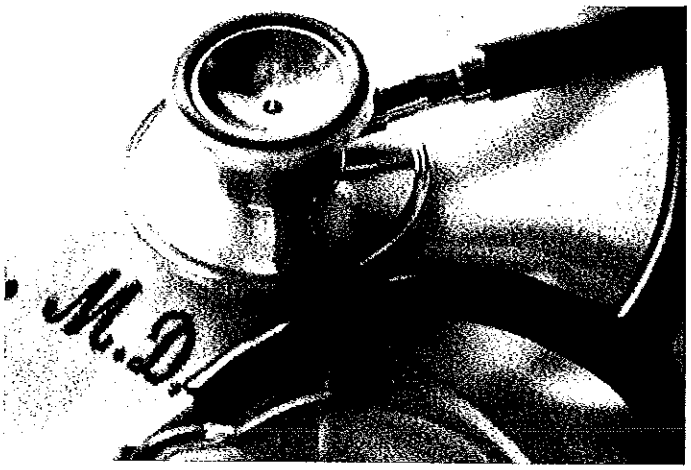
# INTERNAL MEDICINE

# 2009-2010




DEVANGNA BHATIA

# Examination questions

for Clinical examination in internal medicine

## Practice

Clinical examination of patient

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## Theory - part I

General history
Special history according to heart diseases
Special history according to lung diseases
Special history according to renal diseases
Special history according to gastrointestinal a hepatic diseases
Special history according to endocrine diseases
Special history according to haematological diseases
Special history according to peripheral arteries and veins diseases
Special history according to rheumatic diseases
Special history according to immunological diseases

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## Theory part II

Laboratory investigation and clinical examination in heart diseases
Laboratory investigation and clinical examination in lung diseases
Laboratory investigation and clinical examination in renal diseases
Laboratory investigation and clinical examination in gastrointestinal diseases
Laboratory investigation and clinical examination in hepatic diseases
Laboratory investigation and clinical examination in billiary and pancreatic diseases
Laboratory investigation and clinical examination in endocrine diseases
Laboratory investigation and clinical examination in haematological diseases
Laboratory investigation and clinical examination in peripheral arteries and veins diseases
Laboratory investigation and clinical examination in rheumatic diseases
Laboratory investigation and clinical examination in immunology diseases

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## Theory part III

Normal ECG
ECG in the acute myocardial infarction
ECG in myocarditis, metabolic disorders ( $K^+$, $Ca^{2+}$)
ECG in bradyarrhythmias
ECG in tachyarrhythmias
ECG in conductive disturbances (LBBB, RBBB, LAH, LPH)
ECG in left and right ventricle hypertrophy
Normal chest X-ray
Normal blood count and haemocoagulation parameters
Urine investigation - normal and pathology findings

QRS wide = ventricular extrasystole or blockage

The exam will be consist from practice and three questions from theory - one from the part I, second from the part II and the thirt from the part III

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# Renal diseases

general features: fatigue, pallor + breathlessness (CRF) ⇒ lemon-yellow complexion
↳ brownish discolouration of the distal nail
↳ ↑ in BP (in kidney disease); maybe ↓ in ∅ tubulointerstitial disease
↳ eyes ⇒ conjunctival pallor - anaemia of CRF

count GFR/creatinine clearance
PABA ⇒ total kidney blood flow
GFR adjus ⇒ inulin

Inspection: ab. fr distention ⇒ very large kidneys (polycystic PKD)
Suprapubic swelling ⇒ gross bladder distention
Scars - renal tract surgery in the loins / iliac fossae (transplant surgery)

Palpation: Lower R. Quad ⇒ start here - using fingers
- 1 hand behind the kidney (below lower ribs); other hand over the upper quadrant ant., lat to rectus muscle.
- push hands together while patient breathes out then breathe in
'easier to feel the Right one'

BALLOTTING ⇒ - try to move it abt between ur hands
- if palpable ⇒ assess size, surface + consistency
- Tenderness of the kidney ⇒ post. in the Renal angle - 12th rib + spine
↳ palpate firmly w/ fingers ~~of~~ or firmly strike the angle w/ ur fist. (ulnar side)
↳ PN or Acute urinary obs.

Percussion: distended bladder = dull - start percussing over a resonant area in the upper ab in the midline ⇒ ~~suprapubic~~ symphysis pubis

Auscultation: to detect bruits - arising (poss) fm the Renal A. ⇒ Renal A. stenosis ⇒ abnormal murmur + 2° ↑BP

Rectal exam: benign prostate enlargement / malignant change
females: vag exam ⇒ malig. disease involving ureters/bladder

① Urinary tract US, ② Doppler US of renal A/V, ③ IV urography, ④ renal angiography, ⑤ MRI angiography, ⑥ Renal isotope scanning, ⑦ ab. CT scan, ⑧ biopsy, ⑨ pyelography
scintigraphy

↳ creatinine, uric acid, blood ions + their equilib (↑K⁺ often)
urine exam.

# Gastrointestinal disease

**general**: height, weight, waist circumference, BMI
obesity – truncal / generalised
pallor – anaemia
striae – rapid weight gain / prev preg
atrophic glossitis – pale, smooth tongue
jaundice – yellow sclera
spider naevi } chronic liver disease
palmar erythema

**Inspection**: skin ⇒ colour, spots
hair, visible veins
distention / swelling – fat, ascites, ileus, constipation, pregnancy
scars + stomas (removal of smthing)

**Palpation**: light / superf. first
start away fm source of pain + go thru all the legions
deep palpation
rigid ab wall ⇒ diffuse peritonitis (doesn't move w/ resp).
(Blumberg's sign) "Rebound tenderness" ⇒ gently press, rapidly remove ⇒ pain (Peritonitis)
(Rovsing's sign) palp. of L. left quad + pain in Right Lower quad ⇒ Appendicitis
ascites

**Percussion**: ascites

**Auscultation**: bowel sounds = gurgling sounds – norm. peristaltic activity / every 5-10sec

**Rectal exam**: maybe exam. genitalia

"Acute abdomen" ⇒ sudden severe ab pain ≤24hrs in duration
↳ Murphy's sign: acute cholecystitis
↳ patient takes a deep breath – gently palpate R.U. Quad.
↳ diaphragm descends ⇒ gall bladder comes in contact w/ fingers
↳ patient: "catch the inspiratory effort".

Stool: pale - biliary obs
pale + greasy - steatorrhoea w/ malabsorption
Dark - bleed fm upper GIT
Grey/black - oral iron therapy
Mixed w/ pus - ulcerative colitis, dysentery

Urinalysis - jaundice
Ascitic fluid - biochem, microbio + cytological analysis
↳ Paracentesis! 1/2 way between ASIS + umbilicus
↳ usually clear + straw-coloured
↳ blood-stained ⇒ intra-ab malig
↳ Turbid ⇒ ↑ cell count ⇒ infec or high prot content
↳ Milky ⇒ Chylous - high lipid content - impaired lymphatic drainage

Ab. xray, barium meal, upper ab. US, pelvic US, & Upper/lower GI endoscopy., ERCP (endoscopic retrograde cholangiopancreatography), laproscopy, liver biopsy, pancreatic func. tests, CT, HRI, colonoscopy, capsule endoscopy

- testing of occult bleeding
- Microbio test

CRP!

Liver
AST
ALT
GGT
Alkaline phosphatase

Faecal occult blood test
‼
used to test for haeme, now tests for globin (more sensitive)

# Hepatic diseases:

General: same as before ⇒ jaundice!

Inspection: same as before

Palpation: - start in the Right ~~th~~ Iliac fossa
- patient breathes in
- try to feel the enlarged liver as it moves downwards on inspiration
- keep repeating + moving hand up until it reach the costal margin
- figure out if it is enlarged or not (6-12cm)
- find the liver borders
- scratch the liver - use stethoscope

~~Percuss~~

Percussion: dull to percuss.
lower 3-4 ribs = dull

Causes of hepatomegaly: alcoholic Liver disease
autoimmune hepatitis
viral "
Haematological disorders: 1° biliary cirrhosis
lymphoma RHF
leukaemia Malig: 1° hepatocellular cancer
Myelofibrosis 2° metastatic cancer
Polycythaemia amyloidosis

Upper ab ① US, ② ERCP (endoscopic Retrograde Cholangiopancreatography) ③ Laproscopy, ④ Aspiration cytology, ⑤ Liver biopsy, ⑥ liver enzymes tests, ⑦ CT, ⑧ MRI

AST, ALT, GGT, Alkaline phosphate
CRP - any inflam.

# Biliary + pancreatic diseases

Gallbladder:

Palpation - size + texture

cholecystitis ⇒ sharp pain

enlarged g.bladder = palpable - pear shaped

Murphy's sign ⇒ acute cholecystitis

↳ patient takes a deep breath, gently palpate

otherbook ⇒ * use the thumb; patient cnt take a deep breath because of pain (inflamed g.bladder against the palpating finger) *

Courvoisier's sign: palpable but painless gallbladder ⇒ blockage of the extrahepatic biliary ducts.

Biliary Colic ⇒ sharp, spastic pain - intensity waxing + waning within a few mins

↳ Right hypochondrium, radiates lat or to the back, below the ~~R. scc.~~ Right scapula

- pancreatic involvement ⇒ radiate to the left

Indigestion - common symp. of gallbladder diseases

Pancreatic amylase + lipase

# Endocrine diseases

assess hormones of affected organs:
ACTH ⇒ adrenal gland ; FSH + LH in gonads
TSH ⇒ thyroid
Antibodies ⇒ autoimmune

US, CT ⇒ adrenal, MRI

1° - if gland affected
2° - if hypophysis

Palpation ⇒ thyroid

Inspection ⇒ thyroid, gonads in males

# Haemtological diseases

when there is a suspicion of DVT, pul emb. (PE) or DIC

D-dimers ⇒ ↑ in thrombosis/embolism/DIC test

- fibrin degradation product, present in the blood after a blood clot is degraded by fibrinolysis

DWCC:
- Leukocytes
- RBCs
- Haematocrit
- Thrombocytes

Urine:
- glc
- pH
- Bilirubin
- colour
- nitrites
- appearance
- specific weight

icterus poss. as well

Coagulation:
- Quick test (measure time taken for fibrin strand formation)
- International Normalised ratio (INR = 0.9 - 1.2) ↑ pro-thrombin time
- Fibrinogen

anaemia = pale mouth

VS of spleen, liver, LN (hepato + splenomegaly)
if u think lymphoma ⇒ biopsy of LN if enlarged

palpate: LN, hepato/splenomegaly
Sonography, CT (LN)

Hb values:
- Males: 8.5 - 11.3 mmol/l
- Females: 7.5 - 9.3 mmol/l
- Children: 6.8 - 9.9 mmol/l

Haemocoagulation factors ⇒
- fibrinogen
- blood lipids

## Periph Arteries + Veins disease

Arteries: Common symp: ① Limb symp
② Neurological symp
③ Abdominal "
④ Vasospatic "

Limb symp. ischaemia has 4 stages: ① Asymp. dnt walk far enuf or some other pathology limits them

② Intermittent claudication: pain in legs during walking due to arterial insuff.
↳ usually in the calf + most common symptom
↳ neurogenic claudication: leg pain on walking due to neurological + musculoskeletal disorders of the lumbar spine
↳ venous claudication: pain due to venous outflow obs. in the leg, following extensive DVT.

③ Night / rest pain: falls asleep, wakes up 1-2hrs later due to severe pain - instep y the foot
↳ severe multi-level arterial disease

④ Tissue loss (ulceration and/or gangrene): in patients w/ rest pain due to periph arterial disease w/ critical limb ischaemia
↳ bac. enter → gangrene/ulceration

Examination sequences: head downwards; inspec, palpation + auscultation
① Arms: radial, brachial + carotid pulses
BP in both arms (diff of upto 10mmHg in systolic ⇒ norm)
② Abdomen: obvious pulsation
palpate + listen over the ab aorta
if easily palpable ⇒ ab. aortic aneurysm
③ Legs: legs + feet f/ ischaemia, temp + colour changes
scars - vascular + non-vascular surgery
position, margin, depth + colour of any ulceration between the toes + heels ⇒ ischaemic changes

Doppler US
angiography / CT angiography

④ Femoral pulse: supine patient; use 2-3 fingers
↳ simultaneously check for the radiofemoral delay

⑤ Popliteal pulse: supine, knees flexed to 30°; thumb in front + fingers post - press!
⑥ ↳ if really easy to feel ⇒ poss = aneurysm (US needed)

⑥ Post. tibial pulse: 2cm below + 2cm behind the medial malleolus - index + middle fingers

⑦ Dorsalis pedis pulse: middle of the dorsum of the foot, just lat to the tendon of extensor hallucis longus

Auscultation: lumen >60% narrowed ⇒ turbulent b-flow heard as BRUIT
↳ Bruit disappears at 80% stenosis
↳ common carotids ⇒ above + below the clavicles
↳ inguinal region + med. aspect of thigh ⇒ femoral A.
↳ popliteal fossa ⇒ popliteal As.

BUERGER'S TEST:
↳ supine patient:, raise feet + support legs at 45° for 2-3mins
↳ then " sits up, legs hang over edge of the bed.
↳ observe feet for 2-3 mins
↳ pallor on elevation + reactive hyperaemia ⇒ +ve test
↳ significant periph. A. disease
(elevated!)

~~ALLEN'S TEST~~: grab 1 wrist w/ 2 hands + w/ thumbs press down on ulnar + radial A ~~wrist~~; patient makes a fist repeatedly
↳ fingers turn white - ~~release~~ lower hand, keep arteries compressed
↳ press. over 1 artery is released. ⇒ if fine - turns red within secs
↳ repeat procedure + release other A.
↳ ulnar A ⇒ provides blood supply for superf + deep arches ↓ diff. palpation.

"Subclavian Steal Syndrome": dev. in obs of the subclavian A, ~~close~~ prox. to the origin of the vertebral A.
↳ exercise arm on the affected side ⇒ blood travels up carotid A → circle of Willis → vertebral A → arm ⇒ "stealing" blood fm post. cerebral circ.

