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AS WE ARE QUALIFIED FOR EXAMS SO THAT WE CAN WRITE THE TEST,THESE DISEASES ARE ALSO QUALIFIED BASED ON THE CRITERIA. IF THE DISEASE HAS ONE OR MORE THE GIVEN SYMPTOMS OR PASSES ONE OR TWO TESTS HE IS CONSIDERED TO BE SUFFERING FROM THIS DISEASE.

criterias & classifications

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- 2.Dukes criteria: Endocarditis/Heart failure
- 3.Butchers criteria :mesothelioma
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- 5.Bismuth classification: tumors of hepatic ductal system
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- 50.Glasgow Blatchford score : UGI bleed for medical intervention
- 51.Waterlows classification: Malnutrition in children

Diagnostic assessments supporting the presence of heart failure Assessment Diagnosis of heart failure Supports if present Opposes if normal or absent Compatible symptoms ++ ++ Compatible signs ++ + Cardiac dysfunction on echocardiography +++ +++ Response of symptoms or signs to therapy +++ ++ ECG Normal ++ Abnormal ++ + Dysrhythmia +++ + Laboratory Elevated BNP/NT-proBNP +++ + Low/normal BNP/NT-proBNP + +++ Low blood sodium + + Kidney dysfunction + + Mild elevations of troponin + + Chest X-ray Pulmonary congestion +++ + Reduced exercise capacity +++ ++ Abnormal pulmonary function tests + + Abnormal hemodynamics at rest +++ ++ + = some importance; ++ = intermediate importance; +++ = great importance.

CHRONIC LIVER DISEASE MAINLY CIRRHOSIS The score employs five clinical measures of liver disease. Each measure is scored 1–3, with 3 indicating most severe derangement.

Measure 1 point 2 points 3 points Total bilirubin, µmol/L (mg/dL) <34 (<2) 34–50 (2–3) >50 (>3) Serum albumin, g/dL >3.5 2.8–3.5 <2.8 Prothrombin time, prolongation (s) <4.0 4.0–6.0 > 6.0 Ascites None Mild (or suppressed with medication) Moderate to severe (or refractory) Hepatic encephalopathy None Grade I–II Grade III–IV none

Different textbooks and publications use different measures. Some older reference works substitute prothrombin time (PT) prolongation for International normalized ratio (INR).

In primary sclerosing cholangitis (PSC) and primary biliary cholangitis (PBC), the bilirubin references are changed to reflect the fact that these diseases feature high conjugated bilirubin levels. The upper limit for 1 point is 68 µmol/L (4 mg/dL) and the upper limit for 2 points is 170 µmol/L (10 mg/dL)

GHENT NOSOLOGY FOR MARFAN SYNDROME In the absence of a family history of MFS:

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  Aortic root Z-score ≥ 2 AND ectopia lentis
  Aortic root Z-score ≥ 2 AND an FBN1 mutation
  Aortic root Z-score ≥ 2 AND a systemic score* > 7 points
  Ectopia lentis AND an FBN1 mutation with known aortic pathology
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In the presence of a family history of MFS (as defined above):

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  Ectopia lentis
  Systemic score* ≥ 7
  Aortic root Z-score ≥ 2
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  Points for systemic score:
      Wrist AND thumb sign = 3 (wrist OR thumb sign = 1)
      Pectus carinatum deformity = 2 (pectus excavatum or chest asymmetry =
1)
      Hindfoot deformity = 2 (plain pes planus = 1)
      Dural ectasia = 2
      Protrusio acetabuli = 2
      pneumothorax = 2
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      Reduced upper segment/lower segment ratio AND increased arm/height AND
no severe scoliosis = 1
      Scoliosis or thoracolumbar kyphosis = 1
      Reduced elbow extension = 1
      Facial features (3/5) = 1 (dolichocephaly, enophthalmos, downslanting
palpebral fissures, malar hypoplasia, retrognathia)
      Skin striae (stretch marks) = 1
      Myopia > 3 diopters = 1
      Mitral valve prolapse 1□4 1
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Modified Duke criteria

Established in 1994 by the Duke Endocarditis Service and revised in 2000, the Duke criteria are a collection of major and minor criteria used to establish a diagnosis of infective endocarditis. According to the Duke criteria, diagnosis of infective endocarditis can be definite, possible, or rejected.[19] A diagnosis of infective endocarditis is definite if either the following pathological or clinical criteria are met:

