcg/kg/minute
Vasopressin 0.03 units per minute
Dobutamine
0.5 to 1 mcg/kg/minute
Milrinone 0.125 to 0.75 mcg/kg/minute

Epi-.014 1:23:26 PM
Norepi:- 0.1 to .15
Milrinone:- 0.125 to 0.75
Dobutamine:-0.5 to 1
Phenylephrine:- 0.5 to 2
Dopamine:- 2-5

Tuesday, April 19, 2016

Dr Kamal Deep Fortis Delhi 4:28:50 PM

Antibiotic pharmacodynamics (PD) describes the impact of an antimicrobial agent on a target pathogen and is based on the drug's pharmacokinetics (PK) and microbiologic activity toward

that pathogen, together with the pathogen's susceptibility to the drug. Patient or host factors play an important role in antibiotic PD by affecting drug PK and patient susceptibility to infection. The 3 PD parameters commonly used to predict antibiotic efficacy are (1) the ratio of maximum serum concentration to the minimum inhibitory concentration (MIC) (Cmax/MIC); (2) the ratio of the area under the plasma concentration versus time curve (AUC) versus MIC (AUC/MIC) and (3) the duration of the dosing interval that plasma concentrations exceed the MIC (T>MIC). The Cmax/MIC ratio has been shown to predict aminoglycoside efficacy, AUC/MIC best describes fluoroquinolone, glycopeptide, and ketolide efficacy, and T>MIC best describes the efficacy of beta-lactams and macrolides. Traditionally, PK and PD (PK/PD) parameters have been used to predict antibiotic efficacy, but there is now increasing interest in trying to use PK/PD parameters to minimize development of resistance. With respect to fluoroquinolone resistance, the "mutant selection window" hypothesis has been developed to describe how drug exposures below the mutant prevention concentration may create conditions for the selection of resistant bacterial strains. The AUC/MIC ratio has also been used to describe fluoroquinolone drug exposures associated with either increased or decreased risk of resistance emergence. The accessory gene regulator (agr) locus in Staphylococcus aureus-and particularly agr group II-has been associated with reduced susceptibility or resistance of S aureus to vancomycin. Recent experiments suggest that the AUC/MIC ratio may be used to identify vancomycin exposures associated with emergence of resistance in S aureus. More generally, AUC/MIC ratios may be additive, and combination therapies may represent 1 approach to lowering the emergence rate of bacterial resistance associated with antibiotic therapy. But, further work needs to be done before this conclusion can be verified

Thursday, April 21, 2016

Dr Kamal Deep Fortis Delhi 8:35:40 PM

Assuming that the baseline serum AG is known or can be accurately estimated, the ΔAG/ΔHCO3 ratio in an uncomplicated high AG metabolic acidosis (eg, shock-induced lactic acidosis) is usually between 1 and 1.6.

In ketoacidosis, D-lactic acidosis, or toluene intoxication, the accumulating organic acid anions are excreted by the kidney as sodium and/or potassium salts. In these disorders, the ΔAG/ΔHCO3 ratio is often below 1, and the serum AG may be normal. (See 'D-lactic acidosis and toluene inhalation' above.) 8:36:21 PM

•Patients with early-stage chronic kidney disease often have relatively well-preserved acid anion excretion but more severe tubular damage, which greatly reduces urine ammonium excretion. This leads to a hyperchloremic metabolic acidosis and a ΔAG/ΔHCO3 ratio below 1. (See 'Chronic kidney disease' above.)

•Some clinical disorders, such as severe diarrhea, can generate a

combined high and normal AG acidosis. This is due to a hyperchloremic acidosis resulting from the loss of bicarbonate and potential bicarbonate salts in the stool in combination with a lactic acidosis resulting from hypovolemia and reduced tissue perfusion. (See 'The ΔAG/ΔHCO3 in patients with mixed metabolic disorders' above.)