↳ Symp = giddiness, collapse w/ or without loss of consciousness ∴ Neurological presentations

Abdominal presentations:

- Visceral ischaemia: 2 of 3 major visceral A. need to be critically stenosed before any signs of chronic mesenteric arterial insuff.
  ↳ Sense of central ab pain after eating (10-15min) ⇒ Mesenteric Angina
  ↳ severe ab pain, bloody diarrhoea, shock + profound metabolic acidosis

- Ab. aortic aneurysm: most are asymp until the aneurysm ruptures
  ↳ any doubt - US scan.
  ↳ ruptured ab aorta - diff to diagnose: no classical features of ab &/or back pain

"Blue Toe Syndrome" ⇒ atheroembolism + platelet debris + thrombotic material may arise fm ab aorta aneurysm.
  ↳ purple discolouration of toes + forefoot

Vasospastic presentations:
  ↳ Raynaud's ischaemia: digital ischaemia induced by cold + emotion
    ↳ 3 phases:
      ① pallor: digital A spasm &/or obs
      ② Cyanosis: deO2 of static venous blood
      ③ Redness: due to reactive hyperaemia

Veins: more common in the legs than arms; 4 ways:
① DVT
② varicose veins
③ Superf. thrombosis
④ Ch. V. insuff + ulceration

↳ Common symp: ① pain ② Swelling ③ Discolouration ④ Ulceration

exam:

① exam. w/ patient standing & then lying

② skin ⇒ colour changes, swelling + superf. venous dilatation & tortuosity
temp. diff.

③ elevate limb to 45° - note rate of veins emptying

④ May need Trendelenberg test: used to determine the pattern of venous incompetence in the leg.

↳ patient - sit on the edge of the exam. couch

↳ elevate limb (as far as comfortable) + empty superf. veins by "milking" the legs

↳ press thumb over the saphenofemoral junc. (2-3cm below + lat. to the pubic tubercle)

↳ ask patient to stand + maintain press. over this junc.

↳ if incompetence is present ⇒ varicose veins will not fill until the press is removed.

Chronic venous insuff.: skin changes in the lower leg ⇒ varicose eczema, lipodermatosclerosis, ulceration

↳ due to sustained venous HBP due to reflux (90%) &/or obs (100%) in superf + deep veins

Chronic leg ulceration: don't bandage unless there is inadequate arterial circ.

DVT: asym. swelling
pitting quality of the oedema
slightly ↑ temp

Plantar sign: supine + relaxed muscles; plants are palpated ⇒ pain

Homan's sign: calf pain on dorsiflexion of the foot

~~Loewenberg's sign~~ Loewenberg's sign: sphygmomanometer cuff *press. 100 mmHg + elicit pain in the calf

# Rheumatic diseases.

Auscultation of joint
palpation

Antibodies - asses them fm the fluid

Gout - uric Acid

# PART 1

## General history

① name, age, weight (gender) etc
② presenting complaint (PC) - open ended ques (Why they came to the hosp)
③ History of PC (HPC) - more details - how it started, progressed; when, wht happened next? Happend b4?

For pain SOCRATES
- site
- onset (gradual/sudden)
- character
- radiation
- associations (other symp)
- timing/duration
- exacerbating + alleviating factors
- severity (pain scale of 1-10)

- For any symp! => "Please Carefully Question This Method For Reliability + Resili ence

P = position (site + radiation)
C = character
Q = Quantity = severity
T = Transmission - assoc. features
M = modifying factors
R = Rate = onset gradual, progressive
R = Rhythm = periodicity.

④ Direct questioning - specific ques abt wt diagnosis u have in mind
⑤ Func inquiry/systems review - to find out any other symp.
⑥ Past Medical History (PMH) + Past Surgical history (PSH) - hosp before - when, why, where? any other illnesses conditions? any ops/procedures?

J = jaundice
A = anaemia
M = MI
T = TB
H = HBP + HD
R = Rheumatic fever
E = epilepsy
A = asthma + COPD
D = DM

JAM THREADS
S = stroke

⑦ Drug history (DH) ⇒ medication? tablets, inject? herbal remedies, the pill?
↳ allergies

⑧ Family history (FH) ⇒ parents - dead / alive? g/parents? - cause of death?
↳ children, spouse ; FH of HD or cancer

⑨ Social history (SH) ⇒ live alone? occupation, marital status.
house, apart? ↑ stairs? how many? any dependents? mobility
↳ drugs, alcohol, smoking - how many? since when? how often?

S = Smoking
A = alcohol use
D = drug use

L = living situation
A = activities of daily living
A = Anxiety
D = depression
D = diet
E = exercise
R = relationships
S = sexual history
S = support

STD LADDERS

⑩ FEMALES ⇒ Gynaecological ⇒ menstruation probs? pregnancies? menopause, deliveries?

Acc to HEART DISEASES:
chest pain - how long? characln?
exercise intolerance
shortness of breath (worse when lying - depends on position)
PND = Cardiac asthma = Paroxysmal nocturnal dyspnoea
orthopnoea
oedema
palpitations
dizziness
loss of consciousness
claudication
Ever had an ECG done?
Smoking
↑BP
Cyanosis

## Lung Diseases

COPD - pink puffers - emphysema - large chest, ok skin colour
blue blotters - chronic bronchitis

Cough - wht is it like? , do u use inhalers?
Sputum — have u ever had pneumonia/sinusitis?
Wheeze
haemoptysis - distinguish fm haematemesis
chest pain? - hw long time, any changes, localisation, wt causes an attack, duration ...
dyspnoea - hw " " , constant/coming+going/smoking, cough, blood present, alleviating factors

## KIDNEY / RENAL

irritative vs obstructive symp:
micturition - incontinence, dysuria, haematuria, nocturia, polyuria, hesistancy, terminal dribbling, ↓ force of stream, pain, urine colour, smell
- hw much do u drink a day? hw much do u urinate in 24hrs?

## GIT + HEPATIC

melena => 300cm³ of blood (min. amnt)

ab pain ; appendicitis
unintentional weight loss/gain
difficulty swallowing
indigestion
bloating
cramping
nausea/vomiting
diarrhoea/ ~~cough~~ constipation
inability to pass gas (= obstipation)
haematemesis (vomiting blood)
~~weight loss~~
alcohol, coffee, oig consumption

Bright red blood per rectum (BRBPR, ~~BRBPR~~ haematochezia

foul smelling dark tarry stools (melaena)
dry heaves of the bowels (tenesmus)
↳ the urge to shit wt no ~~poo~~ shit

### LIVER

↳ contact w/ hep. patient?
formely operated - gallstones etc.
alcohol abuse, drug abuse, ab pain, itch, fever, diarrhoea?
hepatomegaly, colour, ascites (+ splenomegaly), weight gain, caput medusa

## ENDOCRINE

parathyroid ⇒ osteoporosis

Adrenal gland
virilism
gynaecomastia

Thyroid symp ⇒ hypothyroid: prefer cold weather, mood swings, sweaty, diarrhoea, weight loss despite ↑ appetite, tremor, palpitations, oligomenorrhoea

- Trumpet sound hypothyroid: prefer hot weather, slow, tired, depressed, thin hair, heavy periods, constipation, dry skin
- walk of a tank (slow walk)
- lat eyebrows affected

Diabetes ⇒ polydipsia

↑ prolactin - lactation or gynaecomastia

prefer cold temp; hair loss

Vasopressin ⇒ diabetes insipidus

- acromegaly; gigantism (~~yes~~ children), small

## HAEMATOLOGICAL

anaemia (fatigue, weakness, melena, bleeding, easy bruising, tachy)

purpura

lymphoid (infx, swelling of LN, pain in LN)

bleeding disorders

Radio

Surgery to GIT

Transplantation

Freq. infx; malaria; hep B etc

FH - sickle cell anaemia, haemophilia, leukemia, thalassemia

Tick bites

- Do u bruise often?
- Shortness of breath while walking.
- dizzy spells
- ascites / liver probs
- coag. probs. (haemocoag disorders)

## PERIPH. A + VEIN DISEASES

| veins | arteries |
|---|---|
| varicose veins, oedema | haematomas, claudications |
| DM - gangrene | |
| DVT ⇒ recent bed rest or operations | cold extremities |
| recent travel (long haul flights) | |
| prev. DVT | |
| preg. | |
| fam. history of thrombosis | |

# RHEUMATIC DISEASES

Pain - joints - SOCRATES! ⇒ timing - acute/chronic
limbs - " ⇒ bone pain, nerve Entrapment, phantom pain, severe pain of sudden onset
elsewhere - SOCRATES! ⇒ S.C ⇒ localise dermatome

Stiffness ⇒ generalised/specific
no. of joints ↓, asymmetrical/symm, large/small joints, colour of joints
worse in the morning
pain while moving them/ can't move them
Swelling ⇒ no, asymm/symm, ↓ when first noticed, getting larger/smaller
Deformity ⇒ misshapen joints, fine course
eyes, mouth ⇒ dry, red eyes, unilat loss of vision
Systemic ⇒ rash (SLE), fatigue/breathlessness, fever, ab pain etc....
Surgery
Tick bites depends on the weather

# IMMUNOLOGICAL DISEASES

allergies - when? how?
autoimmune (rheumatic diseases) antibodies
immunodeficiency - more infec?
↳ 2° after splenectomy, AIDS/HIV

# PART-2

## Lab + clinical exam in Heart Diseases

① Environment => ECG leads, machine

② General appearance => colour - cyanotic, pallid, jaundiced, hyperpigmented
weight loss
syndromes: Turner's, Marfan's, Down's

③ Arms => Take BP, IV drug injec. scars — Systolic murmur => Aortic stenosis + mitral regurg

④ Face => facies & Cushing's, Acromegaly.
Malar flush (mitral stenosis) — 1st → 2nd => systolic pause (s
2nd → 1st => diastolic pause (longer

Inspect chest => scars, deformities, stitches, apex beat, visible pulsations
Heart sounds => 1st + 2nd (normal) — 1st => systole beginning
murmurs — 2nd => " end

mitral stenosis => cold onto left side => hear thrill (closer to chest wall)

Troponin T, CKMB, LDH, Aspartate transaminase (AST), Myoglobin (MB), Actin-H BF

Auscultation => Aortic - R 2nd Interspace parasternally
Pul. - L " " "
Tricuspid - Lower sternum (5th) + lower Left sternal border
Mitral - 6th MCL


① ECG, ② Chest xray (enlarged heart), ③ echo (LV func) - doppler echo
↳ transthoracic or transoesophageal
④ Radionuclide studies
↳ injected IV + γ-gearnera
⑤ Cardiac catheterisation (coronary angiography)
⑥ CT + MRI (congenital HD defects + Sarcoidosis)
↓
coronary arterial calcification

Percussion - upper the fluid
in the chest

Erb's point = 3rd intercostal space, left of the sternum

# Lung Diseases

Cyanosis — central (deoxyHb > 50g/l) — lips + tongue — arterial hypoxaemia
\ peripheral - fingers + toes → tissue hypoxia due to circ. disorders or cold

**Inspection** - bilat. symmetrical + elliptical in cross section
- scars - prev. Heart/lung surgery
- swellings
- marks + spots on the skin
- Subcutaneous lesions maybe visible (metastatic tumour nodules, neurofibromas)
- vascular abnormalities - spider naevi
  enlarged arterial vascular channels (aortic coarctation
  venous vascular channels (SVC obstruction)
- Shape abnorm: ↑ in AP Ø : "barrel shaped" ⇒ lung hyperinflation (COPD)
- kyphosis + scoliosis : kyphosis ⇒ exagg. ant. curvature of the spine
  scoliosis ⇒ lat. curvature
- pectus carinatum (pigeon chest): prominence of the sternum + adjacent costal cartilg
- " excavatum (funnel " ); localised depression of the lower end of the sternum

**Palpation** - Both sides of the chest expand equally during tidal + max. inspiration
- place hands firmly on the chest wall w/ fingers extending and sides of the chest ; thumbs shud meet in the midline , take a deep breath ⇒ thumbs shud move symmetrically apart 5cm
  ↳ pleural effusion, lung/lobar collapse ; bilat ⇒ COPD + diffuse pul fibrosis

**Percussion** - 4 basic types of percussion notes:
- Tympany ⇒ cavity w/ a lot of air
- Resonance ⇒ lungs
- Dullness ⇒ large parenchymal mass - liver
- Flatness ⇒ large muscle mass

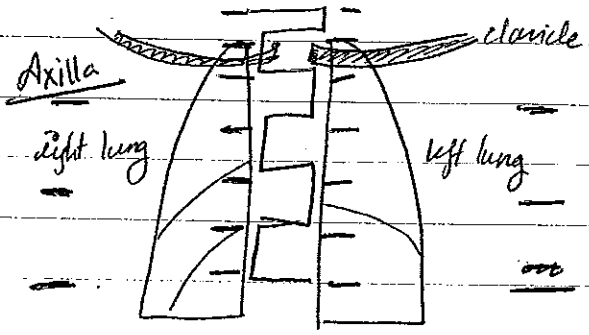
Anti-Pro BNP
↓↓
distinguish heart or lung (Heart Fail) disease
BNP = Brain Natriuretic Peptide

**Auscultation** : wheeze, crackles

## Percussion Note

| Type | Detected over |
|---|---|
| ① Resonant | Normal lung |
| ② Hyperresonant | Pneumothorax |
| ③ Dull | Pul. consolidation |
| | " collapse |
| | Severe pul. fibrosis |
| ④ "Stony dull" | pleural effusion |
| | haemothorax |

## Auscultation




Crackles: pul oedema
pul. fibrosis (fine)
bronchial secretions in COPD, pneum
lung abscess, bronchiectasis

Wheeze: implies airway narrowing
louder on expiration
inspiratory wheeze - severe airway narrower
high pitched wheeze ⇒ smaller airways (whistling)
low " " ⇒ larger bronchi
asthma + COPD

transudate ⇒ HF
exudate ⇒ tumour, inflam.