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  One of these pathological criteria:
      Histology or culture of a cardiac vegetation, an embolized vegetation,
or intracardiac abscess from the heart finds microorganisms
      Active endocarditis
  One of these combinations of clinical criteria
      2 major clinical criteria
      1 major and 3 minor criteria
      5 minor criteria
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Diagnosis of infective endocarditis is possible if one of the following combinations of clinical criteria are met:

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  1 major and 1 minor criteria
  3 minor criteria are fulfilled
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Major criteria

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  Positive blood culture with typical IE microorganism, defined as one of
the following:[19]
      Typical microorganism consistent with IE from 2 separate blood
cultures, as noted below:
          Viridans-group streptococci, or
          Streptococcus bovis including nutritional variant strains, or
          HACEK group, or
          Staphylococcus aureus, or
          Community-acquired Enterococci, in the absence of a primary focus
      Microorganisms consistent with IE from persistently positive blood
cultures defined as:
          Two positive cultures of blood samples drawn >12 hours apart, or
          All of 3 or a majority of 4 separate cultures of blood (with first
and last sample drawn 1 hour apart)
          Coxiella burnetii detected by at least one positive blood culture
or IgG antibody titer for Q fever phase 1 antigen >1:800. This was
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previously a minor criterion
  Evidence of endocardial involvement with positive echocardiogram defined
as
      Oscillating intracardiac mass on valve or supporting structures, in
the path of regurgitant jets, or on implanted material in the absence of an
alternative anatomic explanation, or
      Abscess, or
      New partial dehiscence of prosthetic valve or new valvular
regurgitation (worsening or changing of preexisting murmur not sufficient)
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Minor criteria

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  Predisposing factor: known cardiac lesion, recreational drug injection
  Fever >38 °C
  Embolism evidence: arterial emboli, pulmonary infarcts, Janeway lesions,
conjunctival hemorrhage
  Immunological problems: glomerulonephritis, Osler's nodes, Roth's spots,
Rheumatoid factor
  Microbiologic evidence: Positive blood culture (that doesn't meet a major
criterion) or serologic evidence of infection with organism consistent with
IE but not satisfying major criterion
  Positive echocardiogram (that doesn't meet a major criterion) (this
criterion has been removed from the modified Duke criteria)
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The Corpulence Index (CI) or Ponderal Index (PI) is a measure of leanness (corpulence) of a person[1] calculated as a relationship between mass and height. It is similar to the body mass index, but the mass is normalized with the third power of body height rather than the second power.

FOR A baby it is calculated as CI = birthweight/crown-heel-length^3 (power3)

it is useful is assesing intrauterine growth restriction

The body mass index (BMI) or Quetelet index is a value derived from the mass (weight) and height of an individual. The BMI is defined as the body mass divided by the square of the body height, and is universally expressed in units of kg/m2, resulting from mass in kilograms and height in metres.

In transplantation medicine, the Milan criteria are set of criteria applied in consideration of patients with cirrhosis and hepatocellular carcinoma (HCC) for liver transplantation with intent to cure their disease. Their significance derives from a landmark 1996 study in 48 patients by Mazzaferro et al which showed that selecting cases for transplantation according to specific strict criteria led to improved overall and disease-free survival at a 4-year time point.

The threshold Milan criteria are as follows:

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  one lesion smaller than 5 cm; alternatively, up to 3 lesions, each smaller
than 3 cm
  no extrahepatic manifestations
  no evidence of gross vascular invasion
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Subglottic stenosis is graded according to the Cotton-Meyer classification system from one to four based on the severity of the blockage.