●A higher-than-expected value (ie, ΔAG/ΔHCO3 ratio above 1.6) usually reflects a mixed acid-base disorder in which a high AG acidosis coexists with a process that elevated the serum HCO3. The high bicarbonate concentration may be due to a preexisting or concurrent metabolic alkalosis, as with vomiting or diuretic use, or may reflect the compensatory increase in serum HCO3 induced by a preexisting chronic respiratory acidosis

Dr Kamal Deep Fortis Delhi 9:45:06 PM

When the serum glucose reaches 200 mg/dL (11.1 mmol/L) in DKA or 250 to 300 mg/dL (13.9 to 16.7 mmol/L) in HHS, the IV saline solution is switched to dextrose in saline, and it may be possible to decrease the insulin infusion rate to 0.02 to 0.05 units/kg per hour [10,12,26]. Reducing the serum glucose at this time below 200 mg/dL (11.1 mmol/L) in DKA or 250 to 300 mg/dL (13.9 to 16.7 mmol/L) in HHS may promote the development of cerebral edema

During insulin therapy, beta-hydroxybutyrate is converted to acetoacetate, resulting in an increasingly positive nitroprusside test for acetoacetate as ketosis is improving (figure 1) [25]. As a result, assessments of urinary or serum ketone levels by the nitroprusside method should not be used for monitoring resolution of DKA. 9:47:00 PM

Dr Kamal Deep Fortis Delhi 10:28:54 PM

The diagnosis of ventilator-associated pneumonia (VAP) is made when a patient who has been mechanically ventilated for ≥48 hours develops a new or progressive infiltrate and the respiratory specimens are positive (ie, increased neutrophils are seen in the microscopic analysis and growth of a pathogen in culture exceeds a predefined threshold).

Myoglobin has a half-life of only two to three hours, much shorter than that of CK. Because of its rapid excretion and metabolism to bilirubin, serum levels may return to normal within six to eight hours. 10:41:18 PM

Thus, it is not unusual for CK levels to remain elevated in the absence of myoglobinuria

Saturday, April 23, 2016

Dr Kamal Deep Fortis Delhi 8:36:02 PM

Thus, during an acute asthma exacerbation, a PaCO2 of 42 mmHg or greater, while technically "normal," may suggest incipient respiratory failure. On the other hand, hypercapnia alone is not an indication for mechanical ventilation in the absence of decreased mental status or exhaustion.

Saturday, October 22, 2016

Dr Kamal Deep Fortis Delhi 1:18:50 AM

When TH is used, arterial blood gas values can be interpreted with or without correction for the patient's temperature. When a patient's core temperature is 33°C, the patient's actual PaCO2 may be 6 to 7 mmHg lower than the value reported by the blood gas machine [33]. However, there is no evidence to determine whether it is better to use corrected (pH-stat) or uncorrected (alpha-stat) values to adjust ventilation. It is prudent to target slightly higher PaCO2 values in order to avoid accidental hyperventilation and the resulting cerebral vasoconstriction.

Wednesday, November 16, 2016

Dr Kamal Deep Fortis Delhi 10:56:23 AM

ESTIMATING ENERGY AND PROTEIN REQUIREMENTS
A patient's basal energy expenditures (BEE, measured in kilocalories per day) can be estimated from height, weight, age, and gender by using the Harris-Benedict equations:

Men: BEE = 66.47 + 13.75W + 5.00H – 6.76A

Women: BEE = 655.10 + 9.56W + 1.85H – 4.68A

where W is weight in kilograms; H is height in centimeters, and A is age in years. After these equations are solved, total energy requirements are estimated by multiplying BEE by a factor that accounts for the stress of illness. Multiplying by 1.1–1.4 yields a range 10–40% above basal that estimates the 24-h energy expenditure of the majority of patients. The lower value (1.1) is used for patients without evidence of significant physiologic stress; the higher value (1.4) is appropriate for patients with marked stress such as sepsis or trauma. The result is used as a 24-h energy goal for feeding.

When it is important to have a more accurate assessment of energy expenditure, it can be measured at the bedside by using

indirect calorimetry. This technique is useful in patients who are believed to be hypermetabolic from sepsis or trauma and whose body weights cannot be obtained accurately. Indirect calorimetry can also be useful in patients who have difficulty weaning from a ventilator, as their energy needs should not be exceeded to avoid excessive CO2 production. Patients at the extremes of weight (e.g., obese persons) and/or age are good candidates as well, because the Harris-Benedict equations were developed from measurements in adults with roughly normal body weights.