Pleural friction rub: when inflamed parietal + ~~visceral~~ visceral pleura move over 1 another

Pneumothorax click: rhythmical sound synchronous w/ cardiac systole
air between the 2 lays of pleura overlying the heart

Chest ①Xray, ②Sputum exam, ③pulse oximetry ($O_2$ sat ⇒ $SaO_2$ ⇒ ≥95%), ④arterial blood gas analysis, ⑤spirometry, ⑥peak expiratory flow, CT, echo, exam. of fluid

Astrup test ⇒ Acid-base equilib; $[O_2]$ $[CO_2]$

Immunological

hypersensitivity

Immunodeficiency ⇒ electrophoresis

ESR, CRP, opportunistic infec - atypical pneumonia

# PART 3

## Norm. ECG:

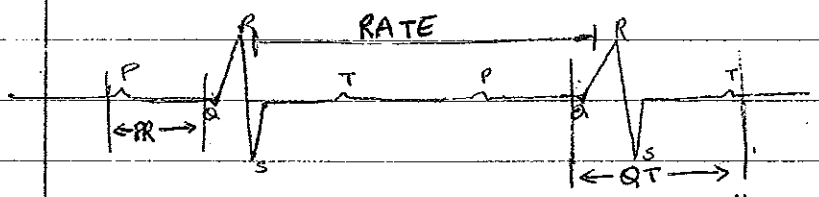
Rate, Rhythm, Intervals, QRS axis

Rate ⇒ RR interval (300/# no. of Large squares)

5 Large sq ⇒ 60 beats/min — Bradycardia = <60

4 " " ⇒ 75 " " — Tachycardia = >100

3 " " ⇒ 100 " "




P = atrial depol

QRS = vent. "

T = " repol

- Norm. HR = 60 - 90 bpm
- Norm PR interval = 0.12 - 0.22 secs
- QRS ⇒ <0.12 sec (3 small sq.) (0.06 - 0.10 sec)
- QT interval ⇒ <0.4 sec
- Frontal plane QRS Axis ⇒ +90° ⇒ -30° (adult)
  - ↳ QRS = +ve in leads I + II
- Regular Sinus rhythm = P wave + QRS complex w/ consistent PR interval
  - ↳ P waves in leads I + II = +ve

Normal Conduction ⇒ PR interval & QRS duration - within their limits

↳ Norm. Heart Axis ⇒ ① +ve QRS in lead I

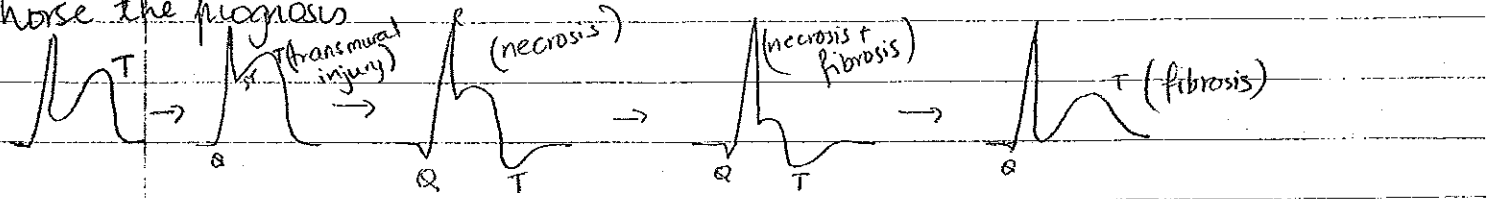
② +ve QRS in lead aVF

## ECG in Acute MI

MI ⇒ total coronary occlusion ⇒ Q-wave MI pattern

Subtotal " " ⇒ non-Q wave MI pattern (2/3 of MI) ⇒ ST segment depression or T wave inversion

Generally ⇒ more leads w/ MI changes (Q waves + ST elevation) ⇒ larger the infarct size + worse the prognosis



# Q waves MI's

Inferior MI ⇒ - leads II, III + aVF
- largest Q in III then aVF then II

True post. MI ⇒ - ant. precordial leads V1-3, but are a mirror image of anteseptal
- ↑ R wave amplitude + duration
- ST depression } Hyperacute ST-T wave changes
- large inverted T waves in V1-3

Anteroseptal MI ⇒ - Q, QS or qrS complexes in leads V1-V3(V4)
- evolving ST-T changes

Anterior MI ⇒ - same as above but V1 is spared
- if V4-6 = "anterolat"

High lateral MI ⇒ - MI features in leads I &/or aVL

MI + RBBB - rSR' V1 MI + LBBB

Non-Q wave MI: evolving ST-T changes over time without formation of patho Q wave

Changes ⇒ Convex downward ST segment depression only (common)
" upwards / straight ST segment elevation only (uncommon)
Symmetrical T wave inversion only (common)
Combination of above changes

Myocarditis, Metabolic disorders ($K^+$, $Ca^{2+}$)

Myocarditis: diffuse T wave inversions
↓ mimics acute MI
Saddle shaped ST elevations

Hyperkalemia ⇒ ↓ in size of P wave
tall & peaked T waves
widening of the QRS complex (prolonged depol) (> 0.12sec poss)

Hypokalemia ⇒ flattened/inverted T waves
U wave - prominent
prolonged QT interval (can lead to arrhythmias)
narrowed QRS complexes
ST depression

Hypocalcemia ⇒ prolonged QT interval
narrowing of the QRS complex
↓ PR interval
T wave - flattening + inversion
prolonged ST & ST-depression
} Same as hypokalaemia except 1!

Hypercalcaemia ⇒ (speeds repolarisation)
↳ MILD ⇒ broad based tall peaking T waves
↳ SEVERE ⇒ v/v/ wide QRS
low R wave
no P wave
tall, peaking T waves

Bradyarrhythmias:
- Bradycardia < 60 bpm
- P waves after QRS complex ⇒ Sinus arrest w/ junctional or ventricular escape rhythm + retrograde atrial activation
↓ narrow QRS
↳ wide QRS

- QRS + irregular P waves, outnumbering QRS complexes ⇒ some P waves prod QRS others don't ⇒ 2nd degree AV block

Sinus arrhythmia ⇒ irreg. QRS rhythm w/ 1:1 relationship between P waves + following QRS complex.

Tachyarrhythmias (>100 bpm) 4 groups => regular or irregular
narrow vs wide QRS complex

① Irreg. Narrow QRS complex => AF, Aflutter or true Atrial tachycardia w/ variable AV conduction + multifocal atrial tachycardia

AF => continuous, irreg in timing & morphology
>300/min, no discrete P waves

Multifocal atrial tachycardia => discrete P waves - vary fm beat to beat w/ atleast 3 diff morphologies

A flutter => regular, discrete, uniform atrial signals without intervening isoelectric periods

② Irregular, wide QRS complex => above 4 tachyarrh. w/ either BBB or ventricular pre-excitation & polymorphic VT (>250/min)

③ Regular, narrow QRS complex => Sinus tachycardia, Aflutter or true atrial tachycard: w/ a consistent AV conduction ratio

④ Regular, wide QRS complex => same as above w/ either BBB or ventricular pre-excitation & monomorphic VT

## Conductive disturbances:

↳ can occur at SAN, AV node or bundle branch system
↳ if it is a AV level: ↑ PQ interval or P waves not followed by QRS complexes

* 1st degree AV block:
↳ PQ > 0.20 sec (prolongation of PQ interval)
P wave still followed by QRS
↳ cause: degeneration of the conduction system
delay of conduction in the AV node => ↑ vagal tone, hyperkalemia, digitalis
Inf MI => AV block since AV node is supplied by the right Coronary A
ischaemia => injure AV node => delay/block of conduction

* 2nd degree AV block: every other P wave passes ⇒ 2:1 block; every 3rd wave ⇒ 3:1 block
↳ not all P waves are followed by QRS (beat dropout occurs); 3 types:

1) 2nd deg. AV block type I (Wenckebach):
↳ PQ interval prolongs fm beat to beat up until (the drop-out of one QRS complex) no P wave or QRS complex
↳ PQ interval that follows upon a dropped beat is the shortest
↳ RR interval ↓ every beat, until the dropped beat
↳ narrow QRS complex ; clustering of QRS complexes

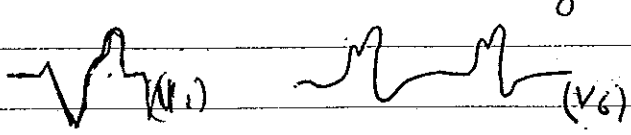
2) 2° AV block type II (Mobitz):
↳ beats are dropped irregularly without ↑ PQ interval prolongation
↳ wide QRS complex
↳ indicator for pacemaker

3) High grade AV block:
↳ 2 or more p-waves NOT followed by QRS complexes

* 3rd degree AV block: = total block - absence of AV conduction
↳ P waves + QRS complexes = no temporal relationship ⇒ AV dissociation
↳ ventricular rhythm can be nodal (ventricular rate 40-50bpm + narrow QRS complexes)
idioventricular (rate = 30-45 bpm + wide QRS (>0.12s))
absent
↳ Heart block = common complication of Inf. MI

LBBB vs RBBB:
↳ in $V_1$ ; QRS ≥0.12s ⇒ QRS in $V_1$ = below the baseline (i.e QS wave) = LBBB
LV → R / M ← RV
⇒ = above " " (i.e. RSR' wave) = RBBB
⇒ neither of the 2 ⇒ intraventricular conduction delay

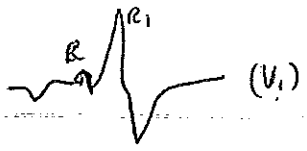
LBBB: QRS >0.12sec
Broad monomorphic R waves in I + $V_6$ w/ NO Q waves
" " S waves in $V_1$, may have a small r wave
(in $V_1$ ⇒ last depol. of the left ventricle, this is directed away fm $V_1$ ∴ -ve)



RBBB: QRS >0.12s

Slurred S wave in lead I + $V_6$

RSR'-pattern in $V_1$ where R' > R




LAH: left anterior hemiblock = Left Ant. fasicular block (LAFB)

↳ left axis deviation (< -30°) (no/few QRS widening)

no/very small S in lead I → (aVL = Tallest R wave)

Normal small q in lead I

S > R in leads II + III

LPH (left post. hemiblock) = LPFB = left post fasicular block

↳ right axis deviation (> 120°)

deep S in I (no/few QRS widening)

small q in III

Tallest R wave = III

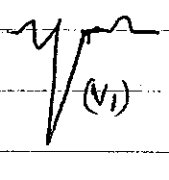
**Left + Right ventricle hypertrophy:**

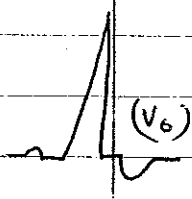
* LEFT ⇒ due to ↑BP or aortic valve stenosis

↳ QRS larger (V1-V6) ⇒ S wave in $V_1$ = deep

R wave in $V_4$ = high

Sometimes ST depression = $V_5$ + $V_6$



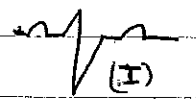

Sokolow-Lyon criterion: R in $V_5$ or $V_6$ & S in $V_1$ ≥ 35mm

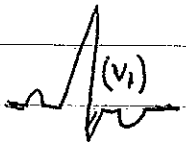
* RIGHT ⇒ emphysema or pul. embolisation or congenital HD

↳ QRS ⇒ -ve in I (∴ right heart axis)

(<0.12s) +ve in $V_1$

R wave ≥ S wave in $V_1$




* QRS in leads I, II + III for heart axis = +ve in all but highest in lead II!

Right Axis Deviation: QRS in I = -ve, II + III = +ve

Left " " : QRS in I = +ve, II + III = -ve

# Normal Chest Xray

- Chest xray - name, date
- Post-Ant (PA) most xrays (xrays fm back, plate in front)
- Trachea - central; paratracheal masses
- dark = air; light colour = dense material (Lung apices masses, cavitation, consolidation)
- any fracture / trauma?
- Cardiothoracic Index ⇒ Heart width = < 1/2 of the chest (2/3 of the heart on the RIGHT side)
- vascular vessels - fanning out?
- any liquid ⇒ congestion, haemothorax, fluid/thorax, inflam, oedema
- " infiltrate ⇒ inflam or tumours
- Costophrenic + cardiophrenic angles ⇒ not blunted (sharp) = suggesting an effusion.
- Right hemidiaphragm should be higher than the left

# Normal blood count + haemocoag. parameters

RBCs: Females: 3.5 - 5.5 $\times 10^{12}/l$
Males: 4.2 - 6.9 $\times 10^{12}/l$
Children: 3.8 - 5.5 $\times 10^{12}/l$

Hb: Females: 1.8 - 2.5 mmol/l (130 - 160 g/l)
Males: 2 - 2.7 mmol/l (130 - 175 g/l)

| | | DWCC |
|---|---|---|
| WBCs: Neutrophils (granulocytes) (neg): | 1.3 - 8 $\times 10^9/l$ | |
| | 45 - 74 % of WBCs | 59% |
| Neutrophils (banded): | 0.7 $\times 10^9/l$ | |
| | 3 - 5 % of WBCs | 4% |
| Lymphocytes: | 0.7 - 4.8 $\times 10^9/l$ | |
| | 16 - 45 % of WBCs | 30% |
| Monocytes: | 0.1 - 0.8 $\times 10^9/l$ | |
| | 3 - 10 % of WBCs | 6% |
| | | 1 (Basophils) |

(Granulocytes)

Eosinophils : 1-7 % of WBCs

Basophils : 0-2 % of WBCs

## Coagulation:

Platelet / Erythrocyte count (PLT) : 140 - 450 $\times 10^9$/l

Prothrombin time (PT) : 10 - 15 s

INR (corrected ratio of a patients PT to normal) : 0.9 - 1.2

Activated Partial thromboplastin time (APTT) : 18 - 45 s

Thrombin clotting time (TCT) : 11 - 18 s

Fibrinogen : 1.7 - 4.2 g/l

Antithrombin : 0.8 - 1.2 kIU/l

Bleeding time : 2 - 9 minutes

Viscosity : 1.5 - 1.72 cP (centipoise)

## Urine investigations ⇒ norm. + path findings

- urine pH ⇒ 4.4-8 (usually close to 7)
- " vol/d ⇒ 1-2 l/day
  - polyuria = > 2.5 l/d
  - oliguria = < 400 ml/d
  - anuria = < 100 ml/d
- Urea ⇒ 1.2 - 7 mmol/l
- Uric A ⇒ 0.18 - 0.48 mmol/l
- Creatinine ⇒ Males : 60 - 118 µmol/l
  - Females : 50 - 98 µmol/l

# Examination of the abdomen



Methods of physical examination: **observation** (inspection), **percussion**, **palpation**, and **auscultation**..

For orientation in the abdominal area **topographic division** by lines is used:

- Horizontal - running below the costal arcs and connecting the flat parts of pelvic bones.
- Vertical - running along the external margins of the straight abdominal muscles.

The **regions** created are called:

- In the upper part: epigastrium, right and left hypochondrium.
- In the middle part: right and left mesogastrium and periumbilical region.
- In the lower part: right and left hypogastrium and suprapubic region.

Another possibility is to divide the abdomen into **quadrants** by means of vertical and horizontal lines running through the umbilicus into the right upper and lower quadrants, and left upper and lower quadrants.

The abdomen is examined in a recumbent patient with bent knees, in a quiet place. The examiner comes from the right, during the examination he/she should be sitting.