Grade 1 – <50% obstruction Grade 2 – 51–70% obstruction Grade 3 – 71–99% obstruction Grade 4 – no detectable lumen

ABDOMINAL PREGNANCY Criteria

To diagnose the rare primary abdominal pregnancy, Studdiford's criteria need to be fulfilled: tubes and ovaries should be normal, there is no abnormal connection (fistula) between the uterus and the abdominal cavity, and the pregnancy is related solely to the peritoneal surface without signs that there was a tubal pregnancy first.Studdiford's criteria were refined in 1968 by Friedrich and Rankin to include microscopic findings

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HODGKIN'S LYMPHOMA
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On the basis of this staging, the patient will be classified according to a staging classification (the Ann Arbor staging classification scheme is a common one):

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  Stage I is involvement of a single lymph node region (I) (mostly the
cervical region) or single extralymphatic site (Ie);
  Stage II is involvement of two or more lymph node regions on the same side
of the diaphragm (II) or of one lymph node region and a contiguous
extralymphatic site (IIe);
  Stage III is involvement of lymph node regions on both sides of the
diaphragm, which may include the spleen (IIIs) or limited contiguous
extralymphatic organ or site (IIIe, IIIes);
  Stage IV is disseminated involvement of one or more extralymphatic organs.
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The absence of systemic symptoms is signified by adding A to the stage; the presence of systemic symptoms is signified by adding B to the stage. For localised extranodal extension from mass of nodes that does not advance the stage, subscript E is added. Splenic involvement is signified by adding S to the stage. The inclusion of bulky disease is signified by X.

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MALNUTRITION FOR CHILDREN
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Definition by Gomez In 1956, Gómez and Galvan studied factors associated with death in a group of malnourished (undernourished) children in a hospital in Mexico City, Mexico and defined categories of malnutrition: first, second, and third degree.[34] The degrees were based on weight below a specified percentage of median weight for age.[35] The risk of death increases with increasing degree of malnutrition.[34] An adaptation of Gomez's original classification is still used today. While it provides a way to compare malnutrition within and between populations, the classification has been criticized for being arbitrary and for not considering overweight as a form of malnutrition. Also, height alone may not be the best indicator of malnutrition; children who are born prematurely may be considered short for their age even if they have good nutrition.[36]

Degree of PEM % of desired body weight for age and sex Normal 90%-100% Mild: Grade I (1st degree) 75%-89% Moderate: Grade II (2nd degree) 60%-74% Severe: Grade III (3rd degree) <60% SOURCE:Serum Total Protein and Albumin Levels in Different Grades of Protein Energy Malnutrition Definition by Waterlow John Conrad Waterlow established a new classification for malnutrition. Instead of using just weight for age measurements, the classification established by Waterlow combines weight-for-height (indicating acute episodes of malnutrition) with height-for-age to show the stunting that results from chronic malnutrition. One advantage of the Waterlow classification over the Gomez classification is that weight for height can be examined even if ages are not known.

Degree of PEM Stunting (%) Height for age Wasting (%) Weight for height Normal: Grade 0 >95% >90% Mild: Grade I 87.5-95% 80-90% Moderate: Grade II 80-87.5% 70-80% Severe: Grade III <80% <70% SOURCE: Classification and definition of protein-calorie malnutrition. by Waterlow, 1972 These classifications of malnutrition are commonly used with some modifications by WHO

UPPER GI BLEEDING Rockall risk scoring system attempts to identify patients at risk of adverse outcome following acute upper gastrointestinal bleeding. Rockall et al. identified independent risk factors[1] in 1996 which were later shown to predict mortality accurately. The scoring system uses clinical criteria (increasing age, co-morbidity, shock) as well as endoscopic finding (diagnosis, stigmata of acute bleeding). It is named for Professor Tim Rockall, who was the main investigator and first author of the studies that led to its formulation. A convenient mnemonic is ABCDE - i.e. Age, Blood pressure fall (shock), Co-morbidity, Diagnosis and Evidence of bleeding.

Variable[2] Score 0 Score 1 Score 2 Score 3 Age <60 60- 79 >80 Shock No shock Pulse >100 BP >100 Systolic SBP <100 Co-morbidity Nil major CHF, IHD, major morbidity Renal failure, liver failure, metastatic cancer Diagnosis Mallory-Weiss All other diagnoses GI malignancy Evidence of bleeding None Blood, adherent clot, spurting vessel

The score is calculated using the table below:

Glasgow-Blatchford Score Admission risk marker Score component value Blood Urea (mmol/L)[5] 6.5-8.0 2 8.0-10.0 3 10.0-25 4

> 25 6

Haemoglobin (g/L) for men 12.0-12.9 1 10.0-11.9 3 <10.0 6 Haemoglobin (g/L) for women 10.0-11.9 1 <10.0 6 Systolic blood pressure (mm Hg) 100–109 1 90–99 2 <90 3 Other markers Pulse ≥100 (per min) 1 Presentation with melaena 1 Presentation with syncope 2 Hepatic disease 2 Cardiac failure 2 In the validation group, scores of 6 or more were associated with a greater than 50% risk of needing an intervention.