Because urea is a major by-product of protein catabolism, the amount of urea nitrogen excreted each day can be used to estimate the rate of protein catabolism and determine whether protein intake is adequate to offset it. Total protein loss and protein balance can be calculated from urinary urea nitrogen (UUN) as follows:

Protein catabolic rate (g/d) = [24-h UUN (g) + 4] x 6.25 (g protein/g nitrogen)

The value of 4 g added to the UUN represents a liberal estimate of the unmeasured nitrogen lost in the urine (e.g., creatinine and uric acid), sweat, hair, skin, and feces. When protein intake is low (e.g., less than about 20 g/d), the equation indicates both the patient's protein requirement and the severity of the catabolic state (Table 75-5). More substantial protein intakes can raise the UUN because some of the ingested (or infused) protein is catabolized and converted to UUN. Thus, at lower protein intakes the equation is useful for estimating requirements, and at higher protein intakes it is useful for assessing protein balance.

Protein balance (g/d) = protein intake – protein catabolic rate

Wednesday, November 23, 2016

Dr Kamal Deep Fortis Delhi 2:35:18 PM

Describe the clinical tests for brainstem death and which cranial nerves are tested. [1 for each, max. 6]
1. Pupils are fixed and unresponsive to light – 2nd and 3rd cranial nerves.
2. There is no corneal reflex – 5th and 7th cranial nerves.
3. There is no oculo vestibular reflex: no eye movement to 50 mL ice-cold saline in each ear over 1 minute with clear tympanic membranes – 3rd , 6th and 8th cranial nerves.
4. There is no motor response to pain within cranial nerve distribution – 5th and 7th cranial nerves.
5. There are no gag or cough reflexes to stimulation – 9th and 10th cranial nerves.
6. There is apnoea to PaCO2 > 6.65kPa (confirmed with arterial blood gas).

DELETED 2:37:18 PM

Thanx

Dr Kamal Deep Fortis Delhi 5:12:04 PM

Unhelpful and contraindicated therapies

Enhanced elimination — Several case reports describe the successful use of various therapies for TCA toxicity, including plasma exchange and charcoal hemoperfusion [63-66]. However, these are generally isolated cases and it is difficult to determine efficacy, as well as whether coingestants were involved. In general, interventions to enhance drug elimination are not likely to be effective due to the large volume of distribution of TCAs, and plasma exchange and hemoperfusion are not recommended [67].

Medications — Flumazenil is contraindicated in patients with known or suspected TCA use. In patients who acutely coingest TCAs and benzodiazepines, flumazenil may lower the seizure threshold. In patients with an isolated TCA ingestion who use benzodiazepines chronically, flumazenil can induce benzodiazepine-withdrawal seizures.

Despite prominent anticholinergic toxicity in some patients with TCA poisoning, physostigmine is contraindicated as it is associated with cardiac arrest in the setting of TCA toxicity [38].

With the exception of lidocaine and magnesium (discussed above), concerns surround the use of all classes of antiarrhythmic drugs in the setting of TCA poisoning. Class IA (eg, procainamide) and Class IC agents (eg, flecainide) are contraindicated given their inhibition of rapid sodium channels, an effect similar to that induced by TCAs. Class III agents (eg, amiodarone) are poorly studied in this setting and the QTc prolongation associated with these drugs makes them potentially dangerous. Although a Class IB agent like lidocaine, phenytoin is generally not recommended in the setting of TCA poisoning.

Dr Kamal Deep Fortis Delhi 5:30:12 PM

Aortic dissection/ anti impulse therapy
Nitroprusside should not be used without first controlling heart rate with beta blockade since vasodilation alone induces reflex activation of the sympathetic nervous system, leading to enhanced ventricular contraction and increased aortic shear stress

DELETED 5:31:51 PM

True sympathetic reflex

Dr Kamal Deep Fortis Delhi 5:34:54 PM

Emailing Classification of aortic dissection - UpToDate.pdf

().pdf 212 KB 5:34:56 PM

Download ()

Tuesday, December 6, 2016

MS **Mandeep Singh** 3:44:52 PM

Hi

Dr Kamal Deep Fortis Delhi upgraded the group to a supergroup