**Observation (inspection)**

is used to assess the level of the abdomen in to the thorax, symmetry, and progress of the breath wave.

**Based on the nutritional condition, the physiological abdomen is the level or below the level of the chest. The navel is pulled in typical location. The breath wave proceeds bilaterally to the groin.**

Anaemia, senile cachexia

Obesity, monstrous ventral hernia

In addition to the abdomen, the inspection should be focused on the assessment of possible extra-abdominal disease manifestations in other locations.

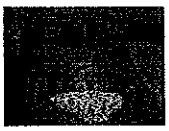
## General inspection

- Appearance:
  - **Cachexia** - occurs in tumours, especially in GIT.
  - **Bulky abdomen, asthenic trunk and extremities** can be found in decompensated liver cirrhosis and celiac disease.
  - **Obesity** is often associated with cholelithiasis.

- Position
  - **Immobile patient** - usually in diffuse peritonitis.
  - **Restless patient, often changing position** - in abdominal colic
  - **"On all fours"** - usually in patients with pancreatitis or pancreatic tumour.

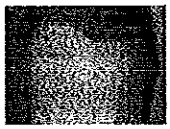
- Skin
  - **Pale** - anaemia
  - **Icteric** in praehepatic or hepatic jaundice (icterus).
  - **Icteric with excoriations** in posthepatic jaundice.
  - **Haemorrhagic diathesis** with petechia, purpura, and/or haematomas occurs in liver failure.
  - **Spider nevi** located in the upper part of the trunk or in the face and upper extremities occur in liver cirrhosis. The extent of lesions is influenced by the activity of the disease (possible non-specific incidence of the nevi in a small extent e.g. in pregnancy)

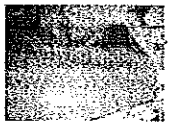

Icterus


Icterus


Icterus of the sclerae and skin of the face


Icterus


Spider nevi in the face


Spider nevi

## Extra-abdominal inspection

### Head

- Pale conjunctivae in anaemia.
- Yellow sclerae in icterus.
- Freckles surrounding eyes, mouth, and nose wings - occur in Peutz-Jeghers syndrome.
- Lips
  - Dried up in dehydration;
  - Smooth, red in liver cirrhosis.

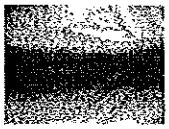

Icterus of the sclerae

### Oral cavity

- Foetor ex ore - hepatic in liver failure (resembles the smell of mice).
- Yellow-coloured palate in icterus.
- Tongue
  - Furred - connected with a disorder of the self-cleaning function;
  - Dried up - occurs in dehydration;
  - Smooth, reddish, so called Hunter's glossitis - occurs in pernicious anaemia.

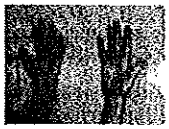

Dried-up tongue


Dried-up tongue


Erythema nodosum

### Lower extremities

- Hypoproteinaemic oedemas - perimalleolar or of a greater extent occur in liver cirrhosis, malabsorption syndromes etc.
- Erythema nodosum is manifested on the crura in patients with idiopathic intestinal inflammations (idiopathic proctocolitis, Crohn's disease).




Dupuytren's contracture, palmar erythema, tattoo of the forearm + detail

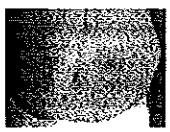
### Upper extremities

- Palmar erythema occurs in liver cirrhosis.
- Dupuytren's contractures in palms are more frequent in patients with cirrhosis.

## Abdomen

### Wall

- Navicular retraction occurs in extreme cachexia in tumours of the digestive tract.


Abdomen - scar

- Above the level of the chest - it occurs in obesity, meteorism, pregnancy, and ascites, where abdominal shape is changed according to the patient's position.
- Breathing movements do not proceed through the abdominal wall in localised or diffuse peritonitis.
- Visible pulsation of the abdominal aorta can be observed in thin patients or in aorta dilated by aneurysm.

### Colour of the skin

- Diffuse yellow in icterus, de-colouring is slower compared to the plasmatic level of bilirubin.
- Paraumbilical violet (Cullen's sign) occurs due to propagation of retroperitoneal haematoma in severe acute pancreatitis.
- Blue - haematomas of various age in haemorrhagic diathesis, related to subcutaneous application of heparin or insulin.
- Pigmentation in the extent of linea alba in Addison's disease or after radiotherapy.

### Striae

- Pearly striae are formed by the rapid distension of the abdominal wall in extension of the volume of the abdomen due to ascites, obesity, or pregnancy.
- Violet in Cushing's syndrome.

### Venous pattern

"Caput medusae" - the veins radially converge to the navel or are visible in lateral parts of the abdomen. Both findings occur in portal hypertension.

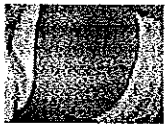
### Anasarca

Means advanced generalised effusion of the epidermis. The fluid is gathered also in the abdominal, thoracic, and pericardial cavities. It occurs in advanced right heart failure, hepatic cirrhosis, and serious hypoproteinaemia.

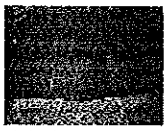
### Postoperative scars

have typical localisation according to the type of operation. The most frequent are:

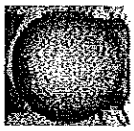
following the upper middle laparotomy + haematomas after the s.c. application of low-molecular heparin



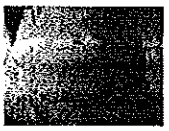
Pearly striae



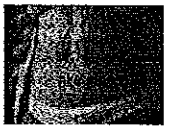
Violet striae



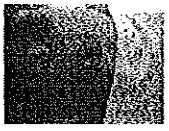
Caput medusae ascites, eversion of the navel, collateral venous pattern



Anasarca - effusion of the abdominal wall



Scar after the upper middle laparotomy



Scar after the gallbladder surgery

Abdomen - scar after the upper middle

- After the upper middle laparotomy (surgeries of the stomach and duodenum, gallbladder, and biliary duct).
- After lower middle laparotomy (gynaecologic, obstetric and urologic surgeries).
- After the combined laparotomy (extensive abdominal surgery).
- Right subcostal region (operation of the gallbladder).
- In the right hypogastrium (appendectomy).
- Suprapubic area (gynaecologic surgeries).
- After the right-sided and left-sided lumbotomy (kidney surgery).
- Combination of the mentioned scars with small scars of irregular shape (operations connected with drainage).
- Short scars in various locations after diagnostic or therapeutic laparoscopy.

Miserere

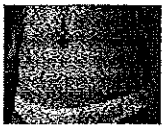
The colour of the scar indicates its age (red-pink - recent surgery, skin-coloured scar - of older date). The complicated healing can result in formation of a hernia in the scar. In some patients, keloid scars can be found.

**Physiological abdomen is symmetrical.**

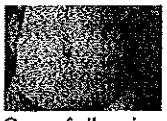
Pathological features that can be seen:

- Overall arch (bulge) in obese patients, in meteorism, iliac disorders, and in ascites (the shape of the abdomen changes relative to its position)
- Local bulge due to cysts, hernias, diastases of the straight abdominal muscles, tumours, enlarged liver, or spleen, distended full stomach and/or intestine, and urinary bladder.
- Hernias occur most often in the navel, groin, and postoperative scars (the size fluctuates depending on the intra-abdominal pressure).
- Eversion of the navel occurs in extensive ascites.
- Peristalsis of the stomach and intestine is usually visible in pylorostenosis or intestinal obstruction (ileus).

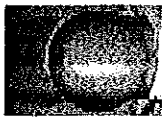
laparotomy + haematomas after the s.c. application of low-molecular heparin



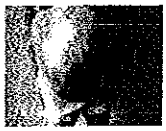
Abdomen - scar after the upper middle laparotomy, vertical scar along m. rectus + scars following the drainage + striae on the surface of the abdomen



Scar following appendectomy and cholecystectomy

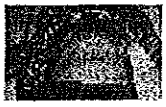


Overall arch (bulge) of tje abdomen, eversion of the navel



Hernia, obesity, monstrous ventral hernia

Obesity, monstrous ventral hernia, and ascites hepatic cirrhosis



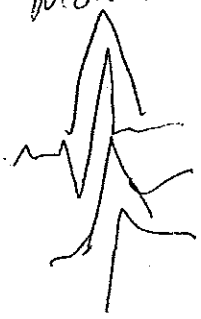
Murphy's sign : supine, R. costal margin + apply pressure just below it ; deep breath + press ⇒ they stop breathing ⇒ feco = Acute cholescytitis.

Tender liver : RHF, hepatitis, acute cholescytitis

ST elevation = MI

_M_ = "M" like = left bundle block

monomorph = Premature Ventricular Contraction - PVC



R. bundle branch block = $V_1 + V_2 + 2^{nd}$ "R" (+ followed by -ve T wave) is a bit higher

L. bundle branch block = $V_1 + V_6$

Premature Atrial Contraction = PAC

No relation between P + QRS complex (between Atria + Vent) ⇒ 3rd degree heart block

- murmur between 1st + 2nd sound = systolic = aortic stenosis
- " " 2nd + 1st " = diastolic

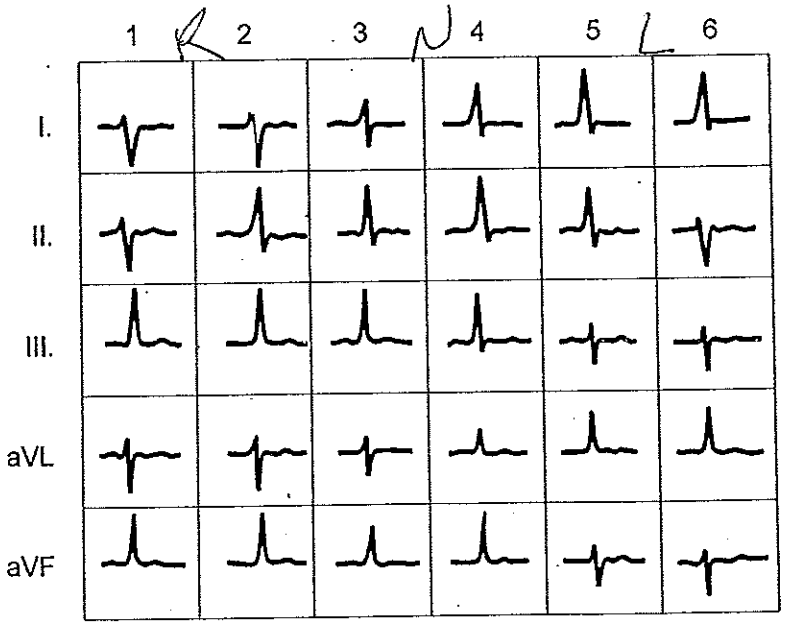
# 10. TABUĽKOVÁ PRÍLOHA K METODIKE HODNOTENIA EKG

## 1. Určovanie frekvencie podľa vzdialenosti R-R v mm

| Rýchlosť posunu EKG papiera 25 mm/s | | Rýchlosť posunu EKG papiera 50 mm/s | |
|---|---|---|---|
| 5 mm /hrubší štvorček = fr 300/min | | 5 mm/hrubší štvorček na EKG = fr 600/min | |
| Počet štvorčekov á 5 mm medzi RR | Frekvencia v úder./min | Počet štvorčekov á 5 mm medzi R-R | Frekvencia v úder./min |
| 1 ( 5 mm) | 300/min | 1 ( 5 mm) | 600/min |
| 2 (10 mm) | 150/min | 2 (10 mm) | 300/min |
| 3 (15 mm) | 100/min | 3 (15 mm) | 200/min |
| 4 (20 mm) | 75/min | 4 (20 mm) | 150/min |
| 5 (25 mm) | 60/min | 5 (25 mm) | 120/min |
| 6 (30 mm) | 50/min | 6 (30 mm) | 100/min |
| 7 (35 mm) | 43/min | 7 (35 mm) | 86/min |
| 8 (40 mm) | 37/min | 8 (40 mm) | 74/min |
| 9 (45 mm) | 33/min | 9 (45 mm) | 66/min |
| 10 (50 mm) | 30/min | 10 (50 mm) | 60/min |
| 11 (55 mm) | 27/min | 11 (55 mm) | 54/min |
| 12 (60 mm) | 25/min | 12 (60 mm) | 50/min |
| 13 65 mm) | 23/min | 13 (65 mm) | 46/min |
| 14 (70 mm) | 21/min | 14 (70 mm) | 42/min |
| 15 (75 mm) | 20/min | 15 (75 mm) | 40/min |

**Atropínový test funkčnej zdatnosti sínusového uzla**
Atropín v dávke 0,5 až 1,0 mg i.v. má medzi 3. a 10. minútou po aplikácii zvýšiť srdcovú frekvenciu minimálne o 30 % v porovnaní s východiskovou hodnotou.

## 2. Schematická orientácia pri určovaní typu krivky EKG




1. Extrémny pravotyp
2. Pravotyp
3. Vertikálny typ
4. Normotyp
5. Ľavotyp
6. Extrémny ľavotyp

# COMMON CARDIAC INVESTIGATIONS

Indications for common cardiac investigations are given in Table 3.27.

## Electrocardiography (ECG)

The standard 12-lead ECG (Fig. 3.30) uses recordings made from six precordial electrodes ($V_1$–$V_6$) and six different recordings from the limb electrodes (left arm, right arm and left leg). The right leg electrode is used as a reference.