Score Score is equal to 0 if the following are all present:

Hemoglobin level >12.9 g/dL (men) or >11.9 g/dL (women) Systolic blood pressure >109 mm Hg Pulse <100/minute Blood urea nitrogen level <6.5 mg/dL No melena or syncope No past or present liver disease or heart failure

DeBakey The DeBakey system, named after cardiothoracic surgeon Michael E. DeBakey, is an anatomical description of the aortic dissection. It categorizes the dissection based on where the original intimal tear is located and the extent of the dissection (localized to either the ascending aorta or descending aorta or involves both the ascending and descending aorta.[16]

Type I – originates in ascending aorta, and propagates at least to the aortic arch and often beyond it distally. It is most often seen in patients less than 65 years of age and is the most lethal form of the disease. Type II – originates in the ascending aorta and is confined to it. Type III – originates in the descending aorta and rarely extends proximally, but will extend distally. It most often occurs in elderly patients with atherosclerosis and hypertension. Stanford The Stanford classification is divided into two groups, A and B, depending on whether the ascending aorta is involved.[17]

A – involves the ascending aorta and/or aortic arch, and possibly the descending aorta. The tear can originate in the ascending aorta, the aortic arch, or more rarely, in the descending aorta. It includes DeBakey types I and II. B – involves the descending aorta or the arch (distal to the left subclavian

artery), without the involvement of the ascending aorta. It includes DeBakey type III. The Stanford classification is useful as it follows clinical practice, as type A ascending aortic dissections generally require primary surgical treatment, whereas type B dissections generally are treated medically as initial treatment with surgery reserved for any complications.

The reason for surgical repair of type A dissections is that ascending aortic dissections often involve the aortic valve, which, having lost its suspensory support, telescopes down into the aortic root, resulting in aortic incompetence. The valve must be resuspended in order to be reseated, as well as to repair or prevent coronary artery injury. Also, the area of dissection is removed and replaced with a Dacron graft to prevent further dissection from occurring. However, type B dissections are not improved, from a mortality point of view, by the operation, unless leaking, rupture, or compromise to other organs, e.g. kidneys, occurs.

The Oxford Community Stroke Project classification (OCSP, also known as the Bamford or Oxford classification) relies primarily on the initial symptoms; based on the extent of the symptoms, the stroke episode is classified as total anterior circulation infarct (TACI), partial anterior circulation infarct (PACI), lacunar infarct (LACI) or posterior circulation infarct (POCI). These four entities predict the extent of the stroke, the area of the brain that is affected, the underlying cause, and the prognosis.[21][22] The TOAST (Trial of Org 10172 in Acute Stroke Treatment) classification is based on clinical symptoms as well as results of further investigations; on this basis, a stroke is classified as being due to (1) thrombosis or embolism due to atherosclerosis of a large artery, (2) an embolism originating in the heart, (3) complete blockage of a small blood vessel, (4) other determined cause, (5) undetermined cause (two possible causes, no cause identified, or incomplete investigation).[23] Users of stimulants, such as cocaine and methamphetamine are at a high risk for ischemic strokes.

The Wallace rule of nines is a tool used in pre-hospital and emergency medicine to estimate the total body surface area (BSA) affected by a burn. In addition to determining burn severity, the measurement of burn surface area is important for estimating patients' fluid requirements and determining hospital admission criteria.

Body Part Estimated BSA Entire left arm 9% Entire right arm 9% Entire head 9% Entire chest 9% Entire abdomen 9% Entire back 18% Entire left leg 18% Entire right leg 18% Groin 1%

For example, if a patient's entire back (18%) and entire left leg (18%) are burned, about 36% of the patient's BSA is affected. The BSAs assigned to each body part refer to the entire body part.[3] So, for example, if half of a patient's left leg were burned, it would be assigned a BSA value of 9% (half the total surface area of the leg). Thus, if a patient's entire back (18%), but only half of their left leg (9%) was burned, the amount of BSA affected would be 27%.