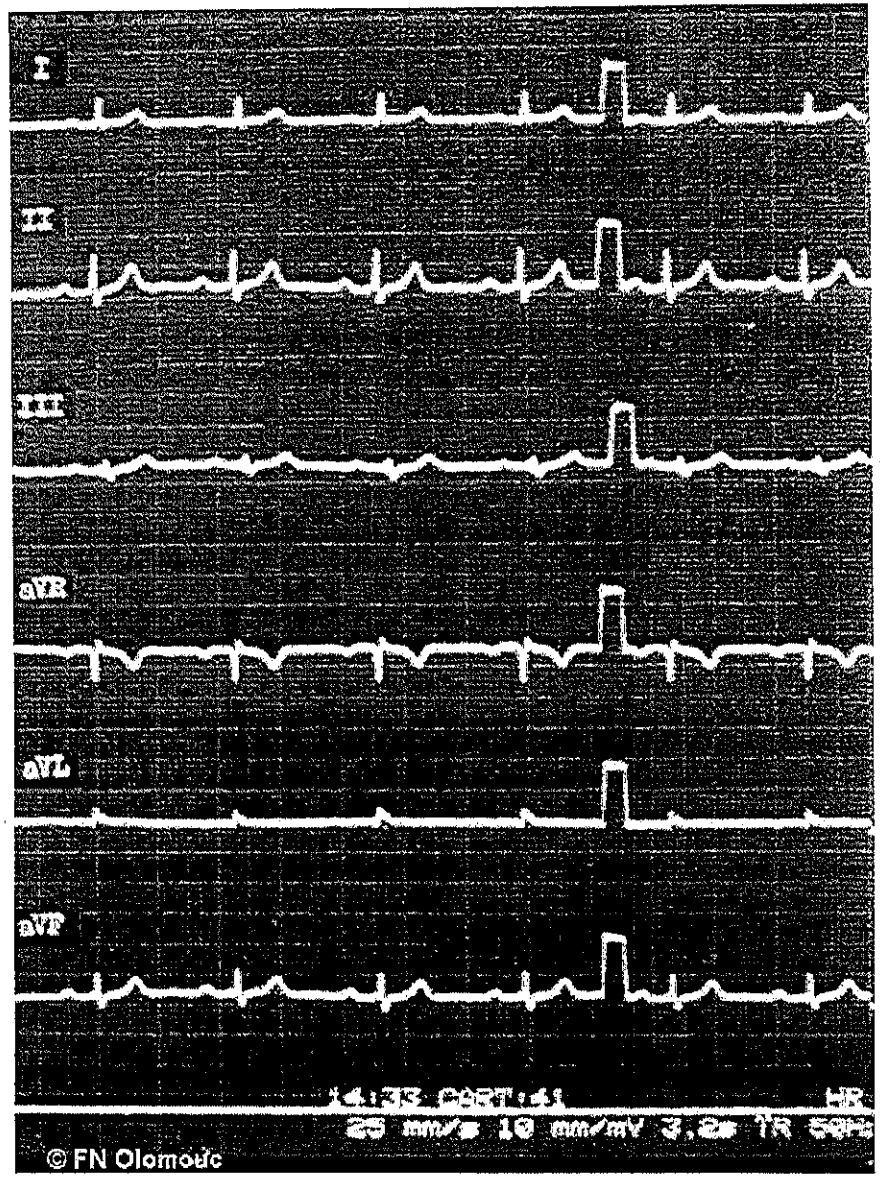
### *Ambulatory ECG monitoring*

Ambulatory recording can be made using cassette tape recorders or solid-state devices with digital memory. These make a continuous ECG recording that can be analysed by computer and checked by a cardiac technician. A typical recording lasts 24–48 hours. Patient-activated recorders are useful for capturing occasional arrhythmias and are activated only when symptoms occur (Fig. 3.31).

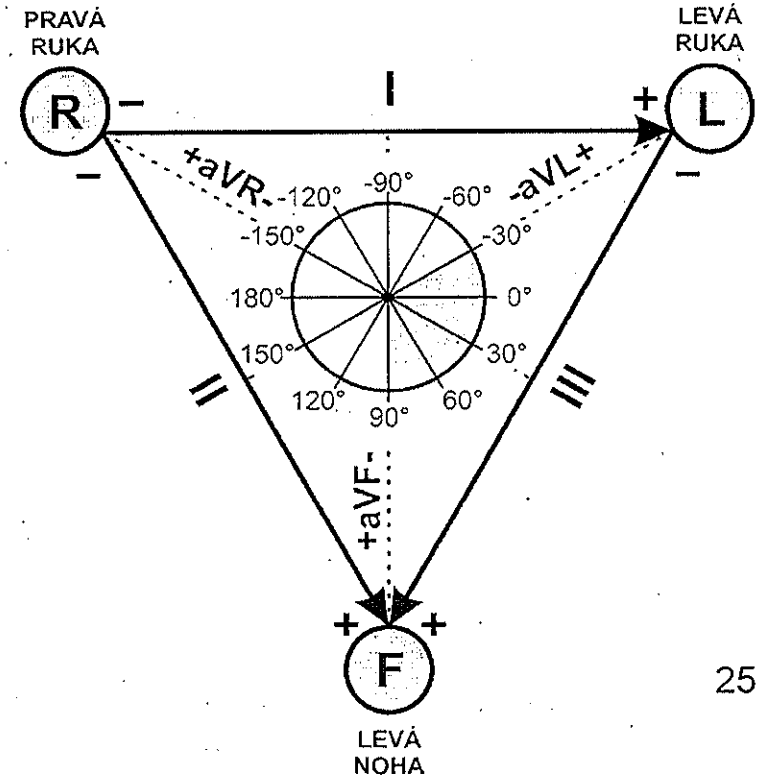
**Common cardiac investigations**

| Investigation | Indications | Implications |
|---|---|---|
| ECG | Numerous (medical and medico-legal) | Confirms the cardiac rhythm and reveals abnormalities in conditions such as left bundle branch block and Wolff–Parkinson–White syndrome<br>Diagnosis of myocardial infarction<br>Assessing for left ventricular hypertrophy. May reveal ischaemia; however, the resting ECG is usually normal in patients with angina |
| Exercise ECG | Chest pain | Ischaemic changes during exercise, especially when associated with symptoms, support a diagnosis of angina. However, exercise test can be normal in angina (false negative) and abnormal in healthy individuals (false positive) |
| | Post-myocardial infarction | Provides prognostic information |
| Ambulatory ECG monitoring | Palpitation | Confirms whether patients' symptoms are coincident with cardiac arrhythmia, e.g. ventricular ectopic beats or atrial fibrillation |
| | Syncope or presyncope | May show intermittent bradycardia or tachyarrhythmia if symptoms occur during monitoring |
| Chest X-ray | Numerous | Cardiothoracic ratio: maximum width of the cardiac silhouette/widest part of lung fields, usually the base. Increased in heart failure and valve disease<br>Pulmonary oedema in heart failure |
| Echocardiography | Cardiac murmur | Stenotic valve lesion readily diagnosed and accurately quantified<br>Regurgitation readily detected with semiquantitative assessment |
| | Breathlessness | Left ventricular function can be assessed. Impaired in heart failure |
| | Infective endocarditis | Valve vegetations confirm the diagnosis. Transoesophageal echocardiogram is more sensitive |
| Radionuclide studies | Breathlessness | Blood pool scanning provides an accurate assessment of left ventricular function, usually expressed as ejection fraction (end-diastolic volume – end-systolic volume/end-diastolic volume) |
| | Chest pain<br>Pulmonary embolism | Myocardial perfusion scan reveals ischaemic deficits in ischaemic heart disease<br>Lung scan shows a perfusion deficit compared with simultaneous ventilation scan |
| Cardiac catheterization | Angina | Coronary angiography reveals the extent and severity of coronary stenoses. This determines the therapeutic approach |
| | Valve disease | Better evaluated non-invasively by echocardiography. Cardiac catheterization is only indicated to assess the coronary anatomy in patients who require heart valve surgery |
| | Heart failure | Right heart catheterization in patients with severe heart failure helps determine suitability for cardiac transplantation |

# Srdeční osa (elektrická)




- vidíme, že nejvíce si jsou podobné negativní a pozitivní výchylky QRS komplexu ve svodu III
- na svod III je kolmý svod aVR, který ztotožníme se srdeční osou
- úhel srdeční osy tedy může být -150° nebo 30°
- průběh ve svodu aVR je více negativní, **srdeční osa je tedy +30° což je normální osa**



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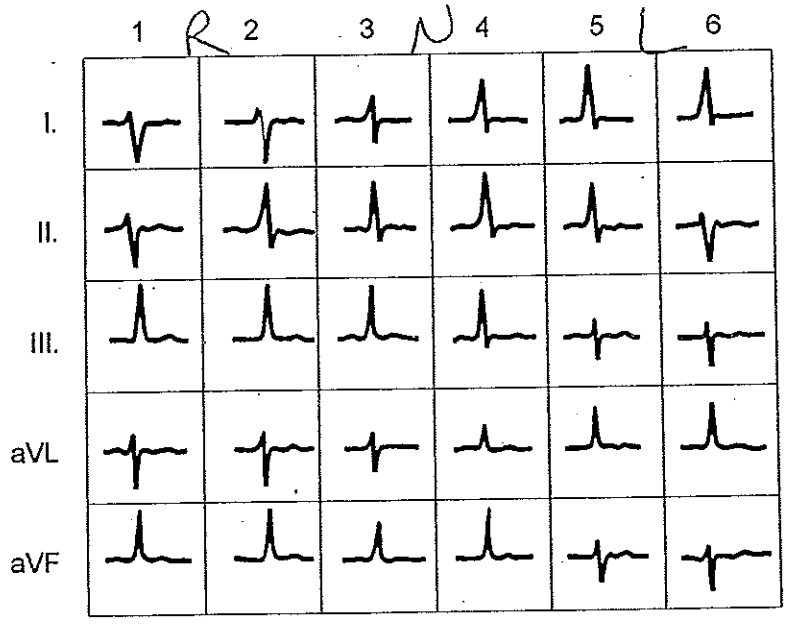


1. Extrémny pravotyp
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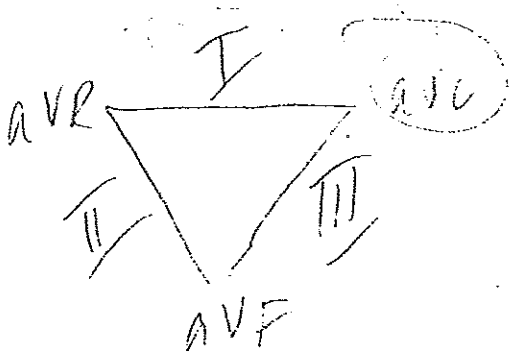
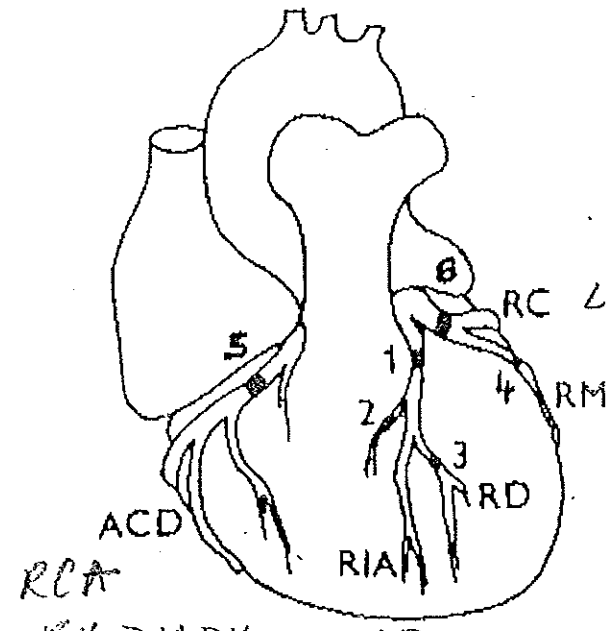
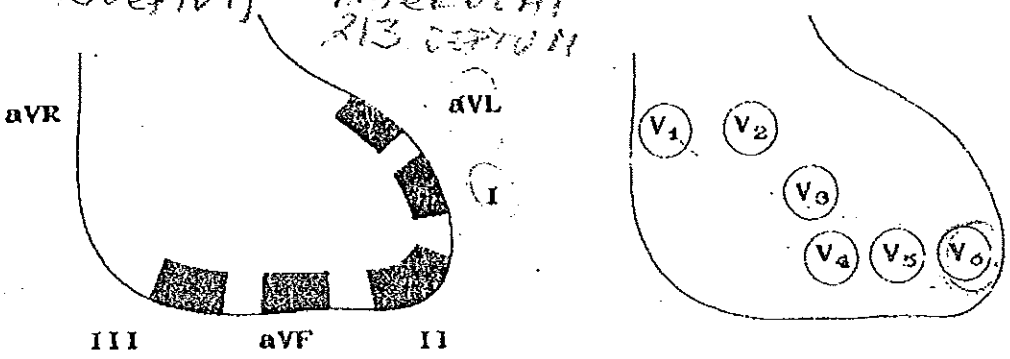
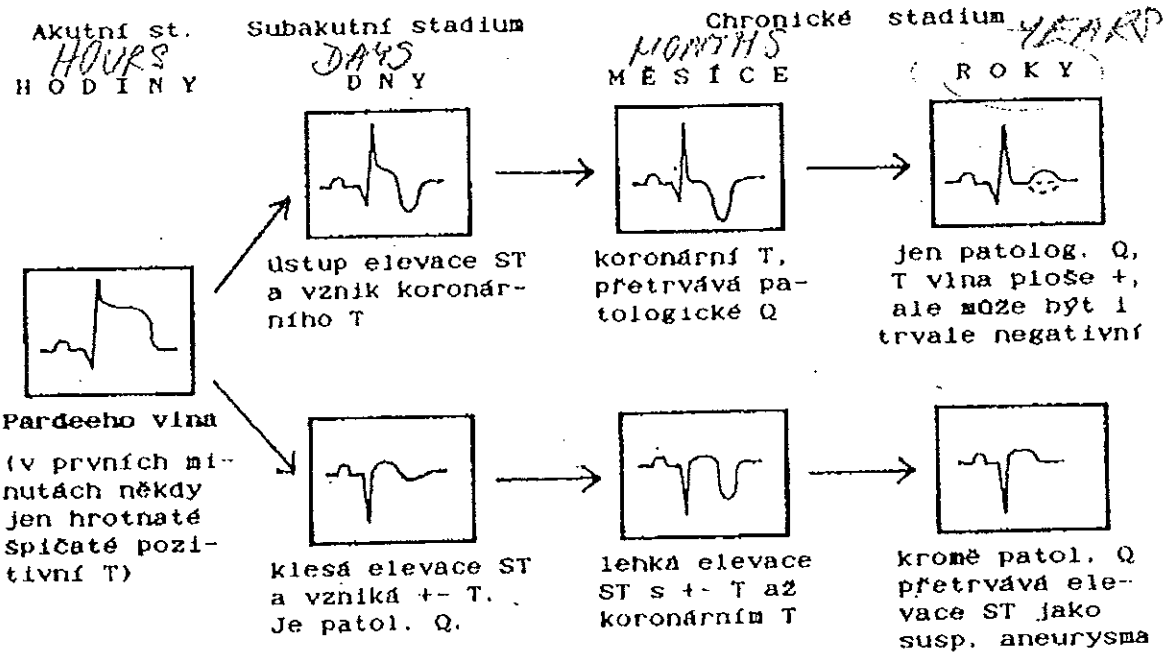
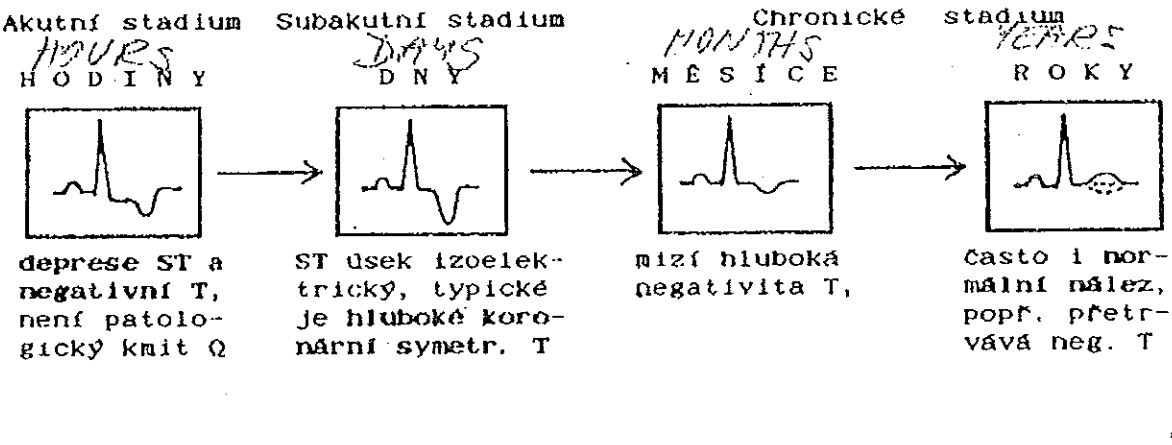


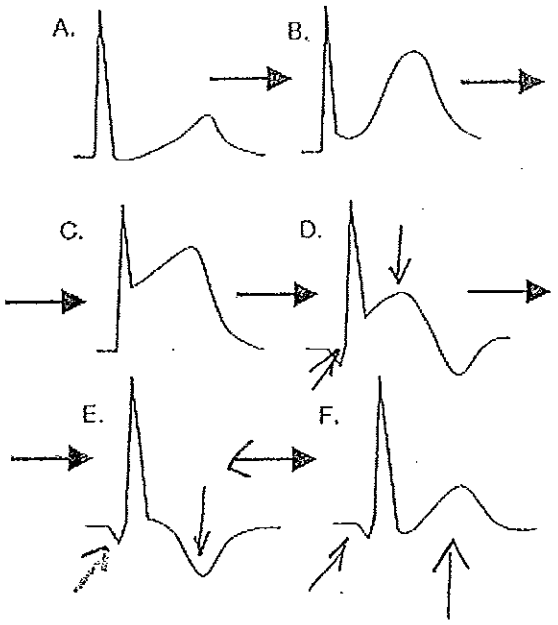




| | | |
|---|---|---|
| 1 | IM přední stěny | V1-V6, popř. I,aVL |
| 2 | IM anteroseptální | V1-V4 |
| 3 | IM anterolaterální | V4-V6 |
| 4 | IM vysoký laterální | I, aVL |
| 5 | IM diafragmatický(spodní) | I, III,aVF |
| 6 | IM diafragmatickolaterální | I, II, aVF, V5, V6 |
| 7 | IM cirkulární | V1-V6, I, II, III,aVF |
| 8 | IM zadní stěny | V7-V9 |
| 9 | IM pravé komory | V1, V3R-V6R |


Akutní st.
HODINY
Subakutní stadium
DNY
Chronické stadium
MĚSÍCE
ROKY
Pardeeho vlna
(v prvních minutách někdy jen hrotnaté špičaté pozitivní T)
ústup elevace ST a vznik koronárního T
koronární T, přetrvává patologické Q
jen patolog. Q, T vlna ploše +, ale může být i trvale negativní
klesá elevace ST a vzniká +- T. Je patol. Q.
lehká elevace ST s +- T až koronárním T
kromě patol. Q přetrvává elevace ST jako susp. aneurysma


Akutní stadium
HODINY
Subakutní stadium
DNY
Chronické stadium
MĚSÍCE
ROKY
deprese ST a negativní T, není patologický kmit Q
ST úsek izoelektrický, typické je hluboké koronární symetr. T
mizí hluboká negativita T,
často i normální nález, popř. přetrvává neg. T

ACUTE MI




Evolution of Acute MI

NSTEMI
STEMI

CHRONIC MI

Q-MI
non Q-MI

PATOLOGIC Q

10..
5

> 1/3 R

↔ > 40ms

## What Are the Symptoms of Arthritis?

Different types of arthritis have different symptoms. In general, people who have arthritis feel pain and stiffness in the joints. Some of the more common symptoms are listed in the box. Early diagnosis and treatment help decrease further joint damage and help control symptoms of arthritis and many other rheumatic diseases.

**Common Symptoms of Arthritis**

- Swelling in one or more joints
- Stiffness around the joints that lasts for at least 1 hour in the early morning
- Constant or recurring pain or tenderness in a joint
- Difficulty using or moving a joint normally
- Warmth and redness in a joint

## How Are Rheumatic Diseases Diagnosed?

Diagnosing rheumatic diseases can be difficult because some symptoms and signs are common to many different diseases. A general practitioner or family doctor may be able to evaluate a patient or refer him or her to a rheumatologist (a doctor who specializes in treating arthritis and other rheumatic diseases).

The doctor will review the patient's medical history, conduct a physical examination, and obtain laboratory tests and x rays or other imaging tests. The doctor may need to see the patient more than once to make an accurate diagnosis.

### Medical History

It is vital for people with joint pain to give the doctor a complete medical history. Answers to the following questions will help the doctor make an accurate diagnosis:

- Is the pain in one or more joints?

- When does the pain occur?

- How long does the pain last?

- When did you first notice the pain?

- What were you doing when you first noticed the pain?

- Does activity make the pain better or worse?

- Have you had any illnesses or accidents that may account for the pain?

- Is there a family history of any arthritis or other rheumatic disease?

- What medicine(s) are you taking?

Because rheumatic diseases are so diverse and sometimes involve several parts of the body, the doctor may ask many other questions.

It may be helpful for people to keep a daily journal that describes the pain. Patients should write down what the affected joint looks like, how it feels, how long the pain lasts, and what they were doing when the pain started.

**Physical Examination and Laboratory Tests**

The doctor will examine the patient's joints for redness, warmth, damage, ease of movement, and tenderness. Because some forms of arthritis, such as lupus, may affect other organs, a complete physical examination that includes the heart, lungs, abdomen, nervous system, eyes, ears, and throat may be necessary. The doctor may order some laboratory tests to help confirm a diagnosis. Samples of blood, urine, or synovial fluid (lubricating fluid found in the joint) may be needed for the tests.

Common laboratory tests and procedures include the following:

**Antinuclear Antibody (ANA)** - This test checks blood levels of antibodies that are often present in people who have connective tissue diseases or other autoimmune disorders, such as lupus. Since the antibodies react with material in the cell's nucleus (control center), they are referred to as antinuclear antibodies. There are also tests for individual types of ANAs that may be more specific to people with certain autoimmune disorders. ANAs are also sometimes found in people who do not have an autoimmune disorder. Therefore, having ANAs in the blood does not necessarily mean that a person has a disease.

**C-Reactive Protein Test** - This is a nonspecific test used to detect generalized inflammation. Levels of the protein are often increased in patients with active disease such as rheumatoid arthritis, and may decline when corticosteroids or nonsteroidal anti-inflammatory drugs (NSAIDs) are used to reduce inflammation.

**Complement** - This test measures the level of complement, a group of proteins in the blood. Complement helps destroy foreign substances, such as germs, that enter the body. A low blood level of complement is common in people who have active lupus.

**Complete Blood Count (CBC)** - This test determines the number of white blood cells, red blood cells, and platelets present in a sample of blood. Some rheumatic conditions or drugs used to treat arthritis are associated with a low white blood count (leukopenia), low red blood count (anemia), or low platelet count (thrombocytopenia). When doctors prescribe medications that affect the CBC, they periodically test the patient's blood.

**Creatinine** - This blood test is commonly ordered in patients who have a rheumatic disease, such as lupus, to monitor for underlying kidney disease. Creatinine is a breakdown product of creatine, which is an important component of muscle. It is excreted from the body entirely by the kidneys, and the level remains constant and normal when kidney function is normal.

**Erythrocyte Sedimentation Rate (sed rate)** - This blood test is used to detect inflammation in the body. Higher sed rates indicate the presence of inflammation and are typical of many forms of arthritis, such as rheumatoid arthritis and ankylosing spondylitis, and many of the connective tissue diseases.

**Hematocrit (PCV, packed cell volume)** - This test and the test for hemoglobin (a substance in the red blood cells that carries oxygen throughout the body) measure the number of red blood cells present in a sample of blood. A decrease in the number of red blood cells (anemia) is common in people who have inflammatory arthritis or another rheumatic disease.

**Rheumatoid Factor** - This test detects the presence of rheumatoid factor, an antibody found in the blood of most (but not all) people who have rheumatoid arthritis. Rheumatoid factor may be found in many diseases besides rheumatoid arthritis, and sometimes in people without health problems.

**Synovial Fluid Examination** - Synovial fluid may be examined for white blood cells (found in patients with rheumatoid arthritis and infections), bacteria or viruses (found in patients with infectious arthritis), or crystals in the joint (found in patients with gout or other types of crystal-induced arthritis). To obtain a specimen, the doctor injects a local anesthetic, then inserts a needle into the joint to withdraw the synovial fluid into a syringe. The procedure is called arthrocentesis or joint aspiration.

**Urinalysis** - In this test, a urine sample is studied for protein, red blood cells, white blood cells, and bacteria. These abnormalities may indicate kidney disease, which may be seen in several rheumatic diseases, including lupus. Some medications used to treat arthritis can also cause abnormal findings on urinalysis.

**White Blood Cell Count (WBC)** - This test determines the number of white blood cells present in a sample of blood. The number may increase as a result of infection or decrease in response to certain medications or in certain diseases, such as lupus. Low numbers of white blood cells increase a person's risk of infections.

### X Rays and Other Imaging Procedures

To see what the joint looks like inside, the doctor may order x rays or other imaging procedures. X rays provide an image of the bones, but they do not show cartilage, muscles, and ligaments. Other noninvasive imaging methods such as computed tomography (CT or CAT scan), magnetic resonance imaging (MRI), and arthrography show the whole joint. The doctor may look for damage to a joint by using an arthroscope, a small, flexible tube which is inserted through a small incision at the joint and which transmits the image of the inside of a joint to a video screen.

Oddělení klinické hematologie
FN u sv. Anny, Brno, Pekařská 53
656 91 Brno
tel.:4318 3151

Nálezová zpráva-schválená
Odběr :25-05-2010 09:00
Příjem:09:54 Statim
Přenos do NIS:25-05-2010/10:45

Rodné číslo: 231219726
Příjmení: PASEKA
Jméno: Vladimír
Požadavek č: 100525H355V

adresát:
ambulance interní
f.interní kardio.klinika

ateriál:...krev

| Metody: | Výsledky: | Jednotky: | Norm.hodnoty: | Grafika: |
|---|---|---|---|---|
| **Krevní obraz:** | | | | |
| Erytrocyty | 1.93 | 10E12/l | ( 3.50 - 6.00 ) | <=( ) |
| | ko opakovaně | | | |
| eukocyty | 5.6 | 10E9/l | ( 3.6 - 10.0 ) | ( * ) |
| Hemoglobin | 54.0 | g/l | ( 120.0 - 180.0 ) | <=( ) |
| Hematokrit | 0.150 | | ( 0.360 - 0.540 ) | <=( ) |
| rombocyty | 102 | 10E9/l | ( 150 - 450 ) | <-( ) |
| PV | 10.1 | fl | ( 7.4 - 11.0 ) | ( * ) |
| MCV | 77.7 | fl | ( 80.0 - 100.0 ) | <-( ) |
| MCH | 28.0 | pg | ( 27.0 - 34.0 ) | (* ) |
| | 360.0 | g/l | ( 330.0 - 360.0 ) | ( *) |
| | Anisocytosis | | | |
| | Microcytosis | | | |
| | Anemia | | | |
| | Thrombocytopenia | | | |
| **Koagulace:** | | | | |
| Quickův test | 0.87 | jedniny | ( 0.70 - 1.20 ) | ( * ) |
| nternational normalized ratio | 1.08 | | ( 0.88 - 1.22 ) | ( * ) |
| *Akt.parciální tromboplast.čas | 27.3 | s | ( 20.0 - 45.0 ) | ( * ) |
| Přepočet Aptt na normál | 0.85 | | ( 0.70 - 1.20 ) | ( * ) |
| Fibrinogen | 2.30 | g/l | ( 1.80 - 4.00 ) | ( * ) |

Laboratoř je akreditována ČIA podle normy ČSN EN ISO 15189 pod č.:M8053.
Protokol nesmí být reprodukován jinak, než v celku. Akreditovaná vyšetření jsou označena *.
APTT(SOP16),Antitrombin(SOP17),D-dimer(SOP18),DM(SOP19),Fibrinogen(SOP20),KOD(SOP21),QUICK+INR(SOP22),retikulocyty-analyzátor(SOP23),
retikulocyty mikroskopicky(SOP24),sedimentace erytrocytů(SOP25),trombocyty mikroskopicky(SOP26),von Willebrandův faktror(SOP27).
U akreditovaných metod jsou nejistoty k nahlédnutí na intranetu FN.