Probability

Swelling in the leg from fluid (edema) can result in pitting after pressure is applied. (This person did not have DVT.) In those with suspected DVT, a clinical assessment of probability can be useful to determine which tests to perform.[51][52] The most studied clinical prediction rule is the Wells score.[3][53]

Wells score or criteria: (possible score −2 to 9)

Active cancer (treatment within last 6 months or palliative): +1 point Calf swelling ≥ 3 cm compared to asymptomatic calf (measured 10 cm below tibial tuberosity): +1 point Swollen unilateral superficial veins (non-varicose, in symptomatic leg): +1 point Unilateral pitting edema (in symptomatic leg): +1 point Previous documented DVT: +1 point Swelling of entire leg: +1 point Localized tenderness along

the deep venous system: +1 point Paralysis, paresis, or recent cast immobilization of lower extremities: +1 point Recently bedridden ≥ 3 days, or major surgery requiring regional or general anesthetic in the past 12 weeks: +1 point Alternative diagnosis at least as likely: −2 points[4] Those with Wells scores of two or more have a 28% chance of having DVT, those with a lower score have 6% odds. Alternatively, Wells scores can be categorized as high if greater than two, moderate if one or two, and low if less than one, with likelihoods of 53%, 17%, and 5%, respectively.

Probability testing The most commonly used method to predict clinical probability, the Wells score, is a clinical prediction rule, whose use is complicated by multiple versions being available. In 1995, Philip Steven Wells, initially developed a prediction rule (based on a literature search) to predict the likelihood of PE, based on clinical criteria.[26] The prediction rule was revised in 1998[27] This prediction rule was further revised when simplified during a validation by Wells et al. in 2000.[28] In the 2000 publication, Wells proposed two different scoring systems using cutoffs of 2 or 4 with the same prediction rule.[28] In 2001, Wells published results using the more conservative cutoff of 2 to create three categories.[29] An additional version, the modified extended version, using the more recent cutoff of 2 but including findings from Wells's initial studies[26][27] were proposed.[30] Most recently, a further study reverted to Wells's earlier use of a cutoff of 4 points[28] to create only two categories.[31]

There are additional prediction rules for PE, such as the Geneva rule. More importantly, the use of any rule is associated with reduction in recurrent thromboembolism.[32]

The Wells score:[33]

clinically suspected DVT — 3.0 points alternative diagnosis is less likely than PE — 3.0 points tachycardia (heart rate > 100) — 1.5 points immobilization (≥ 3d)/surgery in previous four weeks — 1.5 points history of DVT or PE — 1.5 points hemoptysis — 1.0 points malignancy (with treatment within six months) or palliative — 1.0 points Traditional interpretation[28][29][34]

Score >6.0 — High (probability 59% based on pooled data)[35] Score 2.0 to 6.0 — Moderate (probability 29% based on pooled data)[35] Score <2.0 — Low (probability 15% based on pooled data)[35] Alternative interpretation[28][31]

Score > 4 — PE likely. Consider diagnostic imaging. Score 4 or less — PE unlikely. Consider D-dimer to rule out PE. Recommendations for a diagnostic algorithm were published by the PIOPED investigators; however, these recommendations do not reflect research using 64 slice MDCT.[35] These investigators recommended:

Low clinical probability. If negative D-dimer, PE is excluded. If positive D-dimer, obtain MDCT and based treatment on results. Moderate clinical probability. If negative D-dimer, PE is excluded. However, the authors were not concerned that a negative MDCT with negative D-dimer in this setting has a 5% probability of being false. Presumably, the 5% error rate will fall as 64 slice MDCT is more commonly used. If positive D-dimer, obtain MDCT and based treatment on results. High clinical probability. Proceed to MDCT. If positive, treat, if negative, more tests are needed to exclude PE. A D-dimer of less than 750 ug/L does not rule out PE in those who are at high risk.[36] Pulmonary embolism rule-out criteria The pulmonary embolism rule-out criteria (PERC) helps assess people in whom pulmonary embolism is suspected, but unlikely. Unlike the Wells score and Geneva score, which are clinical prediction rules intended to risk stratify people with suspected PE, the PERC rule is designed to rule out risk of PE in people when the physician has already stratified them into a low-risk category.

People in this low risk category without any of these criteria may undergo no further diagnostic

testing for PE: Hypoxia — SaO2 <95%, unilateral leg swelling, hemoptysis, prior DVT or PE, recent surgery or trauma, age >50, hormone use, tachycardia. The rationale behind this decision is that further testing (specifically CT angiogram of the chest) may cause more harm (from radiation exposure and contrast dye) than the risk of PE.[37] The PERC rule has a sensitivity of 97.4% and specificity of 21.9% with a false negative rate of 1.0% (16/1666).

POLYCYSTIC OVARIAN DISEASE Definition Two definitions are commonly used:

NIH In 1990 a consensus workshop sponsored by the NIH/NICHD suggested that a person has PCOS if they have all of the following:[54] oligoovulation signs of androgen excess (clinical or biochemical) exclusion of other disorders that can result in menstrual irregularity and hyperandrogenism Rotterdam In 2003 a consensus workshop sponsored by ESHRE/ASRM in Rotterdam indicated PCOS to be present if any 2 out of 3 criteria are met, in the absence of other entities that might cause these findings[17][55][56] oligoovulation and/or anovulation excess androgen activity polycystic ovaries (by gynecologic ultrasound) The Rotterdam definition is wider, including many more women, the most notable ones being women without androgen excess. Critics say that findings obtained from the study of women with androgen excess cannot necessarily be extrapolated to women without androgen excess.[57][58]

Androgen Excess PCOS Society In 2006, the Androgen Excess PCOS Society suggested a tightening of the diagnostic criteria to all of the following:[17] excess androgen activity oligoovulation/anovulation and/or polycystic ovaries exclusion of other entities that would cause excess androgen activity

The Hunt and Hess scale, introduced in 1968, is one of the grading systems used to classify the severity of a subarachnoid hemorrhage based on the patient's clinical condition. It is used as a predictor of patient's prognosis/outcome, with a higher grade correlating to lower survival rate. Other scales which describe the clinical presentation of subarachnoid hemorrhage patients include the World Federation of Neurosurgical Societies classification, which combines consciousness and motor deficit in its scoring.

Description grade Asymptomatic, mild headache, slight nuchal rigidity Moderate to severe headache, nuchal rigidity, no neurologic deficit other than cranial nerve palsy Drowsiness, confusion, mild focal neurologic deficit Stupor, moderate-severe hemiparesis Coma, decerebrate posturing It gives an index of the mortality associated with the various grades. The mortality is minimum with grade 1 and maximum with grade 5.

In anesthesia, the Mallampati score or Mallampati classification, named after the Indian-born American anaesthesiologist Seshagiri Mallampati, is used to predict the ease of endotracheal intubation.[1] The test comprises a visual assessment of the distance from the tongue base to the roof of the mouth, and therefore the amount of space in which there is to work. It is an indirect way of assessing how difficult an intubation will be; this is more definitively scored using the Cormack-Lehane classification system, which describes what is actually seen using direct laryngoscopy during the intubation process itself. A high Mallampati score (class 3 or 4) is associated with more difficult intubation as well as a higher incidence of sleep apnea.

The score is assessed by asking the patient, in a sitting posture, to open his or her mouth and to protrude the tongue as much as possible.[1] The anatomy of the oral cavity is visualized; specifically, the assessor notes whether the base of the uvula, faucial pillars (the arches in front of and behind the tonsils) and soft palate are visible. Scoring may be done with or without phonation. Depending on whether the tongue is maximally protruded and/or the patient asked to phonate, the scoring may vary.

Modified Mallampati Scoring:[3]

Class I: Soft palate, uvula, fauces, pillars visible. Class II: Soft palate, uvula, fauces visible. Class III: Soft palate, base of uvula visible. Class IV: Only hard palate visible. Original Mallampati Scoring:[1]

Class 1: Faucial pillars, soft palate and uvula could be visualized. Class 2: Faucial pillars and soft palate could be visualized, but uvula was masked by the base of the tongue. Class 3: Only soft palate visualized. Further research may be needed to determine the most effective consistent and predictive approach on which to standardize Mallampati Scoring.