Prošlo analytickou a počítačovou kontrolou.
Kontroloval:Klevetová Marcela lab.spec.
Uvolnil :RNDr. Dagmar Jandlová

Vytištěno z NIS 28.05.2010 09:13:25

| | |
|---|---|
| FN u svaté Anny v Brně<br>Oddělení klinické biochemie<br>656 91 Brno, Pekařská 53<br>Telefon: 54318 3184<br>IČP:72001840 | Protokol o výsledku vyšetření<br>**********<br>AKUTNÍ LABORATOŘ - schválený<br>Přenos do NIS:22-03-2010 07:05 |

| | |
|---|---|
| Požadavek č: 100319A393L<br>Příjmení: PASEKA<br>Jméno: Vladimír<br>Rodné číslo: 231219726<br>Plátce : VZP /1<br>Diagnóza: D509<br>Dat. odběru: 19-03-2010<br>Čas odběru :<br>Dat. příjmu: 19-03-2010<br>Čas příjmu: 15:23 | Adresát:<br>lůžková část standard<br>I.interní kardioang.klin.<br>FN u sv. Anny v Brně<br>Pekařská 53<br>656 91 Brno<br><br><br>IČP: 72001601<br>1INT49 0114 tel.:2234 |

Poznámka k odběru:

| Metody: | Výsledky: | Jednotky: | Refer.hodnoty: | Grafika: |
|---|---|---|---|---|
| **Moč - zákl.chem.vyšetření:** | | | | |
| *pH | 5.5 | | ( 5.0 - 7.0 ) | ( * ) |
| Bílkoviny v moči PROTEINS | 1 | arb.j. | ( < 1 ) | ( *) |
| kóza | 0 | arb.j. | ( < 1 ) | (* ) |
| Urobilinogen | 1 | arb.j. | ( < 1 ) | ( *) |
| *Bilirubin | 0 | arb.j. | ( < 1 ) | (* ) |
| *Ketony | 0 | arb.j. | ( < 1 ) | (* ) |
| *Dusitany NITRITES | 0 | arb.j. | ( < 1 ) | (* ) |
| *Leukocyty | 0 | arb.j. | ( < 1 ) | (* ) |
| *Krev BLOOD | 2 | arb.j. | ( < 1 ) | ( )-> |
| *Vzhled APPEARANCE | čirá | | | |
| *Specifická hmotnost SPEC.WEIGHT | .021 | kg/l | ( 1.010 - 1.025 ) | ( * ) |
| *Barva COLOUR | žlutá | | | |
| **Moč - morfologické vyšetření :** | | | | |
| Erytrocyty | 1 | arb.j. | ( < 1 ) | ( *) |
| Leukocyty | 2 | arb.j. | ( < 1 ) | ( )-> |
| Válce hyalinní HYALINE CASTS | 1 | arb.j | ( < 1 ) | ( *) |
| Bakterie | 1 | arb.j | ( < 1 ) | |
| Amorfní soli AMORPHOUS SALTS | 2 | arb.j | ( < 1 ) | |

Protokol nesmí být reprodukován bez souhlasu laboratoře jinak, než celý.
Nejistoty jsou vyjádřeny pro 95% interval spolehlivosti a jsou v případě
potřeby k dispozici v laboratoři.Akreditovaná vyšetření jsou označena *.
HCG(total + beta)(SOP28),Thyreotropin(ultars.TSH)(SOP45),Kreatinin(SOP51),
volný tyroxin(fT4)(SOP54),Troponin I(cTnI) (SOP57),Bilirubin celkový(SOP64),
Prokalcitonin(SOP86),ABR-pCO2(SOP89),ABR-pO2(SOP90),ABR-pH(SOP91),Celkové
bílkoviny(SOP98),C-reaktivní protein(CRP)(SOP99),Močovina(SOP100),Fosfor(SOP101),
Calcium(SOP103),Natrium(SOP104),Kalium(SOP105), Chloridy(SOP106),GGT(SOP108),
Osmolalita(SOP109),Glukóza(SOP110),Chemické vyšetření moči testovacími
proužky(SOP111),

Prošlo analytickou a počítačovou kontrolou.
Kontroloval:Beránková Irena, atest. lab.
Autorizoval:DOC. MUDr.Vladimír Soška,CSc..

Oddělení klinické hematologie
FN u sv. Anny, Brno, Pekařská 53
656 91 Brno
tel. :4318 3151

Nálezová zpráva-schválená
**********

Odběr :15-04-2010 06:00
Příjem:07:43 Statim
Přenos do NIS:15-04-2010/13:47

Rodné číslo: 231219726
Příjmení: PASEKA
Jméno: Vladimir
Požadavek č: 100415R100F

adresát:
lůžková část standard
I.interní kardioang.klin.

ateriál:...krev

| Metody: | Výsledky: | Jednotky: | Norm.hodnoty: | Grafika: |
|---|---|---|---|---|
| **Krevní obraz:** | | | | |
| Erytrocyty | 3.53 | 10E12/l | ( 3.50 - 6.00 ) | (* ) |
| eukocyty | 4.9 | 10E9/l | ( 3.6 - 10.0 ) | ( * ) |
| emoglobin | 97.0 | g/l | ( 120.0 - 180.0 ) | <-( ) |
| Hematokrit | 0.288 | | ( 0.360 - 0.540 ) | <-( ) |
| Trombocyty | 55 | 10E9/l | ( 150 - 450 ) | <-( ) |
| MPV | 11.6 | fl | ( 7.4 - 11.0 ) | ( )-> |
| CV | 81.6 | fl | ( 80.0 - 100.0 ) | (* ) |
| CH | 27.5 | pg | ( 27.0 - 34.0 ) | (* ) |
| MCHC | 336.8 | g/l | ( 330.0 - 360.0 ) | ( * ) |
| **Diferenciální rozpočet leukocytů - mikroskopicky:** | | | | |
| ...menty } NEUTROPHILS < OLD YOUNG | 41.0 | % | ( 35.0 - 75.0 ) | (* ) |
| Tyčky | 8.0 | % | ( 0.0 - 5.0 ) | ( )-> |
| Lymfocyty | 35.0 | % | ( 20.0 - 55.0 ) | ( * ) |
| Monocyty | 13.0 | % | ( 0.0 - 10.0 ) | ( )-> |
| Eosinofily | 1.0 | % | ( 0.0 - 11.0 ) | (* ) |
| Lymfomonocytoidní buňky | 1.0 | % | ( 0.0 - 0.1 ) | ( )-> |
| Metamyelocyty | 1.0 | % | | |

Anisocytosis
Anemia
Thrombocytopenia
toxická granulace neutrofilů
- dif opakován



Prošlo analytickou a počítačovou kontrolou.
Kontroloval:Veronika Danielová lab. bez odb. dohledu
Uvolnil :RNDr. Dagmar Jandlová

## (1) Chapter title: Immunological Disorders and Tests

(a) [immunological disorders and tests (Google Search)] [index]

HYPERSENSITIVITY

### (2) Hypersensitivity

(a) Hypersensitivities are inappropriate immune responses to foreign material that is either within or in contact with the body

(b) Essentially, the body mounts a sometimes dramatic immune response against an otherwise harmless, or at least less-harmful substance, thereby doing more harm to the body in the course of the immune response than might have the original allergen

(c) Hypersensitivities may be divided into four types:

(i) Type I: Immediate hypersensitivity

(ii) Type II: Cytotoxic hypersensitivity

(iii) Type III: Immune complex hypersensitivity

(iv) Type IV: Cell-mediated Hypersensitivity (Delayed Hypersensitivity)

(d) [hypersensitivity reactions (Google Search)]

### (3) Anaphylaxis (anaphylactic shock)

(a) Anaphylaxis is a general term used to describe the detrimental effect(s) associated with hypersensitivities

(b) Anaphylaxis may be localized (annoying but not life threatening) or generalized (systemic and life threatening)

(c) Anaphylactic shock is a generalized anaphylaxis characterized by a significant, life-threatening drop in blood pressure

(d) [hypersensitivity reactions (Google Search)] [index]

### (4) Prophylaxis

(a) Prophylaxis refers to the protective effects associated with an immune response

(b) [prophylaxis (Google Search)] [index]

### (5) Immediate hypersensitivity (type I hypersensitivity; allergy)

(a) Immediate hypersensitivity occurs following the production of IgE antibodies against typically otherwise-harmless foreign antigens (which are known as allergens)

(b) Type I sensitivities are allergies

(c) [immediate hypersensitivity, allergy, reagin and (anaphylaxis OR hypersensitivity) (Google Search)] [index]

(6) Allergen

(a) An allergen is an antigen, the exposure to which results in a hypersensitivity reaction

(b) Note that allergens are non-self (i.e., foreign) antigens

(c) Since hypersensitivity (e.g., immediate hypersensitivity) is the result of a kind of specific immunity, an individual must be exposed to the allergen at least once (to sensitize the individual by inducing B cells that produce specific IgE antibodies) before exposures (subsequently) result in an allergic response

(d) [allergen (Google Search)] [index]

(7) Histamine (degranulation)

(a) The signs and symptoms of immediate hypersensitivity are a consequence of the release of histamine and other chemical mediators from body cells

(b) In the case of histamine, release occurs when IgE antibodies bound to basophils or mast cells bind to allergens

(c) Histamine is found intracellularly within vesicles (the granules within these cells) and **degranulation** is the term used to describe the release of histamine via the fusion of these vesicles with the basophil or mast-cell plasma membranes

(d) (in addition to histamine, prostoglandins and leukotrienes are reaction mediators that play important roles in mediating airway constriction)

(e) **See Figure 18.1, The mechanism of immediate (Type I) hypersensitivity, or anaphylactic hypersensitivity**

(f) [histamine, degranulation, degranulation and histamine (Google Search)] [index]

(8) Cytotoxic hypersensitivity (type II hypersensitivity)

(a) The term *cytotoxic* in cytotoxic hypersensitivity refers to host-cell damage caused by an over-zealous immune response

(b) Recall that a normal aspect of both specific and non-specific immune responses is extracellular killing, particularly the killing of host cells that are thought to be pathogen-infected

(c) Cytotoxic hypersensitivities are mediated by the binding of antibody's to body tissues which leads to the lysis of cells (either via ADCC or via the activation of complement)

(d) The negative consequences of not correctly matching blood types for transfusions are examples of the damaging effects of cytotoxic hypersensitivities (erythroblastosis fetalis is a related, additional example of a cytotoxic hypersensitivity)

(e) [cytotoxic hypersensitivity, type II hypersensitivity (Google Search)] [index]

(9) Immune complex hypersensitivity (type III hypersensitivity)

(a) One role of phagocytic cells (macrophages) is the removal of debris from body tissues (e.g., blood) and one kind of debris that results from specific immune reactions (specifically humoral immunity) are large complexes of antibody and antigen

(b) These complexes form as a consequence of the multivalent nature of both antibodies and antigens (i.e., an individual antibody molecule can bind to more than one epitope and thus, potentially, more than one antigen, while a large antigen or organism can display large numbers of individual epitopes)

(c) The phrase *immune complex* as in immune complex hypersensitivity refers to these antigen-antibody complexes, and type III hypersensitivity refers to an immune response that produces an excess of these immune complexes, particularly faster than macrophages (and the liver) can remove them

(d) The accumulation of these immune complexes can result in their depositing in otherwise healthy tissues followed by a damaging hypersensitivity immune response in those tissues to the not-engulfed immune complexes

(e) Certain autoimmune diseases (rheumatoid arthritis and lupus) are consequences of type III hypersensitivities as well as the serum sickness that results from a second exposure to an antitoxin

(f) [immune complex hypersensitivity (Google Search)] [**index**]

**(10) Cell-mediated hypersensitivity (type IV hypersensitivity, delayed hypersensitivity)**

(a) Cell-mediated hypersensitivity is mediated by T lymphocytes (rather than by antibodies)

(b) Cell-mediated hypersensitivity is also known as delayed hypersensitivity because the time between exposure to the eliciting antigen and the occurrence of symptoms can take many hours

(c) A common example of type IV hypersensitivity is poison ivy sensitivity (where, of course, the rash appears only after many hours—e.g., next day—following exposure to the poison ivy urushiol, the triggering oil)

(d) [cell-mediated hypersensitivity, delayed hypersensitivity (Google Search)] [**index**]

## IMMUNODEFICIENCY

**(11) Immunodeficiency**

(a) Immunodeficiency is characterized by an inadequate immune response, either in general or against specific antigens or pathogens

(b) This inadequacy contrasts with the temporary inadequacy of specific immunity as immune responses normally develop following first-time exposure to antigens

(c) Instead, immunodeficiency is characterized by an abnormally under response to antigens over the long (as well as the short) term and is indicated by a weakness in the ability of the body to fight legitimate pathogens

(d) We may speak of immunodeficiencies as being either inborn (primary) or acquired (secondary)

(e) Things that can lead to acquired immunodeficiencies include:

(i) Drugs (e.g., anti-cancer chemotherapies)

(ii) Pathogens (e.g., HIV/AIDS)

(iii) Inadequate nutrition and injury

(iv) Some cancers

(f) [immunodeficiency -AIDS (Google Search)] [**index**]

(g) Extreme exposure to sunlight that comes from maintaining a deep tan can also lead to pathogen-fighting inadequacies [impacts of UV radiation on the globe today (UV Rays and Global Changes)] [the ultraviolet light in sunlight can also stimulate herpes infections and might stimulate HIV infection (AIDS Treatment News)**◆** and other infections (UV Rays and Global Ghanges) [safe sun? (MicroDude)] [**index**]

(12) **Cyclosporin**

(a) Cyclosporin is a transplant anti-rejection drug that intentionally serves to induce a highly specific immunodeficiency

(b) That is, cyclosporin interferes with cell-mediated immunity, which is one of the mechanisms by which organ-transplant rejection occurs

(c) Unfortunately, cell-mediated immunity is important in fighting viral infections, serving as the means by which virus-infected cells are destroyed by the immune system; consequently, individuals on a cyclosporin regimen are more susceptible to viral infections

(d) This immunosupression is not complete, however (i.e., the rest of the immune system still functions), thus allowing the benefits of the drug (significant boost in transplantation efficacy since it greatly reduces the need to type-match tissues) to outweigh the costs (increased susceptibility to viral infections)

(e) In addition to viruses, cyclosporin increases tumor risks, an observation that is consistent with the tumor-fighting role of cell-mediated immunity, but, apparently, may also be a consequence of cyclosporin actually promoting the growth of certain tumors [*Nature* review on cyclosporin and TGF Beta (Biocognizance.com)]

(f) To prevent the rejection of transplanted organs, organ-transplant recipients must remain on a cyclosporin regimen for life

(g) [cyclosporin (Google Search)] [**index**]

(13) **Acquired Immune Deficiency Syndrome (AIDS)**

(a) The most-popularly understood cause of immunodeficiencies is, of course, AIDS, which is an immunodeficiency brought on by the infection with the Human Immunodeficiency Virus (HIV)

(b) (note that AIDS typically stands for acquired *immunodeficiency* syndrome as well as the *immune deficiency* phrase used in your text; a Google search for "acquired immunodeficiency syndrome" gives 79,800 hits on 3/14/02 while a Google search on the same day for "acquired immune deficiency syndrome" gives 54,600 hits)

(c) Immunodeficiency caused by HIV occurs because this virus preferentially infects host immune system cells, specifically those that carry the antigen that designates T lymphocytes as helper T lymphocytes (but the same antigen also is carried by macrophages and other cell types)

(d) HIV ultimately kills the cells it infects (e.g., via cell-mediated immunity by the body against HIV-infected cells); this creates a constant drain on the number of helper T cells present in the body, which in turn interferes with the functioning of both the cell-mediated and the humoral arms of specific immunity

(e) The virus is always replicating and the body is always fighting off the virus, with the virus mutating to evade specific immunity (more scientifically stated, with mutationally generated evavion-capable HIV variants are selected by specific immunity), and the specific immunity of the body must periodically produce new primary immune responses against the new variants of the virus

(f) Thus, HIV infection is characterized by

(i) an initial (~6 week) period of flu-like disease before specific immunity brings the infection under control

(ii) a steady-state period during which viral replication is kept more-or-less under control, with some break outs of viral replication as immune-system evading virus variants arise (this steady state can occur over many years, usually <10)

(iii) a gradual decline in immune system resilience and functioning until the growth of newly arising virus variants is no longer successfully brought back under immune-system control (AIDS)

(g) **See Figure 18.22, CDC classification of HIV disease and AIDS**

(h) The immunodeficiency characterized by AIDS is actually only the end-product of a long decline in immune system functioning and represents only the end stage of a typically decade-long disease process; that is, not all individuals who are HIV infected have AIDS (though all people with AIDS are HIV infected), but most people who are HIV infected (95%+), who are not successfully treated using modern antiviral chemotherapeutics, will eventually succumb to AIDS

(i) As a further complication, note that most HIV-infected people do not die with AIDS as a direct cause, but instead from secondary infections that are brought on the increases in susceptibility to infection that results from immunodeficiency

(j) Various external links: [**index**]

(i) [AIDS (Google Search)]

(ii) [The AIDS Knowledge Project]

(iii) [AIDS lectures: (1) definitions, origins, and prevalence, (2) the virus, (3) HIV disease and therapy, (4) the human immune response, (5) the biology the stages of HIV disease, (6) how is HIV transmitted? (7) preventing HIV transmission, (8) HIV testing, (9) AIDS and social issues (University of Michigan Bio 118)]

(iv) [does HIV prevention work? (JAMA HIV/AIDS Information Center)]

(v) [early impact on HIV infection, effects of treatment (JAMA HIV/AIDS Information Center)]

(vi) [the origin of AIDS (HIV InSite)]

(14) Human Immunodeficiency Virus (HIV)

(a) HIV is a plus-stranded, diploid, single-stranded RNA virus

(b) HIV is an enveloped virus that derives its envelope from the host-cell plasma membrane

(c) Also as part of the maturation of an HIV virion the virus envelope proteins are formed via the proteolytic cleavage of a precursor (larger) protein (without this cleavage the resulting virus particle is not functional and it is this cleavage that is blocked by anti-HIV protease inhibitors)

(d) HIV is a retrovirus that employs the enzyme reverse transcriptase to process its single-stranded RNA genome into a double-stranded DNA genome

(e) This double-stranded DNA genome is then inserted into a host chromosome

(f) **See Figure 10.13, Replication of RNA viruses**

(g) Not all inserted genomes are immediately active, thus allowing some virus-infected cells to evade immune system recognition (as well as drug treatment) over long periods (years, perhaps decades) thus making it nearly impossible to cure an HIV infection

(h) There are two major groups of HIV viruses in circulation among humans, HIV-1 which is probably derived from a chimpanzee virus (the revenge of the chimpanzees, who probably passed on the virus to humans as ♦bush meat♦) and HIV-2 which is probably derived from a monkey virus (one kind of SIV or simian immunodeficiency virus) (ditto re: the revenge of♦) [Nature on HIV origin (Biocognizance.com)] [the AIDS pandemic is new, but is HIV new? (*Systematic Biology*)]

(i) HIV-1 is by far the more prevalent (in the U.S.) and the more virulent of the two

(j) [HIV (Google Search)] [anti-HIV strategies (and additional HIV information) (Biocognizance.com)] [index]

(15) HIV epidemiology

(a) HIV/AIDS is a pandemic disease with estimates of world-wide cumulative prevalence (i.e., including those that have died♦so far a minority) as high as 50 million people or more

(b) HIV is transmitted via body fluids such as semen and blood

(c) Contact with the body fluids of others can occur particularly

(i) During unprotected vaginal intercourse (the prominent route of transmission in sub-Sahara Africa) or during anal intercourse (in both cases the recipient is the more susceptible to infection)

(ii) From needle sharing during intravenous drug use

(iii) From the transfusion of blood or blood products (rare since the implementation of immunological testing of the blood supply)

(iv) From mother to child either in utero, during passage down the birth canal, or from breast milk

(d) ♦It is not possible to acquire the HIV virus by donating blood because new, sterile needles are used.♦

(e) Health-care workers should observe universal precautions to avoid exposure to blood-borne pathogens including HIV

(f) [HIV epidemiology (Google Search)] [index]

**(16) HIV vaccination**

(a) **Difficulties in developing vaccines:** (not responsible for material under this subheading, i.e., subheading (a))

(i) While from a public health point of view vaccines are wonderful things, in practice it is not necessarily easy to engineer effective vaccines against a given disease

(ii) Reasons that vaccine development is not always a fruitful endeavor can include:

} *limited range:*

a given vaccine tends to be effective only against individual serovars of pathogen species (some species have hundreds of serovars♦a serovar is a strain that is differentiated from other strains of a given organism by serological means)

} *disease isn't immunizing:*

for some pathogens even exposure to disease (the ultimate form of immunization) does not confer active immunity

} *rapid evolution:*

development of vaccines against particularly rapidly evolving pathogens (such as HIV) is also difficult because the pathogen, essentially, is an immunologically moving targets♦at best such vaccines are rapidly made obsolete by pathogen evolution (e.g., anti-influenza vaccines)

} *exacerbation of disease:*

vaccines of certain types, against certain pathogens can actually exacerbate disease when it occurs

} *cause of disease:*

live vaccines retain at least some potential for causing the disease they are charged with preventing; this is especially true with regard to immunodepressed individuals (e.g., live polio vaccine)

} *cost-benefit problems:*

successful vaccine delivery is not always economically or politically justifiable

(b) In the mid-to-late 1980s optimism was high that an anti-HIV vaccine could be rapidly developed. This optimism was based on the premise that molecular techniques in biology had advanced so far that the development of a molecular (recombinant, subunit) vaccine against any pathogen was possible given the application of sufficient resources.

(c) Stemming from this optimism the more-easily developed whole live or killed vaccine strategies were rejected as too dangerous:

(i) a live HIV could infect indefinitely, possibly reactivated as a pathogen given future host immunodepression

(ii) a dead HIV might not be completely dead, or completely harmless given subsequent exposure to living HIV

(d) However, it turns out that HIV possesses many of the qualities that would lead one to predict difficulty in vaccine development:

(i) There exists numerous and extensive serological variation among wild isolates (contrast polio for which only three serovars are known).

(ii) We lack data on having the disease actually being immunizing; after all, HIV-infected individuals successfully control their infections for years without actually eliminating the infection, nor staving-off disease

(iii) HIV is the poster child for rapidly evolving pathogens; almost nothing else mutates or evolves faster than HIV

(iv) See immediately above discussion of the dangers of whole vaccine use

(e) Even if a disease-preventing vaccine existed, how many of us would volunteer to receive a vaccine that

(i) By definition made us HIV *seropositive* (if not necessarily HIV infected)

(ii) That could prevent (in most cases) the progression of an HIV infection towards AIDS, but could not actually prevent the occurrence of an HIV infection

(iii) Possessed a less-than full (<100%) potential to prevent the progression of infection towards AIDS

(f) Furthermore, consider that those who are most at-risk for HIV infection (in the U.S., at least) are also the same individuals (with the likely exception of upper- and middle-class homosexuals) who are most-likely to fall through the cracks of health-care systems and therefore the least likely to be vaccinated

(g) An effective anti-HIV vaccine may never arrive, and when it does it may not be able to achieve its promise for bringing the HIV pandemic under control (especially if vaccination serves as a signal to everyone and their mother to go out and have unprotected sex with multiple partners)

(h) In the mean time the best we can do is to live an HIV-defensive life and, as a society, to vigorously protect our tissue (e.g., blood) supplies through vigorous serological screening for HIV infection

(i) [HIV vaccination (Google Search)] [HIV biology, vaccine-strategy emphasis (Bio 160: Vaccine Development)] [index]

## IMMUNOLOGICAL TESTING (SEROLOGY)

### (17) Immunological tests (serology)

(a) A variety of experimental methods exist that employ immunological reagents, particularly antibodies

(b) These tests are typically employed

(i) as a means of testing for the presence of certain antigens in experimental unknowns

(ii) for quantifying the presence of specific antigens

(iii) as a means of detecting the anigen's owner (e.g., a pathogen), or

(iv) as a means of characterizing an immune response (including detecting and quantifying antibodies)

(c) Because the majority of these tests employ antibodies (as opposed to cell-mediated immune responses) and since the crudest and earliest-to-be-worked-with antibody-containing reagent is serum (i.e., the liquid portion of blood once clotting has taken place), the study and development of such tests is called serology

(d) Here, for the sake of brevity, we will consider in depth two of the more-modern serological methods, the ELISA and the Western blot, particularly to enhance our understanding of how laboratories assay for HIV seroconversion

(e) [immunological tests, serological tests, serology (Google Search)]

### (18) Serum

(a) Whole blood may be allowed to clot upon exposure to air (oxygen); if one removes the solid portion of the clot (using centrifugation), the remaining liquid is called serum

(b) Serum contains large quantities of proteins including high concentrations of antibodies

(c) Harvesting serum represents the means by which the antibody portion of blood is crudely purified

(d) [serum (Google Search)] [index]

### (19) Seroconversion

(a) Seroconversion is the production of antibodies following exposure to an antigen

(b) The production of specific antibodies can be used as a diagnostic for previous exposure to specific antigens (e.g., HIV)

(c) [seroconversion (Google Search)] [index]

### (20) ELISA

(a) The ELISA technique (which stands for Enzyme-Linked Immunosorbent Assay) is a method by which tagged antibodies are used to visualize specific proteins

(b) This immunological technique is very powerful because, by varying proteins and antibodies, it allows a rapid detection of very specific proteins or antibodies

(c) ELISAs consist of:

(i) Binding of a substance, such as a protein or a specific antibody, to the plastic well of a assay plate

(ii) Washing excess (unbound) substance from the well

(iii) Blocking unbound plastic with an otherwise inert substance (such as skim milk and then washing)

(iv) Probing with a substance that binds to the first substance (e.g., an antibody to the bound protein or a protein to the bound antibody and then washing)

(v) Probing with a substance that is linked to an enzyme (this second probe can be done simultaneously with the first probe, e.g., an enzyme-linked antibody as the only probe and then washing)

(vi) Addition of substances that undergo a color reaction in the presence of the enzyme tag

(vii) Only if all of the steps work in this assay (e.g., protein bound to plastic followed by antibody bound to protein followed by enzyme-tagged antibody bound to the first antibody) will the color reaction occur since washing removes all unbound reagents from the reaction well

(viii) A positive color reaction thus is used as a test for the presence in an experimental unknown of the presence of one of the necessary components (e.g., a specific protein or a specific antibody); see a microtiter plate used for ELISA with color reaction increasingly intense going from bottom to top ☒

(d) **See Figure 18.34, Enzyme-linked immunosorbent assay (ELISA) is a modification of RIA**

(e) The use of the ELISA technique is extensive in microbiology and immunology, in both the clinic and research, but it is perhaps best known as the primary means by which people and blood are tested for HIV seroconversion (as illustrated below)

(f) [ELISA (Google Search)]

**(21) HIV antibody test**

(a) The ELISA commonly employed to test for HIV seroconversion specifically is at test for the presence of anti-HIV antibodies in blood

(b) The ELISA consists of (second Roman numerals are from general ELISA description above)

(i) (i) partially purified HIV antigen is bound to plastic (picture above right.. not albumin employed to block plastic)

(ii) (iv) patient serum is used as the antibody probe (middle, right)

(iii) (v) enzyme-linked anti-human antibody is then used to probe for the presence of bound serum (bottom, left)

(iv) the enzyme-linked antibody remains bound in a well only if the patient's serum contains antibodies that bind to HIV proteins, and for the most part an individual will possess anti-HIV antibodies (of sufficient titer) only if they have been infected with the HIV virus

(c)

(d) "Generally, serum antibodies to HIV can be detected by indirect ELISA within 6 weeks of infection." (p. 529)

(e) If this ELISA test indicates seroconversion (i.e., the presumed presence of anti-HIV antibodies; bottom, right in above illustration), then a second, more rigorous test is employed to rule out false positives (that is, tests that falsely indicate HIV seroconversion)

(f) The more rigorous method typically employed is a Western blot assay

(g) [HIV antibody test (Google Search)] [index]

(22) Western Blot

(a) History (not responsible for material under this subheading, i.e., subheading (a))

(i) There exists a series of gel-based blotting methods known as the Southern Blot, the Northern Blot, and the Western Blot (the originator was named Southern and scientists being scientists subsequent blotting methods were named within this tradition)

(ii) The Southern Blot separates DNA on a gel (different sizes migrate at different rates) and probes with DNA (e.g., radioactive DNA); the tagged DNA is the visualizer of the DNA in the gel so only those gel DNAs (bands) that are probed for are visualized

(iii) The Northern Blot separates RNA on the gel and probes with DNA

(b) The Western Blot separates proteins on the gel (producing a protein profile) and probes with antibodies

(c) Those antibodies are labeled for example with radioactive elements (or various enzymes) [Western blot chemiluminescence reagents (NEN Life Sciences Products]

(d) In the case of HIV testing, the proteins on the gel are HIV proteins and the antibodies come from the serum of individuals; similar to variations on the ELISA technique, these human antibodies, if they bind HIV proteins, are visualized by labeled anti-human antibodies

(e) **See Figure 18.36, Western blotting test for HIV antigens in blood (note how blotting techniques get their name from the transfer blotting of, in this case, proteins in the gel to a non-gel material, which is the material that is probed with the labeled materials)**

(f) The nice thing about Western blotting is that the results can be very specific where protein bands are only visualized if the antibodies bind the protein, and then only those protein-bound antibodies are then visualized (e.g., a mixture of all of the proteins in a cell could be probed with a single monoclonal antibody that visualized only a single protein type from that mixture)

(g) However, the Western blot is also more time-consuming and expensive than the ELISA so is used in HIV testing only to further characterize ELISA positives