Seddon's classification In 1943, Seddon described three basic types of peripheral nerve injury [2] that include:

Neurapraxia (Class I) Main article: Neurapraxia It is a temporary interruption of conduction without loss of axonal continuity.[3]In neurapraxia, there is a physiologic block of nerve conduction in the affected axons.

Other characteristics:

It is the mildest type of peripheral nerve injury. There are sensory-motor problems distal to the site of injury. The endoneurium, perineurium, and the epineurium are intact. There is no wallerian degeneration. Conduction is intact in the distal segment and proximal segment, but no conduction occurs across the area of injury.[4] Recovery of nerve conduction deficit is full, and requires days to weeks. EMG shows lack of fibrillation potentials (FP) and positive sharp waves. Axonotmesis (Class II) Main article: Axonotmesis It involves loss of the relative continuity of the axon and its covering of myelin, but preservation of the connective tissue framework of the nerve (the encapsulating tissue, the epineurium and perineurium, are preserved).[5]

Other characteristics:

Wallerian degeneration occurs distal to the site of injury. There are sensory and motor deficits distal to the site of lesion. There is no nerve conduction distal to the site of injury (3 to 4 days after injury). EMG shows fibrillation potentials (FP), and positive sharp waves (2 to 3 weeks postinjury). Axonal regeneration occurs and recovery is possible without surgical treatment.Sometimes surgical intervention because of scar tissue formation is required. Neurotmesis (Class III) Main article: Neurotmesis It is a total severance or disruption of the entire nerve fiber.[6]A peripheral nerve fiber contains an axon (Or long dendrite), myelin sheath (if existence), their schwann cells, and the endoneurium. Neurotmesis may be partial or complete.

Other characteristics:

Wallerian degeneration occurs distal to the site of injury. There is connective tissue lesion that may be partial or complete. Sensory-motor problems and autonomic function defect are severe. There is no nerve conduction distal to the site of injury (3 to 4 days after lesion). EMG and NCV findings are as axonotmesis. Because of lack of nerve repair, surgical intervention is necessary. Sunderland's classification In 1951, Sunderland expanded Seddon's classification to five degrees of peripheral nerve injury:

First-degree (Class I) Seddon's neurapraxia and first-degree are the same.

Second-degree (Class II) Seddon's axonotmesis and second-degree are the same.

Third-degree (Class III) Third-degree is included within Seddon's Neurotmesis.

Sunderland's third-degree is a nerve fiber interruption. In third-degree injury, there is a lesion of the endoneurium, but the epineurium and perineurium remain intact. Recovery from a third-degree injury is possible, but surgical intervention may be required.

Fourth-degree (Class III) Fourth-degree is included within Seddon's Neurotmesis.

In fourth-degree injury, only the epineurium remain intact. In this case, surgical repair is required.

Fifth-degree (Class III) Fifth-degree is included within Seddon's Neurotmesis.

Fifth-degree lesion is a complete transection of the nerve. Recovery is not possible without an appropriate surgical treatment.

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GASTRIC CARCINOMA OR STOMACH CANCER
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Staging may not be complete until after surgery. The surgeon removes nearby lymph nodes and possibly samples of tissue from other areas in the abdomen for examination by a pathologist.

The clinical stages of stomach cancer are:[57][58]

Stage 0. Limited to the inner lining of the stomach. Treatable by endoscopic mucosal resection when found very early (in routine screenings); otherwise by gastrectomy and lymphadenectomy without need for chemotherapy or radiation. Stage I. Penetration to the second or third layers of the stomach (Stage 1A) or to the second layer and nearby lymph nodes (Stage 1B). Stage 1A is treated by surgery, including removal of the omentum. Stage 1B may be treated with chemotherapy (5-fluorouracil) and radiation therapy. Stage II. Penetration to the second layer and more distant lymph nodes, or the third layer and only nearby lymph nodes, or all four layers but not the lymph nodes. Treated as for Stage I, sometimes with additional neoadjuvant chemotherapy. Stage III. Penetration to the third layer and more distant lymph nodes, or penetration to the fourth layer and either nearby tissues or nearby or more distant lymph nodes. Treated as for Stage II; a cure is still possible in some cases. Stage IV. Cancer has spread to nearby tissues and more distant lymph nodes, or has metastasized to other organs. A cure is very rarely possible at this stage. Some other techniques to prolong life or improve symptoms are used, including laser treatment, surgery, and/or stents to keep the digestive tract open, and chemotherapy by drugs such as 5-fluorouracil, cisplatin, epirubicin, etoposide, docetaxel, oxaliplatin, capecitabine or irinotecan.[12]

Stomach cancer metastasized to the lungs The TNM staging system is also used

PANCREATITIS

Diagnosis requires 2 of the 3 following criteria:

Characteristic acute onset of epigastric or vague abdominal pain that may radiate to the back (see signs and symptoms above) Serum amylase or lipase levels ≥ 3 times the upper limit of normal An imaging study with characteristic changes. CT, MRI, abdominal ultrasound or endoscopic ultrasound can be used for diagnosis. Amylase and lipase are 2 enzymes produced by the pancreas. Elevations in lipase are generally considered a better indicator for pancreatitis as it has greater specificity and has a longer half life.

The Modified Glasgow criteria suggests that a case be considered severe if at least three of the following are true:[32] Age > 55 years Blood levels: PO2 oxygen < 60mmHg or 7.9kPa White blood cells > 15 Calcium < 2 mmol/L Urea > 16 mmol/L Lactate dehydrogenase (LDH) > 600iu/L Aspartate transaminase (AST) > 200iu/L Albumin < 32g/L Glucose > 10 mmol/L This can be remembered using

the mnemonic PANCREAS:

PO2 oxygen < 60mmHg or 7.9kPa Age > 55 Neutrophilia white blood cells > 15 Calcium < 2 mmol/L Renal urea > 16 mmol/L Enzymes lactate dehydrogenase (LDH) > 600iu/L aspartate transaminase (AST) > 200iu/L Albumin < 32g/L Sugar glucose > 10 mmol/L The BISAP score (blood urea nitrogen level >25 mg/dL, impaired mental status, systemic inflammatory response syndrome, age over 60 years, pleural effusion) has been validated as similar to other prognostic scoring systems.

Revised Diagnostic Criteria (2010) - MCDONALD'S CRITERIA FOR MULTIPLE SCLEROSIS DIAGNOSIS Clinical Presentation Additional Data Needed * 2 or more attacks (relapses) * 2 or more objective clinical lesions None; clinical evidence will suffice (additional evidence desirable but must be consistent with MS) * 2 or more attacks * 1 objective clinical lesion Dissemination in space, demonstrated by: * MRI * or further clinical attack involving different site. New criteria: Dissemination in Space (DIS) can be demonstrated by the presence of 1 or more T2 lesions in at least 2 of 4 of the following areas of the CNS: Periventricular, Juxtacortical, Infratentorial, or Spinal Cord. * 1 attack * 2 or more objective clinical lesions Dissemination in time (DIT), demonstrated by: * MRI * or second clinical attack New criteria: No longer a need to have separate MRIs run; Dissemination in time, demonstrated by: Simultaneous presence of asymptomatic gadolinium-enhancing and nonenhancing lesions at any time; or A new T2 and/or gadolinium-enhancing lesion(s) on follow-up MRI, irrespective of its timing with reference to a baseline scan; or Await a second clinical attack. [This allows for quicker diagnosis without sacrificing specificity, while improving sensitivity.]

* 1 attack * 1 objective clinical lesion (clinically isolated syndrome) New criteria: Dissemination in space and time, demonstrated by: For DIS: 1 or more T2 lesion in at least 2 of 4 MS-typical regions of the CNS (periventricular, juxtacortical, infratentorial, or spinal cord); or Await a second clinical attack implicating a different CNS site; and For DIT: Simultaneous presence of asymptomatic gadolinium-enhancing and nonenhancing lesions at any time; or A new T2 and/or gadolinium-enhancing lesion(s) on follow-up MRI, irrespective of its timing with reference to a baseline scan; or Await a second clinical attack.

Insidious neurological progression suggestive of MS (primary progressive MS) New criteria: One year of disease progression (retrospectively or prospectively determined) and two or three of the following: 1. Evidence for DIS in the brain based on 1 or more T2 lesions in the MS-characteristic (periventricular, juxtacortical, or infratentorial) regions 2. Evidence for DIS in the spinal cord based on 2 or more T2 lesions in the cord 3. Positive CSF (isoelectric focusing evidence of oligoclonal bands and/or elevated IgG index)